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Bypassing Shame and Conversion Disorder

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ABSTRACT

We report a case of conversion disorder (partial aphonia) that was successfully treated with speech therapy. During the one year duration of this illness, the patient regained transiently (minutes) her normal speech on a few occasions, independently of concomitant pharmacological interventions. One year after recovery she developed aphonia for the second time, which responded again to speech therapy, although the response rate was slower. Several mechanisms of conversion disorder are reviewed as specifically applied to this case. The possible role of pertinent shame mechanisms in the conversion pathology is discussed.

FOCUS POINTS

- The prognosis of conversion disorder is poor.
- Comorbidities of conversion disorder with other psychiatric conditions are well documented.
- Psychiatric treatment including psychotherapy, hypnotherapy and pharmacotherapy have been used with a limited success in conversion disorder.
- Better understanding of shame mechanisms in conversion disorder are important in the recovery process.
- Strategical behavioral interventions are likely to result in a speedier resolution of the conversion symptoms.

INTRODUCTION

Conversion disorder is a condition where patients present with a variety of apparent neurological symptoms that do not have a clear neurological explanation. Conversion disorder has been the focus of speculation over the last few millennia.[1] While Freud is credited with coining the term conversion disorder[2] the diagnosis gained notoriety at the turn of the 20th century at the Salpetriere Hospital in Paris, where the most famous neurologist of all times was introducing the public to clinical hysteria case presentations. It is known that for a few months in 1865 and 1886 Freud attended Professor Charcot's presentations and some historians even speculate that Charcot's investigations into the causes of hysteria may have been the seed responsible

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for Freud's own conversion from neurology to psychoanalysis.[3] Due to a lack of clear neurological pathology, conversion disorder is presumed to be the result of an underlying psychological mechanism with the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* classifying it as a somatoform disorder[4] and the *International Classification of Disease, Tenth Edition* classifying it as a dissociative disorder. The different classifications illustrate the ongoing debate about the underlying etiological mechanisms of the disorder.[5] The conversion symptoms are not considered voluntary responses to a known stressor but rather unintentional substitutions of psychological symptoms with somatic manifestations. Most of the hypotheses of conversion disorder try to explain the development of the condition as opposed to the mechanisms that allow the restoration of the previous state.[6] The rate of misdiagnosing this condition (diagnosing conversion disorder when a neurological cause is ultimately determined) has declined over the years from 29% in the 1950s to 4% today[7] partly due to improved diagnostic accuracy and a multidisciplinary approach.[8]

The prognosis of conversion disorder is poor. In one prospective study[9] of 73 patients evaluated 10 years after a diagnosis of conversion disorder was made, 11 patients eventually received a neurologic diagnosis explaining their initial presentation, while 30 patients had no relief of their original symptoms. Shapiro[10] reported that standard behavioral treatment is effective in "acute" patients but not in chronic patients.[3] For nonresponders with chronic illness, strategic behavioral interventions (eg, telling the patients going through treatment that full recovery is proof of organic etiology) was effective in >50% of patients compared to <5% response to standard behavioral interventions.[10] Psychiatric treatment,[11] including psychotherapy and pharmacotherapy,[12] hypnotherapy,[13] and transcranial magnetic stimulation,[12] have all been proposed as methods of treatment for conversion disorder. Comorbidities of conversion disorder with other psychiatric conditions are well documented[14] and should be considered in treatment planning.

Despite a burgeoning literature of brain imaging studies of conversion disorder the neurological circuitry underlying conversion symptoms is yet to be understood. Two competing hypotheses have been proposed. The first hypothesis is conversion symptoms are the result of an excessive inhibitory process. According to this hypothesis the failure to move is the result of decreased activity in the executive motor,[15-18] sensory,[19] and visual areas[20] in patients with hysterical paralysis, hysterical anestesia or blindness respectively. The decreased activation is presumably secondary to excessive activation in the medial, prefrontal, orbitofrontal, and anterior cingulate inhibitory circuit.[15]

The second hypothesis is that conversion symptoms are the result of a direct failure in internal initiation networks, as suggested by hypoactivation in the left dorsolateral prefrontal cortex in conversion patients.[18] In addition, a striatothalamic dysfunction has been proposed based on decreased basal ganglia and thalamic volumes in patients with conversion disorder[21] as well as basal ganglia functional abnormalities. Basal ganglia dysfunction has been suggested as part of either a basal ganglia-insular-lingual gyri-inferior frontal cortex dysfunction,[22] a complex circuit which is purportedly responsible for selecting and/or deciding on short term memory based information or a striatothalamocortical circuit dysfunction,[23] resulting in sensorimotor function and voluntary movement deficits.

There are a number of reasons for such apparently divergent findings: different studies used different brain imaging modalities (single photon emission computed tomography, positron emission tomography, and functional magnetic resonance imaging [fMRI]), diagnostic heterogeneity (sensory or motor conversion symptoms, different symptoms duration), small sample sizes (with a likely increase in sample heterogeneity even for apparently similar subjects and magnification of single subjects outliers effects), methods differences (whole brain based statistical parametrical mapping versus region of interest analysis, patients-controls versus intrasubject comparisons). Such confounders should be considered in the design of future imaging studies aimed at defining the neural circuitry of conversion symptoms.

CASE REPORT

Mrs. A is a 43 year-old white married woman, with one grown child. She has been working at a stable job in a local restaurant as a waitress for the last 15 years. She has a history of bipolar disorder for which she has been treated with olanzapine 5 mg at bedtime and sertraline 50 mg in the morning for the 6 years prior to the first psychiatrist evaluation in 2002. Her compliance with the pharmacological interventions and with appointments with her psychiatrist was sporadic for the first few

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years. In 2006, while taking lithium 300 mg TID and fluoxetine 40 mg in the morning for over 6 months, she suddenly lost her ability to speak in a normal tone. Instead she began to communicate in a whispering voice. After extensive testing, including vocal cords biopsy, Mrs. A's primary care physician and a consulting oto-rhino-laryngologist independently concluded that there was no organic cause underlying her presentation. Several months later a neurological consult also ruled out a neurological explanation for the patient's presentation. A structural brain MRI was normal. As a result of this essentially negative work-up Mrs. A was diagnosed with conversion disorder. Careful questioning failed to reveal any clear triggers for Mrs. A's the condition.

Mrs. A reported a troubled marital history: her ex-husband was extremely violent and was sexually and physically abusive toward Mrs. A. At the time of her presentation Mrs. A had been married for 7 years to her second husband. There were numerous arguments at home, which on several occasions escalated to the point that the police was called by the neighbors. Not surprisingly, after Mrs. A lost her voice, the loud arguments ceased. The patient reported that she had been more submissive during the first part of her current marriage, but that that has changed in the latter years as she became more verbal, in an attempt, albeit only minimally successful, to prevent her husband's verbal, and occasionally physical abuse. Mrs. A reported a chaotic childhood: between 8–12 years of age she was raped by two cousins and one uncle, verbally abused by her mom, and neglected by her father. As a child Mrs. A felt that she could not rely on anyone with the exception of a geographically distant grandmother who would visit her occasionally. The patient was a very shy child until her senior year in high school, when she started to fight with classmates "before I was attacked". This pattern continued through her life, occasionally getting her in trouble with the law as she would be quick to start an argument when she assumed others were going to attack her.

Mrs. A's past medical history was significant for hypertension and peptic ulcer disease. She was status post C-section and G1P1Ab0.

Following the conversion disorder diagnosis, the patient was treated with fluoxetine 80 mg PO qam (increased from 40 mg), lithium carbonate 300 mg PO BID and counseling, including family therapy. The symptoms of depression remitted. Even though she had some insight into her condition (recognizing that her voice is at a much lower pitch than of other people), this insight did not caused her an emotional reaction and no change in her tone of voice was noted. As part of her treatment, the patient was instructed to keep a log of voice variations. Several interesting facts emerged as a result of this process. Mrs. A reported that after being prescribed Vicodin (5/500) for a dental procedure, her voice returned to normal for several minutes (two episodes). There were a few other occasions when her voice also returned to normal for periods of minutes (three episodes). These spontaneous recovery episodes had the following similarities: she was in the presence of friends; her mood was good, with no immediate plans or concerns for the remaining of the day. As she was discussing events with minimal emotional valence, her friends pointed out that her voice returned to a normal tone and volume for a short time, then resumed its whispering quality just as they started to mention it.

After one year of therapy she was referred to a speech therapist. The treatment included weekly sessions of phonetic training. After the sessions, Mrs. A was instructed to continue to practice the exercises taught in the session. After 4 weeks of treatment her symptoms gradually decreased and at 8 weeks resolved. At an 11 months follow up there were no further speech problems. Interestingly, following recovery the patient reported that her friends and husband started to complain that she talks too much and eventually the police were called again due to loud verbal arguments.

One year after the speech therapy, the patient was arrested, after the police could not redirect her after responding to a disturbance call. While in jail, the patient got upset with the officer because she did not get her prescribed medications and because the temperature in her cell was too low. The argument continued and then suddenly the patient lost her voice again because of "the air conditioning". A few months later she did respond again to speech therapy, but only after twice as many speech therapy sessions.

DISCUSSION

Mrs. A's symptoms have resolved with speech therapy. The fact that Mrs. A's symptom's resolved with speech therapy may suggest the case for a physiological, cause. However, a comprehensive review of speech therapy and language therapy interventions after a stroke[24] concluded that these interventions did not show clear effectiveness or ineffectiveness.

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What is the purpose of continued symptoms if most of the identifiable psychological stressors leading to the pathology have been addressed? It is possible that the cost of recovery appears to be greater than the cost of remaining ill. The underpinning of the recovery process could rely heavily on shame mechanisms. According to Tomkins'[25] theory of affect, perceived trauma occurs when the density of negative affect (distress, shame, anger, fear, etc.) is beyond the patient's ability to modulate such a sustained intensity. Mrs. A was entangled in continuous marital quarreling, which escalated from verbal to physical violence and occasionally required the intervention of the police. We propose that that the anger generated in those conflicts needed to be repressed and converted because the shame of public exposure added to the distress that the family was already experiencing. In this way, Mrs. A temporarily redirected her partner's and caretakers' attention away from the marital conflicts to concern about her condition.

During Mrs. A illness, her voice returned to "normal" at what appeared to be random times. When those episodes occurred, the patient did not notice the change in voice but was informed by others. Though normal voice has been present in a few of the occasions when the patient took an opioid for pain, the return of her voice was also reported during times when she felt minimal concerns and contempt. This suggests a more complicated cause-effect relationship. Furthermore, the suggestion of an endorphin excess with transient symptom resolution does not mean that there is a cause effect relation (because these occurrences happen at random times, and were non-reproducible in grossly similar situations).

Under the new circumstances a new script might have developed where the illness was not acknowledged and the new state (observed illness) served its purpose without fail. In this situation, the cost of not having the illness could expose one internally to feelings of inadequacy and shame. According to Nathanson[26] there are four possible ways to deal with shame: withdrawal, avoidance, attack self, or attack other. Nathanson explains that when we experience shame, we withdraw as an action of turning away from something that just a moment ago was very interesting. It represents all the ways we hide from sight and live with a diminished presence and visibility. In the Avoidance Pole we use methods that would allow us not to perceive information that will reduce self esteem (denial, reaction formation, etc.). Examples include: excessive use of cosmetic surgery, drugs, alcohol, wealth display, etc. In the Attack Pole there is a library of scripts directing the affect against oneself to protect from conflictual attachment to another close person. In the Attack Other Pole, Nathanson describes that when shame is experienced as a weakness of the self in relation to another person one defends against that perception by proving someone else even weaker than oneself. In the Attack Self Pole, shame produces a sense of alienation, of being shorn from the herd, as Nathanson says "such people tend to relate to others in ways that guarantee affiliation but at a reduced level of self esteem".

Thus, Mrs. A could have been using aphonia as a non-verbal way to express a powerful message such as "I am not the one who screams; I am voiceless and need to be taken care of". In this way, she succesfully, albeit temporarily, directed attention away from the marital conflicts to concern about her condition from her partner and other caretakers.

Behavioral interventions allowing reframing of the patient's condition as "genuine" and creating the premises for a saving face recovery strategy should be considered as treatment alternatives for conversion disorder. The small rate of success of traditional behavioral interventions could be caused by impairment of insight into the symptoms ("la belle indifference"). Insight into the symptoms is greatly diminished, possibly as a defense mechanism, and therefore could be considered to an extent as ego-syntonic with significant treatment implications.

We propose that diminished insight also plays a role in the symptom development in this case and shame might be a contributing factor in recovery. Indifference to symptoms, especially in motor conversion disorders, is likely to be neurologically different from the indifference seen with specific brain lesions. In conversion disorder patients with sensory loss, stimulation of the affected area did not produce contralateral somatosensory activation on fMRI, while bilateral stimulation did.[19] In another study,[16] patients with conversion paralysis had decreased activation in the hand area and no brain activation paired with movement inhibition was noted when compared with controls. This suggests a disorder of movement conceptualization. Feigned weakness does cause distinct cortex activation when compared with weakness in established conversion disorder.[27]

As the insight into the symptoms for patients

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with conversion disorder is poor (perception) the resulting understanding of the meaning of the illness (a priori knowledge) will also be inaccurate. At the same time, working on correcting perception or meaning of the illness with these patients had very limited therapeutic success.

CONCLUSION

In light of these considerations and Mrs. A presentation and response to treatment we propose that a different approach, using the route of a posteriori thinking (closed systems) might be a more effective intervention. Specifically, the conversion patients contemplating recovery face the cost of future invalidation, criticism and shame if their symptoms are explained away as entirely "mind based". It is not an accident that "hysteria" carries such negative connotations in our modern society. When the cost of recovery with its resulting shame exceeds the cost of maintaining the conversion symptoms, the patient might be stuck is a lose-lose situation. If maintaining symptoms appear as the lesser of the two evils, the patient is likely to forfeit recovery. Thus, an intervention that creates a new script (eg, strategic behavioral interventions) designed to enable the patients to create meaning based on the realities of their daily routines and without a need for recourse to extraordinary explanations or "excuses" is likely to result in a speeder resolution of the conversion symptoms. However, the same intervention might not work every time in the same patient (speech therapy took twice as long for the second treatment) because it loses the characteristics of a closed system and new a paradigm should be sought if necessary.

In Mrs. A's case, speech therapy, as a legitimate intervention promoting speech recovery, might have been just that Mrs. A was never at loss for words; only needed some other way to find her true voice. ***CNS***

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