Journal of Contemporary Psychotherapy (2019) 49:169–176
https://doi.org/10.1007/s10879-018-9408-9

ORIGINAL PAPER

Strategic Symptom Displacement in Therapy of a Motor Conversion Disorder Comorbid with PTSD: Case Presentation

Valery Krupnik[1] · **Mariya V. Cherkasova**[2]

Published online: 6 October 2018

Abstract

There is no psychotherapy of choice for conversion disorders. We present a case of a motor conversion disorder comorbid with posttraumatic stress disorder, where we designed a psychotherapeutic approach based on the Bayesian model of brain functioning, specifically, the Bayesian model of functional neurologic symptoms. The model posits that such symptoms are produced as a result of hyper-precise prior beliefs (priors). Priors can be both conscious and unconscious and exist at different levels from perceptual to cognitive. Decreasing their precision/rigidity may then alleviate the related symptoms. In accord with this rationale, we used cognitive, experiential, and behavioral interventions meant to target and modify the putative priors. Central to this strategy was strategic symptom displacement. Application of this intervention coincided with reduction of the target motor symptom, indicating a possibility of causal relationship. We suggest strategic symptom displacement as an integral part of a universal therapeutic approach targeting priors: strategic modification of priors.

Keywords Functional symptoms · PTSD · Integrative psychotherapy · Bayesian brain · Priors

The Bayesian model of predictive coding has been widely used in machine learning as well as in modeling brain functioning (e.g Clark 2013). The model posits that the brain (or any neural circuit) adapts to the environment by generating a working model of it, updating it through inferring and thus anticipating the environment's future states from the incoming information. When such information does not confirm the model's prediction, a predictive error is generated. Correction of the predictive error is the mechanism of updating and thus increasing the model's predictive accuracy. In informatics, such predictions are called 'priors,' a term denoting a probability distribution of hypotheses about the causes of an event that itself has a probability distribution. Bayesian statistics establish a relationship between these distributions. In a less formal language, a prior is the brain's probabilistic belief/expectation/assumption about the environment (both internal and external). It operates on every neural level from conscious beliefs to circuits inaccessible to consciousness. Prior beliefs are tested with environmental input and are adjusted by prediction errors. The model has been proposed as the universal principle of brain functioning (Friston 2010). A clinical corollary to the model is that malfunction of the brain's predictive coding can be considered a universal cause of most psychiatric conditions and symptoms, as has been suggested for hallucinations (Powers et al. 2017), depression (Badcock et al. 2017), and disorders of personality (Moutoussis et al. 2014). Perhaps, the clearest example is hallucinations. When conditioned to develop auditory hallucinations, people with psychotic features formed more rigid auditory expectations compared to controls, as manifested by their higher rate of cued hallucinations (Powers et al. 2017). Moreover, this effect correlated with the severity of psychosis.

The Bayesian model has also been applied to functional neurological disorders (Edwards et al. 2012). In short, the model suggests that in conversion/functional neurological disorder, functional symptoms result from sensory and motor neural networks' dysfunctional predictive coding. That happens when the brain's prior beliefs about causes of sensory information acquire an abnormally high value (precision in statistical terms). An abnormally high precision of a prior belief may cause its failure to be appropriately updated and corrected by prediction errors. Instead, the brain

✉ Valery Krupnik
valery.y.krupnik.civ@mail.mil

[1] Mental Health Department, Naval Hospital Camp Pendleton, 200 Mercy Circle, Camp Pendleton, CA 92055, USA

[2] University of British Columbia, Vancouver, CA, Canada

will then 'fulfill' the belief by generating a corresponding abnormal sensation or movement, or lack thereof in case of a negative symptom (this notion is cognate to the folk concept of self-fulfilling prophesy). These abnormal sensations or movements are then perceived by the agent as involuntary, i.e. as a symptom, in order to reconcile the contradiction between conscious intention and action, and thus eliminate the prediction error at the highest conscious level.

For example, by inferring future probabilities from the prior ones, a person may have a prediction of pain in an injured limb even after it had healed. If such a prediction becomes abnormally salient (hyper-precise) it may fail correction by the prediction error from the peripheral sensory input. In that case, the pain experience will persist in the absence of the pain's source to fulfill the prediction. In order to suppress the prediction error at the conscious level, the person then will explain the pain as a symptom even in the face of evidence to the lack of organic pathology, believing that something is still wrong with the limb. Phantom limb syndrome may be regarded as an extreme example of this phenomenon, where pain is felt in the absent limb; except that the person cannot explain it away as an organic symptom.

Concerning functional *motor* symptoms Edwards and colleagues (2012) suggest that they are generated by abnormal predictions about *sensory consequences* of a movement, i.e. by hyper-precise proprioceptive prior beliefs. In other words, the person would make a movement for no other reason than to feel it. On a conscious level, the contradiction between the movement and its lack of intention will be explained away as a symptom. Hyper-precise proprioceptive prior beliefs can be negative, causing weakness or paralysis, or positive, causing involuntary movements or tremors.

Abnormally high precision of prior beliefs in conversion disorder is thought to stem from excessive somatically directed attention (Edwards et al. 2012). Such attention, either conscious or unconscious, may lead to increase of the beliefs' salience. Indeed, people with conversion disorder have a body-focused attentional bias (Robbins and Kirmayer 1991). In the model, a hyper-precise prior belief may originate from a relevant precursor event, e.g. a functional pain symptom may follow an injury, or from a random sensation that captures attention, since once a symptom is formed it will draw more attention, reinforcing the belief.

The Bayesian model of conversion disorder, while drawing obvious parallels with the Freudian view of conversion as a symbolic somatic manifestation of the unconscious conflict, has important differences from it. Most notably, it does not require symptom formation and maintenance to be related to an unconscious conflict. Indeed, the Freudian model has been challenged by failure to find a direct link between functional symptoms and psychological stressors in many patients (Stone and Edwards 2011). This has important treatment implications, because it means that "unconscious into conscious" may not necessarily provide a cure. Another significant difference is that the Bayesian model does not stipulate symbolic nature of functional symptoms. Although a symbolic link can often be inferred, it has been shown that in most cases functional symptoms can be traced back to *somatic* triggering events, e.g. physical injury (Stone et al. 2009) or psychological stress that is not directly relevant to the symptom (Rusch et al. 2001).

Treatment Options for Conversion Disorder

Currently, there is no standard effective treatment for conversion disorder, and despite reports of successful attempts, no treatment has an evidence-based status (for review see Nielsen et al. 2013). There have been reports of successful applications of cognitive behavioral therapy (LaFrance et al. 2009), hypnosis (Brooks et al. 2007), transcranial magnetic stimulation (Schönfeldt-Lecuona et al. 2006), strategic behavioral treatment (Shapiro and Teasell 2004), habit reversal training (Azrin and Nunn 1973), physical therapy (Ness 2007), and anti-depressants (Voon and Lang 2005). For conversion disorder comorbid with PTSD or trauma-related conditions, both trauma-focused (Boyd et al. 2016) and conversion disorder-focused (Neer et al. 2016) therapies have been reported effective.

Bayesian Model Approach

The Bayesian model of conversion disorder points at possible targets for therapy. It suggests that a prior belief driving the symptom should be the primary target. We can see two strategies for such targeting. One is top down, where a prior belief is targeted on a cognitive level through insight into its nature and likely connection to a possible trigger event and the entailing symptom. This is a standard cognitive/psychodynamic therapy approach. The approach may, however, prove limited, because, according to the model, functional symptoms are generated and sustained at a pre-cognitive and pre-conscious level by neural networks not directly accessible to consciousness. Still, altering conscious beliefs about functional symptoms to consider them an intended product of the brain's activity may be helpful. It may increase the prediction error at the conscious level, indirectly decreasing the lower level prior beliefs' precision.

The other strategy is a bottom up one, where prior beliefs are targeted at the level of the symptom, which can be achieved through behavioral interventions aimed at symptom displacement. The patient would practice a different behavior that would draw significant attention and would entail proprioceptive sensations different from the ones predicted by the pathological prior belief. The goal is to generate a

salient enough prediction error to affect the target belief. For that to happen, the belief needs to be activated at the same time as the substitute behavior, which can be achieved by a matching context. For example, if a socially stressful situation is known to exacerbate a functional symptom, the substitute behavior needs to be practiced in such situations or/and in anticipation of them. Unlike habit reversal training (Azrin and Nunn 1973), this approach does not require the substitute behavior to be incompatible with the target one, thus placing fewer constraints on behavioral intervention and potentially avoiding resistance.

In practice, the bottom up strategy is hardly possible without at least some top down cognitive processing, so that the patient understands the rationale for the bottom up intervention. These considerations suggest an integrative cognitive behavioral approach for targeting abnormally precise prior beliefs that we call *strategic modification of priors*, whose core intervention is *strategic symptom displacement*.

In this report, we describe a naturalistic case study of PTSD comorbid with a conversion disorder, where resolution of PTSD failed to relieve the functional motor symptom, which was subsequently targeted and improved with strategic symptom displacement.

Methods

Participant

The participant (henceforth Participant) was an active duty serviceman in the US military. He was a 27 year old married man of a mixed race (Asian/Caucasian) with no children, living with his wife of 3 years. His military career spanned 8 years of uninterrupted service and included five deployments: two combat deployments, one combat-related deployment on a training mission, and two non-combat ones.

Participant's first mental health visit occurred in 2014 during his last (non-combat) deployment overseas. Since then he received 2.5 years of treatment in the military mental health system. He initially presented with complaints of high stress-related anxiety, being easily startled, and hypervigilance. The immediate reason for his visit was multiple episodes of "freezing," being unable to move when spotting objects reminding him of combat danger, e.g. a deflated basketball. Such episodes as well as being easily startled felt embarrassing to him. The initial diagnosis was Anxiety Disorder NOS.

In follow up sessions with the psychiatrist and therapist, his diagnosis of PTSD was established. Accordingly, Participant engaged in comprehensive trauma-focused therapy. During that time, Participant also was manifesting short episodes of involuntary head and shoulder movements (he called them "tic") that he had developed before. With time, they grew into an uncontrollable habit and became his main concern.

In 2015, Participant returned to his home duty station in the US and continued his care in our department. His "tic" increased in frequency, intensity, and duration; he then also developed episodes of whole body seizure-like convulsions, which could last up to an hour. At this point, Participant was referred to neurology. A comprehensive neurological assessment followed, and the results showed no abnormalities. The neurologist then established a diagnosis of motor conversion disorder.

Participant continued his care with the psychiatrist, neurologist, and therapist. Although his therapy started as a trauma-focused approach, as at his previous duty station, Participant's "tic" was gradually moving into the treatment's focus. At that time, Participant had to change therapist for administrative reasons. This is when he started therapy with the therapist in this case study and continued until retirement from the military.

Assessment

Diagnoses

The diagnosis of PTSD (*DSM-5, Diagnostic and Statistical Manual of Mental Disorders*, 5th edn; American Psychiatric Association [APA] 2013) was established by Participant's first therapist in a structured interview: for criterion A 1, 2, 3, Participant endorsed exposure to multiple explosions around him, death of his friend by an improvised explosive device, and witnessing a convoy vehicle blown up. For criterion B 1, 3, 4, 5, he endorsed recurrent involuntary memories of the death and explosions, triggered episodes of "freezing," intense anxiety, and physiological arousal caused by the triggers. For criterion C 1, 2, he endorsed avoidance of reminders or discussions about combat (including in therapy), and avoidance of crowds. For criterion D 2, 3, 4, 5, 6, he endorsed suspecting bad intentions in people, guilt for his friend's death, social withdrawal, and emotional detachment reported by his wife. For criterion E, he endorsed high irritability, hypervigilance, high startle response, and decreased concentration. The condition lasted over a month, caused significant distress, impaired performance at work and in interpersonal relationships, and was not due to medical problems or substance use.

The diagnosis of conversion disorder with motor movements was established by Participant's neurologist by excluding neurological pathology (criterion B, DSM-5) for his motor dysfunction (criterion A, DSM-5). His neck, brain, and spine imaging, EEG, and laboratory tests were unremarkable. His symptoms did not manifest in a consistent pattern, since they would stop in certain settings such as shooting at the range, dog training (Participant's private

business), and could be modified in therapy sessions (as described below), thus satisfying criterion C (DSM-5). Participant's motor symptoms caused him significant distress in the form of embarrassment and further social withdrawal, as well as exacerbation of his neck pain. He was relieved of his work duty and his driver's license was suspended. These impairments satisfied criterion D (DSM-5). Participant did not meet the criteria for tic disorder due to the adulthood onset.

Malingering was ruled out for several reasons. Participant's motor symptoms were similar in different settings, as observed by multiple providers and video-recorded by his wife. His symptoms were seen outside of medical offices, when Participant was unaware of being observed. The change in the symptoms' intensity did not correlate with the timeline of his disability evaluation process, thus making it unlikely that the symptoms were driven by secondary gains.

Psychometric Measures

To monitor the dynamics of Participant's psychological symptoms we used military-wide screening and monitoring self-report measures administered at the check-in by the receptionist. They included the PTSD symptoms check list PCL-5 (Wortmann et al. 2016), the patient health questionnaire for depression PHQ9 (Kroenke et al. 2001), and the generalized anxiety questionnaire GAD7 (Spitzer et al. 2006). Their military-wide use allowed for continuity of monitoring through different treatment facilities over the whole duration of treatment.

We did not have any objective measure for Participant's motor symptoms and based our assessment on his self-report and observation by medical providers. They were rated as severe (multiple times a day and pronounced in sessions), moderate (isolated incidents in and outside sessions), mild (isolated incidents in stressful situations and absent from sessions), and absent (either in or outside session). Participant's motor symptoms were subdivided into head and shoulder movements and whole body convulsions.

Treatment

Pharmacological Intervention

Initially, Participant declined an offer of medications and chose to only engage in psychotherapy. He, however, changed his mind later, as his symptoms increased. Participant was tried on multiple medications including SSRI, anticonvulsants, anxiolytics, and an anti-psychotic. There were frequent changes of the regimen due to his complaints of side effects and lack of significant benefits. In the end, all medications were discontinued.

Trauma-Focused Therapy

Since trauma-focused therapy is not the focus of this report, we will describe the work by the first two therapists involved in Participant's care only briefly with a more detailed account of the last therapist's intervention. After having been diagnosed with PTSD, Participant's therapist offered a cognitive processing therapy (CPT) course (Resick and Schnicke 1993), which was augmented by a 2-week intensive outpatient program comprising group CPT as well as combat stress and coping skills training groups.

A year later, after Participant came to our clinic, his next therapist started with mindfulness and relaxation training and then attempted eye movement desensitization and reprocessing (EMDR) therapy (Shapiro 2001). Both had to be discontinued, since they would trigger Participant's motor symptoms culminating in whole body convulsions during the session. Instead, another course of CPT followed.

The last therapist started with confirming the diagnosis of PTSD. Participant endorsed most of the original symptoms, while noting a decrease in their intensity and remission of some. The therapist suggested another trial of EMDR but with tactile stimulation ('tappers'), since eye movements were still exacerbating his 'tic.' The standard eight phase protocol was used (Shapiro 2001). Memories of several combat events were targeted: a firefight, a raid on a house, an explosion, a military truck blown up. In total, four EMDR sessions were conducted.

The treating therapists were a PhD level psychologist and master's level licensed clinical social workers, all with specialized training in CPT and EMDR. The last course of EMDR was conducted by an EMDRIA-certified therapist.

Conversion Disorder-Focused Treatment

Interventions we used for Participant's functional symptoms do not constitute an established protocol and will be briefly delineated here, whereas a detailed description follows in the result section for the ease of following the interventions' rationale in a context of the therapeutic process and history. Once Participant's PTSD symptoms subsided, and a movement disorder specialist confirmed the conversion disorder diagnosis, Participant and the therapist decided to focus on his functional symptoms and started strategic modifications of priors. It included a cognitive-educational component aimed at gaining more insight into the psychological nature of the motor symptoms; a cognitive-experiential component, where the therapist guided reality tests of the mind's effects on the motor symptom. Together, the cognitive work took six sessions and one and a half month. Despite gaining insight and accepting the psychological nature of his motor symptoms, Participant reported no significant change in their rate, nor was such change observed in session.

At that point, we started the third component, strategic symptom displacement, where Participant was guided to practice an alternative behavior: tying and releasing knots on a short rope in situations of high or anticipated stress. Upon completion of this component, the active phase of therapy ended, and soon thereafter all his medications were discontinued. Participant continued maintenance therapy for five more months mostly for monitoring and observation, and no new interventions were attempted in that period.

Results

Pharmacological Intervention

There appears to have been no major benefits from medications. None of them showed a clear effect on any of the psychometric scores, since their dynamics did not correlate with adjustments of Participant's regimen. He did not feel that any of the medications had a discernible effect on his motor symptoms. Of note, Participant was reluctant to medications from the beginning, which indicates a possibility of a nocebo effect.

Trauma-Focused Intervention

Participant was referred to mental health care by his general physician, who suspected a possibility of PTSD. In his initial sessions, Participant was reluctant to fully engage in treatment and accept the severity of his mental condition. He also refused medications. His initial scores on self-report measures where either below (PHQ9 = 1, GAD7 = 7) or close to (PCL-5 = 45) the accepted clinical significance: 10, for PHQ9 and GAD7, and 33–39 for PCL-5.

Once Participant engaged in therapy, his scores on all measures started rising, and by the time he was to leave his duty station they all were in the range of severe pathology (PCL-5 = 77, GAD-7 = 20, PHQ-9 = 23). He also referred to that time as especially stressful due to his work environment and separation from home and family.

Upon return to his home duty station, Participant entered the second half of his treatment that included several approaches before he engaged in a sustained trial of EMDR. The desensitization phase of the protocol (Shapiro 2001) exacerbated Participant's 'tic,' but he was able to de-escalate to the baseline. SUDs (subjective units of distress on a 0/10 Likert scale) never dropped to 0 during the desensitization phase, which was unsurprising, since Participant was hyperaroused due to the intensification of his 'tic.' However, by the end of the fourth session, the target memories did not arouse strong emotional response (SUDs = 2 as oppose to 7–8 at the beginning of the EMDR course). There was little need to work on negative cognitions, since after two courses of CPT and group CPT, Participant's cognitions appeared ecologically valid. By the end of the fourth session, Participant stated that his PTSD symptoms did not bother him "that much" anymore, and that he was much more concerned with his 'tic,' which had grown very prominent and caused him intense distress. At this point, his PTSD was considered in remission, since it no longer met the DSM-5 criterion of significant clinical distress.

Participant's scores decreased by that time to the level of moderate pathology (PCL-5 = 50, GAD-7 = 16, PHQ-9 = 15). We want to underscore that the improvement of depression, anxiety, and PTSD symptoms did not correlate with the rate of Participant's motor symptoms, and they (both head and shoulder, and whole body convulsions) remained at their highest. That prompted more neurological exploration, a referral to a movement disorder specialist, and trials of anti-convulsant and anti-psychotic medications.

Conversion Disorder-Focused Intervention

Gaining insight into the psychological nature of the 'tic' was accomplished by a 'connecting the dots' approach. The fact that the 'tic' had started as a reflexive "ducking" movement in response to explosions was connected to the properties of Patient's job during deployments as an explosive clearing specialist. As Participant 'swept' his metal detector, his whole body was constrained in a habitual sweeping motion, leaving the head as the only body part with a free range of motion responsible for observing the environment and keeping him safe. From that we concluded that his head movements could have been a focus of intense (mostly unconscious) attention. Participant then recalled that the 'tic' increased after he developed neck pain after a minor injury in a 'log race' while on vacation. The pain could serve to amplify the attention allotted to his neck. Participant also acknowledged that the 'tic' intensified in stressful, especially social, situations and was amplified by self-consciousness, again pointing to a potential effect of self-directed attention on his motor symptoms. Although these bits of information provided no straightforward explanation for the 'tic,' they appeared to have relaxed Participant's belief in the strictly autonomous neurological nature of his motor movements and reconciled him with a possibility of them being generated by his mind as a learned reaction to stress. It also helped him accept the diagnosis of conversion disorder.

The second, cognitive-experiential, component was implemented through reality testing. The therapist made use of the fact (observed by the previous therapist) that self-directed attention and mindful awareness had an amplifying effect on the 'tic.' The therapist would direct Participant's attention to his head movements and sensations, thus triggering their increase, and then would direct his attention away, using grounding techniques, helping de-escalate him back to the

baseline. The effect of this intervention was a sense of some control over the movements, since they could be up- and downregulated with the therapist's guidance. The therapist also made use of the fact that Participant's 'tic' would stop when he was involved in a fine motor task. The therapist asked him to make drawings, which he did well and which would be impossible with his head moving. He also encouraged Participant to shoot at the range, where he had no problem hitting the target, which would also be impossible with the 'tic.' These activities seem to have furthered Participant's belief that his head movements could be at least partially controlled by his mind. However, despite these cognitive gains, Participant's motor symptoms were not abating.

The third, behavioral, component was accomplished by designing a behavior alternative to the 'tic'. The behavior of choice was tying and releasing knots on a short rope that Participant would carry everywhere in his pocket. This particular behavior was chosen for two reasons. For one, it was a purposeful fine motor activity highly represented in the somatosensory cortex, since hands have a large somatosensory representation. The other reason was that Participant had learned making knots before, was skillful and enjoyed doing it. Finally, this behavior would meet the requirement of introducing a prediction error, where instead of the expected proprioceptive signal from head and shoulder following a stress response, a sustained signal from hands will ensue. After the first week of practicing this behavior, Participant reported decrease of his motor symptoms, and within 2 weeks, he reported a substantial reduction of the symptoms and a complete elimination of whole body convulsions. From that time, he would only experience occasional episodes of the 'tic' in stressful situations, and the whole body movements did not came back. In session, he did not display head movements and was able to focus attention on his neck and shoulder without triggering the 'tic.' His other symptoms continued to improve to the mild to minimal range (PCL-5 = 33, GAD-7 = 12, PHQ-9 = 7).

After the end of active therapy during the maintenance phase, Participant continued reporting isolated "flare ups" of head movements in stressful situations. Close to the end of treatment he also reported isolated instances of spontaneous symptom substitution in the form of intense sweating spells or brief hip muscle weakness, manifesting as a wobbling gate. His psychometric scores also slightly increased (PCL-5 = 42, GAD-7 = 18, PHQ-9 = 13). This was a time of change for him; he was retiring from the military and moving to another state with all the stressors associated with such transition.

Discussion

The Active Ingredient

The reported case spanned 30 months, 25 months of which Participant was in active treatment with five more months of maintenance/monitoring therapy. Participant has tried many therapeutic approaches in several modalities. In such a complex treatment history it is challenging to single out active ingredients, especially considering such a multi-faceted intervention as psychotherapy, let alone a protracted one with several therapists. Therefore, the case was by no means 'clean.' In the end, Participant's symptoms stabilized. He no longer met the criteria for PTSD, his motor symptoms were reduced to occasional and brief stress-induced "flare ups," and he expressed satisfaction with his overall progress. Moreover, his functioning significantly improved. He was starting driving again, worked out regularly, productively worked in his successful private business, actively networked, and was setting up a nonprofit organization.

While Participant's symptoms of depression, anxiety, and PTSD were gradually decreasing over the 9 months, his motor symptoms precipitously improved within 1 month, and that improvement closely followed the strategic symptom displacement. The time link and the fact that it was an intervention most directly targeting the motor symptoms may indicate cause and effect.

Symptom Formation

Single case study is limited in terms of drawing conclusions beyond the description of its particulars, which inevitably leaves such conclusions *highly speculative*. However, the interventions in this case were designed based on a theoretical model, making it imperative to speculate about the relationship between the hypothesis and the observed effect. From the Bayesian perspective (Edwards et al. 2012), a functional motor symptom results from an abnormally precise prior belief about the proprioceptive consequence of the motor movement (or lack thereof). The hyper-precision results from abnormally high somatically directed attention and makes the belief unsusceptible to prediction errors at the highest (conscious) level of the motor control network. Functional motor symptoms then serve to 'fulfill' the belief, and are interpreted by the agent as involuntary, i.e. a symptom.

In light of this theory, several facts about the development of Participant's conversion disorder appear relevant. His 'tic' reportedly originated as a "ducking" motion at the sound and impact of explosions and initially appeared

a manifestation of his hyper-active startle response. Later, when others noticed it, he became aware of inappropriateness of this reaction and grew embarrassed of it. The more embarrassed he grew, the less controllable these movements appeared to him. Participant also reported that he had been aware of the range of his head movement, when searching for explosives, since the rest of his body was constrained by the sweeping motion of his metal detector, leaving the head relatively free to move and observe the (dangerous) environment. After his combat deployments, Participant became more aware of his 'tic' and its increasing frequency following a slight injury to his neck. One of the consequences of the injury was that the 'tic' would exacerbate his neck pain. These observations point to a possibility of an emotion-mediated hyper-focus (likely both conscious and unconscious) on the neck and head movements and sensations. We believe that such focus could have led to a hyper-precise prediction of proprioceptive feedback from the head movement, which would have been 'fulfilled' by the corresponding movement.

We also think that because of the initial link between the head movement and danger, Participant's *attention* to his head and neck was an overlearned response easily triggered by stress. The 'tic' itself, however, was unlikely to be directly triggered by stress, since relaxation exercises along with guided self-directed attention exacerbated it, while activities requiring attention directed away from self, even stressful ones like training dogs in apprehension (Participant's business) would eliminate the 'tic.' Besides, Participant himself noticed that his 'tic' would always "flare up" in situations making him self-conscious. These observations are in line with the proposed role of attention in mediating formation and maintenance of functional symptoms.

To explain generalization of Participant's head 'tic' to the whole body convulsions we took notice of the fact that they mostly happened either at night, when he was going to sleep, or in therapy sessions. Both situations combined on the one hand his thoughts and memories of combat experience (he used to have nightmares about combat and was often anticipating them) and self-directed attention on the other, given his insomnia at night and therapist's guided attention in session. In both situations, his whole body was disengaged from any purposeful behavior. We believe that a combination of a stressful cognitive content, the idle body, and self-directed attention could be enough to generalize Participant's hyper-precise proprioceptive prior beliefs beyond his neck and shoulder groups of muscles.

The Bayesian model provides a plausible scenario for the presented case, as it accounts for the history and circumstances of the symptom development and manifestations. Moreover, it also provides a plausible causative link between Participant's PTSD and functional symptoms. Although we must strive for the most parsimonious theory, we do not find an alternative hypothesis that would accommodate the complexity of the case in a significantly more parsimonious way.

PTSD as a Disorder of Predictive Coding

Besides conversion disorder (Edwards et al. 2012), the Bayesian model has been implicated in psychotic (Powers et al. 2017), autistic (Pellicano and Burr 2012), mood (Badcock et al. 2017), personality and pain perception (Moutoussis et al. 2014) disorders. According to this view, e.g. 'sickness behavior' in depression fulfills the prior beliefs triggered by interoceptive endocrine signals released by stress, or negatively biased prior beliefs about self-worth are triggered in borderline personality patients by innocuous social stimuli and are fulfilled by self-loathing or paranoid reactions.

PTSD seems to readily align with the model as well. By definition (DSM-5), PTSD develops in response to a traumatic event, i.e. an event that shatters previously held beliefs. We expect that such experience leads to their abrupt and drastic recalibration, which happens too quickly for an accurate incremental adjustment through a series of prediction errors. Instead, the new beliefs precipitously grow hyper-precise (and consequently rigid) in predicting and inferring danger, thus generating a rigid, maladaptive model of self and the world. This would explain such hallmark symptoms of PTSD as hypervigilance, hyperarousal, avoidance, re-experiencing and re-enacting of the trauma, unrealistic negative beliefs about self and the world.

Within the Bayesian account, we see the same etiology behind Participant's PTSD and functional motor symptoms, which brings into question whether his 'tic' should be considered a comorbid conversion disorder or part (extension) of his PTSD. The existence of a functional symptom alongside mood or anxiety symptoms could indicate an incomplete 'conversion' or, in agreement with the Bayesian model, the auto-generative nature of functional symptoms. In the latter case, functional symptoms may be expected to develop ahead of affective ones, since otherwise affective symptoms are likely to recruit attention away from the somatic target. Of note, Participant's 'tic' started before he became aware and acknowledged his anxiety symptoms.

According to Bayesian model of psychopathology, dysfunction of predictive coding may be viewed as a universal principle of symptom formation, which, in turn, suggests a possibility of a universal strategy for psychotherapy. Such a strategy would involve targeting pathogenic prior belief probability distributions in order to better align them with the actual likelihood distributions (in less formal terms: better align the brain's working model of the world with reality). We call this approach *strategic modification of priors*, where strategic symptom displacement is the core intervention, because of its potential to generate prediction errors

at the symptom's level. Examples of such displacement could include behavioral activation for sickness behaviors in depression, forced limb movement and its visual illusion for functional paralysis, or practicing fear-irrelevant behavior upon exposure to a trigger in PTSD.

Acknowledgements This research did not receive funding from agencies in the public, commercial, or not-for-profit sectors. We want to thank our patient, who agreed that his treatment case be made public. MVC joined the project at the stage of conceptualizing and writing the paper and had no part in the clinical work.

Compliance with Ethical Standards

Conflict of interest Valery Krupnik and Mariya V. Cherkasova declare that they have no conflict of interest.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington: American Psychiatric Publishing.

Azrin, N. H., & Nunn, R. G. (1973). Habit-reversal: A method of eliminating nervous habits and tics. *Behaviour Research and Therapy, 11*, 619–628. https://doi.org/10.1016/0005-7967(73)90119-8.

Badcock, P. B., Davey, C. G., Whittle, S., Allen, N. B., & Friston, K. J. (2017). The depressed brain: An evolutionary systems theory. *Trends in Cognitive Sciences, 21*, 182–194.

Boyd, B., Rodgers, C., Aupperle, R., & Jak, A. (2016). Case report on the effects of cognitive processing therapy on psychological, neuropsychological, and speech symptoms in comorbid PTSD and TBI. *Cognitive and Behavioral Practice, 23*, 173–183.

Brooks, J. L., Goodfellow, L., Bodde, N. M. G., Aldenkamp, A., & Baker, G. A. (2007). Nondrug treatments for psychogenic nonepileptic seizures: What's the evidence? *Epilepsy & Behavior, 11*, 367–377. https://doi.org/10.1016/j.yebeh.2007.05.007.

Clark, A. (2013). Whatever next? Predictive brains, situated agents, and the future of cognitive science. *Behavioral and Brain Sciences, 36*, 181–204.

Edwards, M. J., Adams, R. A., Brown, H., Pareés, I., & Friston, K. J. (2012). A Bayesian account of 'hysteria'. *Brain, 135*, 3495–3512. https://doi.org/10.1093/brain/aws129.

Friston, K. (2010). The free-energy principle: A unified brain theory? *Nature Reviews Neuroscience, 11*, 127–138. https://doi.org/10.1038/nrn2787.

Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2001). The PHQ-9. *Journal of General Internal Medicine, 16*, 606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x.

LaFrance, W. C., Miller, I. W., Ryan, C. E., Blum, A. S., Solomon, D. A., Kelley, J. E., & Keitner, G. I. (2009). Cognitive behavioral therapy for psychogenic nonepileptic seizures. *Epilepsy & Behavior, 14*, 591–596. https://doi.org/10.1016/j.yebeh.2009.02.016.

Moutoussis, M., Fearon, P., El-Deredy, W., Dolan, R. J., & Friston, K. J. (2014). Bayesian inferences about the self (and others): A review. *Consciousness and Cognition, 25*, 67–76.

Neer, S. M., Trachik, B., Munyan, B. G., & Beidel, D. C. (2016). Comprehensive treatment. *Clinical Case Studies, 15*, 343–359. https://doi.org/10.1177/1534650116643401.

Ness, D. (2007). Physical therapy management for conversion disorder: Case series. *Journal of Neurologic Physical Therapy, 31*, 30–39. https://doi.org/10.1097/01.NPT.0000260571.77487.14.

Nielsen, G., Stone, J., & Edwards, M. J. (2013). Physiotherapy for functional (psychogenic) motor symptoms: A systematic review. *Journal of Psychosomatic Research, 75*, 93–102.

Pellicano, E., & Burr, D. (2012). When the world becomes 'too real': A Bayesian explanation of autistic perception. *Trends in Cognitive Sciences, 16*, 504–510.

Powers, A., Mathys, C., & Corlett, P. (2017). Pavlovian conditioning-induced hallucinations result from overweighting of perceptual priors. *Science, 357*, 596–600.

Resick, P. A., & Schnicke, M. (1993). *Cognitive processing therapy for rape victims: A treatment manual*. Newbury Park: Sage.

Robbins, J. M., & Kirmayer, L. J. (1991). Cognitive and social factors in somatization. In J. M. Robbins & L. J. Kirmayer (Eds.), *Current concepts of somatization: Research and clinical perspectives* (Vol (31, pp. 107–141). Arlington, VA: American Psychiatric Association.

Rusch, M. D., Morris, G. L., Allen, L., & Lathrop, L. (2001). Psychological treatment of nonepileptic events. *Epilepsy & Behavior, 2*, 277–283. https://doi.org/10.1006/ebeh.2001.0180.

Schönfeldt-Lecuona, C., Connemann, B. J., Viviani, R., Spitzer, M., & Herwig, U. (2006). Transcranial magnetic stimulation in motor conversion disorder: A short case series. *Journal of Clinical Neurophysiology, 23*, 473–476.

Shapiro, A. P., & Teasell, R. W. (2004). Behavioural interventions in the rehabilitation of acute chronic non-organic (conversion/factitious) motor disorders. *The British Journal of Psychiatry, 185*, 140–146. https://doi.org/10.1192/bjp.185.2.140.

Shapiro, F. (2001). *Eye movement desensitization and reprocessing: Basic principles, protocols and procedures* (2nd ed.). New York: Guilford Press.

Spitzer, R. L., Kroenke, K., Williams, J. B., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine, 166*, 1092–1097.

Stone, J., Carson, A., Aditya, H., Prescott, R., Zaubi, M., Warlow, C., & Sharpe, M. (2009). The role of physical injury in motor and sensory conversion symptoms: A systematic and narrative review. *Journal of Psychosomatic Research, 66*, 383–390. https://doi.org/10.1016/j.jpsychores.2008.07.010.

Stone, J., & Edwards, M. J. (2011). How "psychogenic" are psychogenic movement disorders? *Movement Disorders, 26*, 1787–1788. https://doi.org/10.1002/mds.23882.

Voon, V., & Lang, A. E. (2005). Antidepressant treatment outcomes of psychogenic movement disorder. *The Journal of Clinical Psychiatry, 66*, 1529–1534. https://doi.org/10.4088/JCP.v66n1206.

Wortmann, J. H., Jordan, A. H., Weathers, F. W., Resick, P. A., Dondanville, K. A., Hall-Clark, B.,… Hembree, E. A. (2016). Psychometric analysis of the PTSD Checklist-5 (PCL-5) among treatment-seeking military service members. *Psychological Assessment, 28*, 1392–1403. https://doi.org/10.1037/pas0000260.