

# Peripherally Induced Movement Disorders

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## KEYWORDS

- Peripherally-induced • Dystonia • Tremor • Myoclonus
- Hemifacial spasm • Complex regional pain syndrome
- Botulinum toxin

Peripherally induced movement disorders may be defined as involuntary or abnormal movements triggered by trauma to the cranial or peripheral nerves or roots.<sup>1–4</sup> Although patients often recall some history of trauma before the onset of a movement disorder, only anatomically and temporally related injury should be considered relevant. Despite strict criteria, the cause-and-effect relationship may not be always obvious, and the movement disorder may have no direct relationship to the earlier trauma. In some cases where the relationship is not clear, particularly if there is some secondary gain or pending litigation, the cause-effect is decided by the legal system rather than by scientific evidence.<sup>5</sup> Furthermore, the issue is often complicated by the coexistence of various psychosocial problems, and some patients may have a predominant or superimposed psychogenic movement disorder.<sup>6</sup>

It is beyond the scope of this brief review to speculate at length about possible pathophysiologic mechanisms of peripherally induced movement disorders, but individual predisposition and central reorganization in response to the peripheral injury have been considered to play an important role in the pathogenesis of peripherally induced movement disorders.<sup>1,7–9</sup> Central plasticity in response to altered peripheral input may result in maladaptive changes, such as neuropathic pain, hyperreflexia, increased muscle tone, and dystonia. The central changes may include functional alterations of excitatory and inhibitory connections, sprouting of new connections, reorganization of somatosensory and motor maps, altered levels of brain chemicals and neurotransmitters, degeneration and atrophy, and many other changes. A variety of neurophysiologic techniques, such as paired-pulse transcranial magnetic stimulation, have been used to study whether preexisting abnormality of motor cortex excitability may predispose some patients to develop peripherally induced movement disorders.<sup>9</sup> Functional MRI studies of patients with complex regional pain syndrome (CRPS) have found abnormal activations in regions such as the basal ganglia and parietal lobe which

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may explain some of the CNS-related symptoms in patients with CRPS, including movement disorders and behavioral abnormalities.<sup>10</sup>

The relationship between peripheral trauma and subsequent movement disorder has been recognized for at least 2 centuries; Gowers drew attention to this phenomenon in his 1888 book.<sup>11</sup> During the past quarter century, many series of patients with well-documented, peripherally induced movement disorders have been reported in peer-reviewed publications.<sup>12–19</sup> Peripheral trauma has been reported to cause hemifacial spasm, dystonia, tremor, tics, segmental myoclonus, and even parkinsonism (Box 1).

Different types of peripheral injury, including direct trauma, crush, laceration, surgery, or burn, may lead to movement. Because of a growing number of reports of immobilization-induced movement disorders, there is concern that immobilization<sup>20–22</sup> and constraint-induced therapies proposed for patients with dystonia, although the latter may be effective for poststroke hemiplegic patients,<sup>23,24</sup> may actually cause or exacerbate dystonia.

In previous reports, the author’s team proposed the following criteria for the diagnosis of peripherally induced movement disorders: (1) the trauma is severe enough to cause local symptoms for at least 2 weeks or requires medical evaluation within 2 weeks after trauma; (2) the initial manifestation of the movement disorder is anatomically related to the site of injury; and (3) the onset of the movement disorder is within days or months (up to 1 year) after the injury.<sup>12,14,15,25</sup> It is possible that in some patients, the peripheral injury is merely coincidental and not causative, and alternatively, some patients whose movement disorders are likely causally related to peripheral injury do not fulfill these criteria. Despite these and other limitations, the diagnostic criteria provide a useful framework for research into the mechanisms, clinical features, epidemiology, and treatment of this group of neurologic disorders.

The diagnostic criteria do not include many well-recognized features of peripherally induced movement disorders. For example, in many but not all cases, the movement disorder starts locally in the injured region and may later spread to involve adjacent and ipsilateral body parts, eventually crossing over to the contralateral side. Peripherally induced movement disorders are also often associated with pain and other sensory phenomena, including CRPS.<sup>26,27</sup> CRPS, a chronic illness associated with

| Box 1                                                                   |
|-------------------------------------------------------------------------|
| Peripherally induced movement disorders                                 |
| Hemifacial spasm                                                        |
| Dystonia                                                                |
| Tremor                                                                  |
| Parkinsonism                                                            |
| Segmental myoclonus                                                     |
| Jumping postamputation stump and other postamputation dyskinesias       |
| Tics                                                                    |
| Hemimasticatory spasm                                                   |
| Painful legs (arms) moving toes (fingers)                               |
| Synkinesis secondary to aberrant regeneration, fasciculations, myokymia |
| Contractures—congenital torticollis, Dupuytren contractures             |

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