

# NEUROLOGY

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*Neurology* 2010;74;190-191  
DOI: 10.1212/WNL.0b013e3181cb4ea8

**This information is current as of April 8, 2010**

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<http://www.neurology.org/cgi/content/full/74/3/190>

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# Conversion disorder

## Separating “how” from “why”

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*Neurology*® 2010;74:190–191

Patients with neurologic symptoms inconsistent or incongruous with structural disease are frequently encountered in neurologic practice and present diagnostic and therapeutic dilemmas. Also known as conversion or dissociative disorders, and “psychogenic,” “functional,” or “non-organic” symptoms, these symptoms may be debilitating and bewildering, and are often refractory to treatment. Patient with such problems should attract attention from neurologists for practical and theoretical reasons.

Since the late 19th century, psychoanalytic theory has dominated medical thinking about these symptoms. As patients with a presumed psychiatric disorder diagnosed by neurologists, they often end up with a bad deal from both neurologists and psychiatrists, who have difficulty knowing how to understand or treat the problem.<sup>1,2</sup> Unfortunately, neither specialty has successfully tackled the challenge posed by Freud a century ago of making the “mysterious leap from mind to body.”

In this issue of *Neurology*®, Voon et al.<sup>3</sup> report one of only a handful of physiologic studies attempting to make this “mysterious leap” and the first for a conversion movement disorder (rather than weakness or sensory disturbance). Studying 8 subjects with “clinically definite” conversion tremor, they compared functional MRI (fMRI) during conversion tremor to fMRI obtained while they were consciously trying to simulate their conversion tremor. Each subject was his or her own control. They demonstrated that the right temporoparietal junction (TPJ) was hypoactive and had a lower functional connectivity to sensorimotor cortex, cerebellar vermis, and parts of the limbic system during conversion tremor. Based on the notion that the TPJ produces a feed-forward signal to alert other parts of the brain to an impending movement, the authors hypothesize that a reduction in this signal makes the patient unaware of having consciously generated the movement.

Even if confirmed, this will not localize all conversion disorders, since they occur in a wide spectrum. Conversion disorders may cause positive symptoms, like tremor, seizure-like attacks, and dystonia, or negative symptoms, like paralysis, numbness, and blindness. They may be episodic or sustained, acute or chronic. The patients reported by Voon et al. all had a chronic conversion tremor of an unusual kind, which was intermittent and which the patients could trigger themselves. Ascribing TPJ hypoactivation to impaired self-agency—i.e., subjective sense of being responsible for one’s own movements—remains speculative since subjective agency was not directly tested in this study. Also, lower activation of the right TPJ during conversion tremor relative to voluntary tremor could also reflect a role of this region in attention and selective recruitment for a novel rather than a habitual motor behavior.

Other functional imaging studies of conversion disorders have reported a number of intriguing, although sometimes conflicting, observations, but methodologic problems abound.<sup>4</sup> These include small sample sizes and difficulties in comparing studies because of differences in symptoms, comorbidities, and research paradigms. In patients with chronic symptoms, it is hard to know whether abnormalities are a consequence or a cause of the symptoms seen. Even the basic question of whether the problem in conversion disorder is one of an active inhibition of normal voluntary movement pathways or a lack of activation of certain brain structures engaged in voluntary control remains uncertain. Within-subject comparisons, as performed in the Voon et al. study and another recently published study,<sup>5</sup> but with larger numbers of patients, would appear to be the best way forward.

The findings of this study may be one that patients are keen to hear. The study might be interpreted as showing a problem that is “all in the brain,” not “all in the mind” (and different from feigning as

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*Disclosure:* Author disclosures are provided at the end of the editorial.

well). But this is too simplistic a view. All behavior, physical or mental, is, at its base, a collection of electrochemical and neurohumoral expressions, so it should be apparent that actions and thoughts will ultimately be explicable in physiologic terms.<sup>6</sup> The history of conversion disorder and hysteria has been dogged by failed attempts at unifying explanatory theories and by our dualistic thinking about the mind and brain. It seems likely that this is a clinical problem that will require the application of multiple perspectives—biologic, psychological, and social—to be able to understand the symptoms of individual patients.

Currently, large numbers of patients with conversion symptoms are found to have “no neurologic disorder” by the neurologist and “no psychiatric disorder” by the psychiatrist.<sup>1</sup> The importance of physiologic studies lies in their exploration of the mechanisms of conversion symptoms—the “how”—as well as the vulnerabilities of those patients who get them—the “why.” In unveiling physiologic pathways associated with conversion disorder, studies like this may suggest new concepts in diagnosis and treatment. Perhaps more important, they may encourage more neurologists and psychiatrists to take an interest in a common, disabling, and fascinating clinical problem.

#### DISCLOSURE

Dr. Stone has received funding for travel from Sanofi-Aventis; has received speaker honoraria from GlaxoSmithKline and UCB; and receives research support from Remind (Edinburgh Neurology Research Fund). Dr. Vuilleumier serves as an Associate Editor of *Emotion* and of *Frontiers*

in *Human Neuroscience*. Dr. Friedman serves on scientific advisory boards for Teva Pharmaceutical Industries Ltd., EMD Serono, Inc., and ACADIA Pharmaceuticals; serves as Editor-in-Chief of *Medicine & Health/Rhode Island* and on the editorial boards of *Parkinsonism & Related Disorders* and *Neurology Reviews*; receives royalties from publishing *Making the Connection Between Brain and Behavior: Coping with Parkinson's Disease* (Demos Health, 2007); has received speaker honoraria from Teva Pharmaceutical Industries Ltd., AstraZeneca, GlaxoSmithKline, Boehringer Ingelheim, and Novartis; serves as a consultant to ACADIA Pharmaceuticals, Teva Pharmaceutical Industries Ltd., EMD Serono, Inc., and Biogen Idec; serves on speakers' bureaus for Pharmaceutical Industries Ltd., GlaxoSmithKline, and Boehringer Ingelheim; and receives research support from Teva Pharmaceutical Industries Ltd., GlaxoSmithKline, Boehringer Ingelheim, Pfizer Inc., Cephalon, Inc., ACADIA Pharmaceuticals, EpiVax, Valeant Pharmaceuticals International, the NIH [R01 HG002449 (Site PI), R01 NS36630 (Site PI), U10 NS050095 (Site PI), and R01 NS037167 (Site PI)], and the Michael J. Fox Foundation.

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