Complex regional pain syndrome (CRPS) was first described by Weir Mitchell as a vasomotor neurosis and he has been credited for first using the term causalgia in his writing to describe the symptoms associated with this rather elusive disorder.\(^1\)–\(^5\) CRPS still remains a poorly understood and disabling condition.\(^6\)–\(^8\) It is usually preceded by minor to severe trauma or surgical procedure.\(^9\)–\(^11\) Involvement of the motor system is a well-known phenomenon in CRPS, though the pathophysiology mechanisms involved are poorly understood.\(^12\)–\(^17\) Movement disorders in CRPS have been variously described as loss of voluntary control or failure to initiate movement, weakness, bradykinesia, dystonia, myoclonus, spasm, and tremor.\(^18\)–\(^20\) The current literature, which includes studies based on noninvasive brain stimulation techniques like transcranial magnetic stimulation (TMS), support increased cortical excitability in CRPS, and normalizing these cortical aberrancies can potentially help improve the motor performance in CRPS.\(^21\)–\(^23\)

In the present issue of the *Canadian Journal of Pain*, Harvey et al. describe the case of a 58-year-old woman who developed CRPS after sustaining a radial fracture.\(^24\) They treated her with graded motor imagery and noted some improvements in her pain without any significant objective improvement in the motor disability. They additionally performed TMS during the follow-up visits and noted a serial increase in delta scores based on motor evoked potential (MEP) amplitudes.
and interpreted that as being evidence for motor system reorganization. The current case, which occurred after a radial fracture was classified as CRPS type I; however, the authors do not report any findings of electromyography (EMG)/nerve conduction studies, which would be essential for this diagnosis, especially in the setting of a fracture. Absence of baseline and follow-up nerve conduction studies to rule out the possibility of any axonotmesis or neurapraxia makes the interpretation of the findings of the current case report difficult. The authors correctly noted many limitations in their performance and interpretation of the TMS experiments. To note a few, they used delta scores derived using resting motor threshold (RMT) as a surrogate for corticospinal strength, which is incorrect. They did not perform an input-output (IO) curve, which involves giving TMS pulses of varying strengths in a random order to measure the MEPs at the different stimulation intensities. Additionally, using the same swim cap to locate the hotspot for follow-up visits is an erroneous technique. Though using swim caps may be reasonable technique for a single session, it is certainly not appropriate for multiple follow-up visits, and imaging-based guidance should have been used to ensure stimulation of the same cortical site.

The authors suggested improvements noted in delta scores that are based on RMT obtained at follow-up visits as suggestive of strengthening of corticospinal projections. RMT is increased by drugs blocking the voltage-gated sodium channels and decreased by drugs that enhance non-N-methyl-D-aspartate (NMDA)-mediated glutamatergic transmission. There is no clear evidence that any of the interventions performed by the authors influence either of these mechanisms. Mechanisms of graded motor imagery, though poorly understood, implicate influencing the prefrontal and sensorimotor integration cortices. Studying the influence of a complicated therapeutic intervention using measures based on RMT does not have any clear rationale. The authors claim improvements in the MEP recorded as evidence of strengthening of corticospinal projections; however, the clinical improvements in strength are clearly lacking.

The findings of the current case report can be best interpreted as improved recording of the surface EMG as the vasomotor and sudomotor changes associated with CRPS improved. Though the authors noted non-significant changes in the edema, the peripheral changes were not clearly monitored objectively at follow-up, to be certain. Though the changes related to edema can affect the MEPs at both 110% and 130%, the variability in the MEPs is higher at intensities between 120% and 140% RMT and, as a result, the delta scores may be further falsely inflated. Another possible explanation could be the resolution of the nerve damage, which may have occurred due to the fracture and would follow a similar timeline for improvement. Improvement in the strength of corticospinal projections is an unlikely explanation for the findings noted in the current report.

Evidence does exist for motor system reorganization in CRPS and I reference some of the current literature on the subject in this write-up. The efforts by the authors to find objective measures to corroborate improvements in motor function are commendable. However, interpretation of their findings as evidence for motor system reorganization is misguided in my opinion.

Disclosure of Interest

Shabbir Hussain Merchant has no relevant conflict of interest to declare related to this publication.

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