We declare that we have no conflicts of interest.

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Neuroprosthetic control and tetraplegia

The neuroprosthetic achievements reported by Jennifer Collinger and colleagues (Feb 16, p 557)[1] are remarkable. The diagnosis of the tetraplegic patient of the study is, however, puzzling. The patient has spinocerebellar ataxia without cerebellar features. Material available elsewhere[2,3] suggests that her symptoms began rather suddenly 13 years before taking part in the study. She describes relapsing weakness, has normal looking hands, and, head rest excepted, no symptoms above the neck. This is unusual for spinocerebellar ataxia, which typically has slow onset with gradual deterioration.

One of several alternative explanations for this clinical picture, including cervical spinal cord pathology, is that the patient has a functional (psychogenic) tetraplegia, a common and genuine cause of physical disability.[4]

The diagnosis could affect the generalisability of these techniques. It might be harder for a patient with a brain disease to control a neuroprosthetic device than someone with a structurally normal brain. Furthermore, someone with a functional tetraplegia, and an abnormal body image, might have superior ability to control a neuroprosthetic device compared with an amputee or spinal cord injured patient in whom neuroprosthetic ability must be superimposed over a potentially intact cognitive body image.

We do not detract from the authors considerable technical achievements, nor are we suggesting that this patient's disability is anything other than genuine. However, clinical characterisation is essential in understanding the potential of this technology for patients with brain disease compared with patients with other causes of severe disability.

We declare that we have no conflicts of interest.

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Authors' reply

Jon Stone and William Landau's main concern is the possibility that the patient's disorder might be functional (psychogenic) tetraplegia.

We welcome the opportunity to present details of the patient's diagnosis, which were not included in our report[1] due to space limitations. The patient was thoroughly evaluated before enrolment, including a case review with her treating neurologist (GM) of more than 14 years. The patient first noted stiffness in her legs at age 36 years, 17 years before enrolment. Over several months, stiffness progressed to fatigue, weakness in the legs, and then to weakness in the arms over the following year. 3 years after symptom onset, the patient was too weak to walk and used a wheelchair full time; she had also developed subtle sensory symptoms and urinary retention.

Upon presentation at the University of Pittsburgh, 4 years after symptom onset, pertinent examination findings were left lateral gaze-evoked jerk nystagmus, mild left arm weakness, and severe right arm weakness. In the leg, there was complete paralysis except for knee flexion, which was near normal, and hip flexion, which showed severe weakness. She had increased tone in the legs with bilateral plantar reflexes present. She had mild to moderate vibratory sense loss to the knees, without pinprick loss. There were no cerebellar signs in the arm that moved and no obvious truncal ataxia for a patient who could not stand. She had no history of depression or significant pain. She progressed with primarily motor dysfunction and became tetraplegic. Testing, at that time, failed to reveal a genetic disorder. After thorough evaluation for central and peripheral nervous system diseases, resulting in an unknown aetiology, it was determined she had a degenerative disorder diagnosed as spinal cerebellar syndrome. Her father had a very similar set of symptoms, with the addition of ataxia, and more recently, two of her siblings were diagnosed with multiple sclerosis.

At the time of enrolment in the study,[1] she had 0/5 motor strength in all extremities, preserved—although subjectively slightly diminished—sensation, and decreased tone.