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A case of a psychogenic "jumpy stump"

Movement disorders developing in the stump after limb amputation are well described.¹ These movements are varied, consisting of jerking, tremulousness or spasms of the stump, and are often associated with severe stump pain.¹ The so-called "jumpy stump" is used as one example to support the concept that peripheral injury can induce movement disorders.² In our experience, the widespread acceptance of this concept, with little direct scientific evidence to support it, often results in the uncritical application of a diagnosis of a peripheral trauma-induced movement disorder without a careful analysis of other diagnostic possibilities, including that of a psychogenic movement disorder. To further emphasise this possibility, we report a case of a psychogenic jumpy stump.

Case report

A 42-year-old man was assessed for management of pain and painful involuntary movements of his left stump, which developed after a below-knee amputation in 1995. The patient had previously undergone nine surgical procedures to correct bone exostosis of the left foot, in the hope of achieving pain relief. One and a half years after the below-knee amputation, he developed severe stump pain. A few years later, intermittent episodes of violent jerking movements of the left stump began, which progressed to 18-20 spells a day. These could occur at rest or with voluntary movement, and could be triggered and improved by cutaneous stimulation. He had constant pain, which became worse with these movements.

Psychiatric assessment showed multiple periods of absenteeism from work for several painful problems, with very high levels of anxiety and severe depression associated with suicidal ideation at times. His family had a lifelong history of pain. According to the patient, his mother had undergone more than 50 surgical procedures, although he was unclear about the details, only saying that he hardly saw her during his childhood because she was always in hospital. The patient was unmarried and lived with his parents. He worked as a driver but had been on long-term disability since 2004.

On physical examination, the patient appeared uncomfortable and experienced excessive sweating during the episodes. The range of movement in the hip and the knee was well preserved and his strength was good. Patches of hypoaesthesia and hyperaesthesia were seen at the tip of the stump. Rapid, rhythmical jerking movements of the stump occurred in paroxysms. Palpation of the stump during these movements showed

strong contractions of the quadriceps and hamstrings. Jerking movements appeared when the patient became excited, but would stop when he was distracted.

Electrophysiological testing showed that the movement consisted of a reciprocal contraction of hamstrings and quadriceps at 8 Hz. When the patient tapped with his left hand, there was marked modulation of the frequency and amplitude of the activity of the quadriceps. Electromyographic activity increased when the stump was restrained.

The aetiology of the abnormal movements was discussed with the patient. He was offered a programme for management of chronic pain in conjunction with psychiatric follow-up. He refused and did not return for further care.

Discussion

It is well accepted that movement disorders occur in the affected limb after amputation. These movements typically consist of jerking of the stump associated with severe stump pain¹ and begin at variable periods after amputation. Sometimes they occur in the immediate postoperative period and settle over weeks to months, but they may also develop gradually after a variable latent period and persist indefinitely. Jerking is usually precipitated by voluntary movement, but can also be triggered by cutaneous stimuli.

Several involuntary movement disorders occur as a consequence of peripheral nerve dysfunction or injury, including hemifacial spasm, and painful legs and moving toes. Considerable controversy surrounds the pathogenesis or origin of certain other movement disorders, including fixed dystonia, which can be observed after minor peripheral injury, often, but not always, accompanied by features of reflex sympathetic dystrophy.^{3,4} The jumpy stump is often used to support the notion that these other entities are organic.² A debate exists, however, whether some of these entities are non-organic.⁵ Proponents of a psychogenic origin point to several different factors that illustrate the tenuous association between peripheral injury and the subsequent development of movement disorders, including the fact that although many people experience a peripheral trauma (often quite minor), focal movement disorders after these injuries are exceedingly rare. In a large study of 103 patients with fixed dystonia, 63% experienced peripheral trauma before the onset of dystonia. In a subgroup of 41 patients from that study, 44% noted peripheral trauma before the onset of dystonia, 89% of which were thought to have a probable or definite psychogenic aetiology.³ Verdugo and Ochoa⁴ examined 58 patients with complex regional pain syndrome type I (reflex sympathetic dystrophy) and positive motor phenomena (such as muscle spasm, dystonia, tremor or irregular jerks). All these patients showed at least one pseudoneurological sign, including distractibility and variability of the movements, as well as giveway motor weakness.⁴

Our patient showed several features consistent with a psychogenic origin, including variability, distractibility and other comorbid psychiatric features. These features have not been carefully evaluated in most cases of this disorder reported in the literature. Hence, it is unclear how often the jumpy stump may represent a psychogenic movement disorder. Our case illustrates the need for a careful

assessment of patients with a jumpy stump before an organic aetiology is accepted.

Finally, we would like to use this case to propose a revised classification of psychogenic movement disorders. According to the widely used criteria of Fahn and Williams,⁶ a diagnosis of definite psychogenic disorder could not be made in this case because the patient was not surreptitiously observed to be free of movements, and did not have a prolonged remission with psychotherapy or physiotherapy. Instead, on the basis of clinical features, his case would have been classified as one of the "probable psychogenic movement disorders". This scheme fails to acknowledge the ability of electrophysiological testing to define features of tremor and myoclonus that are incompatible with their organic counterparts.⁷

We therefore propose the addition of a *laboratory-supported definite* category to the diagnostic classification of psychogenic movement disorders comparable to the scheme proposed by Brown and Thompson.⁸ A careful evaluation of electrophysiological criteria is necessary before accepting them as diagnostic of either a psychogenic or an organic movement disorder. Indeed, there is a need for future research to establish the sensitivity and specificity of currently used evaluations and to develop new methods (eg, functional imaging) for evaluating the diagnosis and pathogenesis of psychogenic movement disorders.

C Zadikoff

Movement Disorders Centre, Toronto Western Hospital, Toronto, Canada

A Mailis-Gagnon

Comprehensive Pain Program, Toronto Western Hospital

A E Lang

Movement Disorders Centre, Toronto Western Hospital

Correspondence to: Dr A E Lang, Morton and Gloria Shulman Movement Disorders Center, Toronto Western Hospital, MCL-7, 399 Bathurst St, Toronto, ON, Canada M5T 2S8; lang@uhnres.utoronto.ca

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