Posttraumatic movement disorders
Sith Sathornsumetee MD (Dr. Sathornsumetee of Mahidol University, Thailand, has no relevant financial relationships to disclose.)
Mark A Stacy MD (Dr. Stacy of Duke University Medical Center received research support from Parkinson Study Group and consultation fees from Allergan, Eli Lilly, Merz, and Osmotica.)
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Introduction

This article includes discussion of posttraumatic movement disorders and peripherally-induced movement disorders. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

The concept of movement disorders following trauma to the central and peripheral nervous systems has been widely accepted. It has both medical and legal implications to clinicians and their patients. The authors of this clinical article review and provide updates on the pathogenesis, diagnosis, and management of posttraumatic movement disorders.

Key points

• Movement disorders can occur following central and peripheral nervous system trauma.
• Tremors and dystonia are the 2 most common abnormal movements following nervous system injury.
• Pathophysiology of posttraumatic movement disorders is complex and may include but not be limited to functional reorganization and alteration of neurotransmitters.
• Treatments for posttraumatic movement disorders are similar to those of nontraumatic movement disorders; however, the response is variable.
• A multidisciplinary approach is recommended in patients with comorbid psychological conditions and pending litigation.

Historical note and terminology

Although direct causal link between neurologic injury and the development of movement disorders has not been elucidated, tremor, dystonia, and parkinsonism have been reported following trauma to both the central and peripheral nervous system (Gowers 1888). The cause and effect is less apparent in cases of movement disorders following trauma to the peripheral nervous system. Some authors argue against the existence of “peripherally-induced” movement disorders (Weiner 2001; Hawley and Weiner 2011), whereas others have argued that tremor, dystonia, and segmental myoclonus following peripheral trauma are widely accepted (Jankovic 2001; Jankovic 2009; Tyvaert et al 2009). Some authors suggest the term “posttraumatic syndrome” instead of “posttraumatic dystonia” in patients who have developed abnormal posturing of body parts following peripheral injury (Kumar and Jog 2011).

Other movement disorders have been reported following trauma including chorea, hemiballism (Kim et al 2008), ballism, paroxysmal dyskinesia, tics (Fahn 1982), progressive supranuclear palsy (Koller et al 1989), painful legs and moving toes (Schott 1981), cortical reflex myoclonus (Hallett et al 1979), palatal myoclonus (Jacobson and Gorman 1949), hemifacial spasm, hemimasticatory spasm, Meige syndrome (Jacome 2010), segmental myoclonus (Jankovic and Pardo 1986; Camerota et al 2006), jumping postamputation stump, and other postamputation dyskinesia (Jankovic 2009). In addition, trauma to both central and peripheral nervous systems may also trigger or accelerate the progression of preexisting movement disorders.

Clinical manifestations

Presentation and course
Posttraumatic movement disorders have important medical and sometimes legal consequences. To establish the cause-and-effect relationship between trauma and movement disorder, the severity of the injury, time course, and anatomical relationship must be taken into consideration. All types of movement disorders have been reported to follow injury to the central nervous system but kinetic tremors and dystonia remain most common after severe head injury. Although diagnostic criteria for peripherally-induced movement disorders remain controversial, some guidelines have been proposed. These include: (1) trauma severe enough to cause local symptoms for at least 2 weeks or require medical evaluation within 2 weeks after trauma, (2) the initial manifestation of the movement disorder being anatomically related to the side of injury, and (3) the onset of the movement disorder being within days or months (up to 1 year) after the injury (Cardoso and Jankovic 1995). A systematic review demonstrated fixed dystonia, often associated with pain and sensory symptoms, as the most common movement abnormality in 713 peripherally-induced movement disorder patients (van Rooijen et al 2011). In addition, 26% of peripherally-induced movement disorder patients had identifiable nerve injury, and more than one third of patients with peripherally-induced movement disorders developed complex regional pain syndrome. A study of 58 patients with complex regional pain syndrome type 1 (or reflex sympathetic dystrophy) showed that 60% had muscle spasms leading to dystonic posture, 15% had coarse postural or action tremor, 9% had irregular jerks, 9% had dystonic spasms and irregular jerks, 5% had dystonic spasms and postural tremor, and 2% had episodic generalized choreiform movement (Verdugo and Ochoa 2000). Abnormal movements such as dystonia, tremor, and myoclonus can be a presenting symptom of complex regional pain syndrome in children who had a preceding minor trauma to affected limb (Agrawal et al 2009).

**Parkinsonism.**

*Central.* The cause-and-effect relationship between trauma and parkinsonism is supported by some but not by other studies. Parkinsonism is a rare complication of a single head injury except for penetrating injury to the brainstem or the injury is severe enough to result in a coma or vegetative state. Grimberg found only 2 out of 86 cases of parkinsonism reported to follow trauma to be true posttraumatic parkinsonism. These patients suffered from severe head injury and parkinsonism was a part of other extensive neurologic deficits (Grimberg 1934). He proposed that the diagnosis of posttraumatic parkinsonism could be made only if (1) a trauma was of sufficient severity to produce definite damage to the brain, (2) trauma was directly to the head, and (3) there was a clear and definite developmental connection between trauma and disease. In some instances, hemiparkinsonism can develop as a consequence of direct penetrating injury to the contralateral substantia nigra. Although parkinsonism is accepted almost as dogma as complication of repeated head trauma in boxers, case descriptions are not widely available, and often brought forward by authors with no personal contact with these subjects. Given that only a minority of boxers develops cognitive or motor disability, and that these data have rarely, if ever, been controlled by other high-risk hobbies or occupations, contact sports, or other activities, physicians should use more caution when making this leap of causality. A questionnaire study in 704 retired Thai traditional (kick) boxers demonstrated that a high number of professional bouts (more than 100) may serve as a risk factor for Parkinson disease, which was diagnosed in 5 boxers (Lolekha et al 2010). Other environmental factors like paraquat exposure and genetic predisposition, such as ApoE gene mutation and alpha-synuclein Rep1 expansion, may play an important role in the development of Parkinson disease following head injury (Jordan et al 1997; Goldman et al 2012; Lee et al 2012; Kieburtz and Wunderle 2013).

*Peripheral.* The role of peripheral trauma leading to parkinsonism with severe bradykinesia, rigidity, and postural instability is controversial, and every effort must be made to determine whether the patient may have exhibited symptoms prior to the reported injury. Typically, reports of parkinsonism attributed to peripheral nerve injury describe the development of rest or postural tremor within days or weeks after an injury to the affected limb, followed by progressive rigidity and bradykinesia that gradually spreads beyond the site of the original injury. In a report of 28 patients with peripherally induced movement disorders, severe local injury preceded the onset of movement disorders by 60 days. The mean age at onset of movement disorders was 46.5 years. Tremor was present in all 28 patients, 11 of whom exhibited additional parkinsonian features. In 20 patients, the movement disorders spread beyond the original site. Possible predisposing factors were identified in 13 patients; 9 had essential tremor or a family history of essential tremor. In addition, complex regional pain syndrome was present in 6 patients (Cardoso and Jankovic 1995).

**Tremor.**

*Central.* Tremors associated with trauma that are not related to parkinsonism or cerebellar dysfunction have also been described and are the most common movement disorders following head injury (Krauss et al 1996). Posttraumatic tremor usually affects the upper extremities and often exhibits a combination of rest, postural, and kinetic components
most likely due to the lesions involving thalamus, midbrain, red nucleus, cerebral peduncle, or cerebellum. In patients with tremor after severe head injury, tremor usually begins after 1 month to 18 months and usually as an associated hemiparesis is resolving (Andrew et al 1982). Posttraumatic tremor is often associated with other movement disorders such as dystonia, athetosis, and myoclonus, and they are sometimes disabling, particularly with the high amplitude (>10 cm) postural and kinetic (2.5- to 4-Hertz) tremors.

Peripheral. Jankovic and Van der Linden first reported 5 patients with onset of tremor within 9 months after peripheral injury (Jankovic and Van der Linden 1988). Approximately 65% of patients who developed tremor and dystonia after peripheral trauma had predisposing factors such as family history of essential tremor or dystonia, perinatal injury, or a prior exposure to dopamine receptor blocking agents. A majority of patients with tremors following peripheral trauma has tremor spreading beyond the side of original injury. Tremor may occur as an isolated symptom (Costa et al 2006), but in some cases it is associated with dystonia and parkinsonism. Complex regional pain syndrome type I or reflex sympathetic dystrophy may be seen in affected limb of some patients.

Dystonia.

Central. Dystonia is characterized by involuntary, sustained, patterned muscle contractions of opposing muscles that result in repetitive twisting movements or abnormal postures. Lesions in the striatum, particularly in the putamen, thalamus, and pontomesencephalic region can cause dystonia (Marsden et al 1985; Loher and Krauss 2009). Perinatal injury, closed head injury, encephalitis, and thalamotomy have been reported to cause hemidystonia (Pettigrew and Jankovic 1985). The onset of dystonia can be as early as 20 minutes after the injury (Perlmutter and Raichle 1984), but it may be delayed by several months or years after trauma. In addition to limb dystonia axial (neck and trunk) dystonia can also develop 6 months after closed head injury associated with caudate lesion. A majority of adult patients with dystonia following trauma has a history of head injury during the first 2 decades of life (Lee et al 1994). Krauss and colleagues reported 9 patients with dystonia following severe head injury; hemidystonia was the most common, and 7 patients with hemidystonia had contralateral caudate-putamen lesions (Krauss et al 1992). No correlation between initial severity of focal neurologic deficits or severity of trauma and the length of delay until the onset of dystonia.

Peripheral. It is often difficult to distinguish posttraumatic, peripherally-induced dystonias from primary (idiopathic) dystonia, and some patients with idiopathic torsion dystonia will provide a history suggesting that dystonic movements had been precipitated or exacerbated by trauma or some repetitive intense movement, such as exercise or practicing a musical instrument (Katz et al 2013). In these subjects dystonia appeared first in the injured part of the body within days or up to 12 months after the trauma, although the delayed onset following injury of up to 3 years has been reported (Vasileiadis et al 2012). Posttraumatic, peripherally-induced dystonias are often characterized by a fixed posture present at rest often with underlying contracture, limitation of range of motion, absence of sensory tricks (geste antagonistique), and presence of complex regional pain syndrome (about 20% of patients). In fact, Frei and colleagues proposed that complex regional pain syndrome could represent a variant of posttraumatic cervical dystonia that may develop over time after the initiation of dystonia (Frei et al 2004). About 10% to 20% of patients presenting with cervical dystonia have a history of significant neck trauma such as whiplash injury (Fletcher et al 1991; Tarsy 1998). Posttraumatic cervical dystonia can be classified into 2 groups according to the latency between injury and onset of dystonias. Acute-onset cervical dystonia usually begins immediately or within days after trauma and is often associated with significantly increased frequency of laterocollis with prominent shoulder elevation and trapezius hypertrophy, pain, marked limitation of range of motion, abnormal postures without phasic movements, lack of effect of sensory tricks, no increase with activation, and variable response to botulinum toxin (O’Riordan and Hutchinson 2004). Many patients with acute-onset cervical dystonia are involved in litigation.

The other type of posttraumatic cervical dystonia is delayed-onset cervical dystonia, usually occurring between 3 and 12 months after neck trauma; the clinical picture is similar to idiopathic cervical dystonia. In task-specific dystonias such as writer’s cramp, only about 10% of patients report history of hand or forearm trauma (Marsden and Sheehy 1990). Other focal dystonias such as blepharospasm and oromandibular dystonia have been reported following ocular trauma and trauma to the face, mouth, or jaw (including trauma from oral surgery), respectively (Grandas et al 1988; Schrag et al 1999).

An animal model of peripherally-induced dystonia was described (Ip et al 2016). In Tor1a+/- mice that express 50% torsinA, a nerve crush injury was followed by abnormal posturing in the lesioned hindlimb of both mutant and wild type mice, but the phenotypic abnormalities were increased by about 40% (p < 0.05) in the mutant mice. This was exacerbated by treatment with L-Dopa. The investigators concluded that peripheral nerve injury reduced torsinA.
concentration, and coupled with environmental stressors, it resulted in central motor network dysfunction leading to dystonia.

**Prognosis and complications**

Long term prognosis for posttraumatic movement disorders has not been well studied. Complete, spontaneous recovery is, however, rare except in patients with tremors following mild head injury. In the rare cases of dystonia related to subdural hematoma, the prognosis is favorable after hematoma removal. Fixed contractures often develop in posttraumatic dystonias following severe head injury if treatment and physical therapy are delayed.

**Biological basis**

**Etiology and pathogenesis**

Perinatal injury and closed head injuries have been associated with a number of movement disorders. In peripherally-induced movement disorders, several mechanisms of injury have been reported including striking the neck against the floor, whiplash injury, traction injury of the neck or shoulder, twisting the back while lifting, pulling the starter cord of a mowing machine, heavy objects dropping on a limb, ankle sprain fall during active military duty (Carroll et al 2006), surgery such as laminectomy or corpectomy (Takemoto et al 2006), fracture of long bone or rib (Papadimitriou et al 2005), dental procedures (in oromandibular dystonia), electrocution, and burn injury (Jankovic and Van der Linden 1988; Sankhla 1998). Prolonged immobilization following the peripheral insult seems to be an important risk factor, and many movement disorders become manifested after removal of a cast or splint. In 1 study, cortical thickness and white matter fractional anisotropy was examined in 10 patients with injury of the right upper extremity that required at least 14 days of limb immobilization (Langer et al 2012). The patients underwent 2 MRI examinations, the first within 48 hours postinjury and the second after an average time interval of 16 days of immobilization. Immobilization was associated with a decrease in cortical thickness in the left primary motor and somatosensory area as well as a decrease in fractional anisotropy in the left corticospinal tract, providing evidence that immobilization induces rapid reorganization of the sensorimotor system. Using diffusion tensor imaging, another study showed cortical reorganization in response to cervical injury (Freund et al 2012). Unknown genetic predisposition and environmental factors may play an important role in the pathogenesis of movement disorders following trauma, given the rarity of the posttraumatic movement disorders that occur after more common nervous system injury.

**Movement disorders after central trauma.** The pathophysiology of movement disorders relates to basal ganglia dysfunction, and traumatic lesions of cortical regions may alter basal ganglia function indirectly and influence movement disorders (Goetz and Pappert 1992). There are changes in the central nervous system following trauma including inflammatory response, oxidative stress injury, axonal sprouting, demyelination, remyelination, central synaptic reorganization, and other pathophysiologic mechanisms (Freund et al 2012). Several forms of brain plasticity are well documented such as changes in neurotransmitter sensitivity, collateral sprouting, and diaschisis. Development of neuronal injuries and plasticity is thought to be underlying mechanism of movement disorders following trauma to the central nervous systems. Several movement disorders such as dystonia develop several months or years after head trauma; the mechanism of variable latency is unclear (Jankovic 1994). Most studies suggest that intact corticospinal tract is essential for secondary dystonia to develop. Dystonia may be masked by the persistent severe hemiparesis following head injury (Marsden et al 1985). When both pyramidal and extrapyramidal systems are affected by trauma, the subsequent movement disorder may consist of the combination of dystonia and spasticity. In a rat model of traumatic brain injury, alpha-synuclein expression was increased in substantia nigra pars compacta, which may pathologically support the link between head injury and Parkinson disease (Acosta et al 2015).

**Movement disorders after peripheral trauma.** A cause-and-effect relationship between peripheral trauma and subsequent movement disorder is widely, but not universally, accepted (Jankovic 2001; Weiner 2001; van Hilten 2007; Ure et al 2016). Several factors including genetic susceptibility, preexisting brain dysfunction, and prior exposure to certain drugs or toxins may play important roles. Pathophysiologic changes in patients with movement disorders following trauma to the peripheral nervous system may resemble ones in complex regional pain syndrome type I or previously termed “reflex sympathetic dystrophy.” A study demonstrated a decrease in axonal density in complex regional pain syndrome affected limb compared to unaffected ipsilateral and normal contralateral limb controls (Oaklander et al 2006). In addition, symptom-matched control subjects did not have decreased axonal density in the painful site. These results suggested that complex regional pain syndrome type I is specifically associated with posttraumatic distal nerve injury. It remains to be investigated whether distal axonal injury has direct implication in...
pathogenesis of posttraumatic movement disorders. Centrally, functional changes in afferent neuronal input to the spinal cord and central cortical-subcortical reorganization are probably involved (Baron et al 2013). That peripheral injury can affect reorganization in the basal ganglia is suggested by the observation that thermal injury to a hind limb of rat causes bilateral reduction of neuronal activity in basal ganglia (de Ceballos et al 1986). Peripheral nerve deafferentation can also cause expansion of receptive fields of sensory neurons in thalamus. Central reorganization probably explains why peripherally induced movement disorders have a tendency to spread beyond the original site of injury. A functional MRI study in patients with complex regional pain syndrome demonstrated activation of the contralateral primary somatosensory, motor, and parietal association cortices, bilateral secondary somatosensory, insular, and cingulate cortices, and deactivation in the visual, vestibular, and temporal cortices (Maihofner et al 2006). Transcranial magnetic stimulation demonstrated abnormal excitability of motor cortex in peripherally induced dystonia (Bohlhalter et al 2007). Taken together, these findings suggest that peripheral trauma may be associated with functional reorganization in the brain and a shift of activation from tonically active sensory systems, such as visual and vestibular cortices, to somatosensory-related brain areas in complex regional pain syndrome patients (Baron et al 2013; Patel et al 2014).

Epidemiology"

The actual prevalence of posttraumatic movement disorders is unknown. There are few epidemiological studies on the prevalence of posttraumatic movement disorders with variability in methodology and population among studies, yielding a frequency ranging from 13% to 66% (Krauss and Jankovic 2002). Lee and colleagues found only 10 cases with dystonia after head trauma from reviewing 133 patients with symptomatic dystonia by using stringent inclusion criteria that patients must have had either hemiparesis or demonstrated MRI/CT lesions (Lee et al 1994). In a study of 221 surviving patients with head injury admitted to the trauma unit with Glasgow Coma Scale score of 8 or less, posttraumatic movement disorders were found in 22% of patients, which were transient in 10% and persistent in 12%. Tremor was the most common movement disorder found in this series (Krauss et al 1996). In a questionnaire study of 195 patients with essential tremor, history of head trauma was associated with early age of onset, in addition to family history of tremor (Louis and Ottman 2006). A follow-up study of 519 patients with mild to moderate head injury (Glasgow Coma Scale score between 9 and 15 on admission to the hospital) revealed that movement disorders were found in 10%, which were transient in 7% and persistent in 3% (Krauss et al 1997). Several studies demonstrated no association between head trauma and late-onset primary cranial dystonias such as hemifacial spasm (Martino et al 2007) or blepharospasm (Peckham et al 2011). However, in another case-control study of task-specific focal dystonias, a significant association between head trauma with loss of consciousness and development of writer’s cramp was noted (Roze et al 2009). The role of head trauma as a risk factor of Parkinson disease is controversial. The history of trauma usually dates back 20 or 30 years before the onset of Parkinson disease causing the recall bias in several retrospective case-control studies and, therefore, any cause-effect relationship is difficult to establish. Godwin-Austen and colleagues in a survey in England found a positive correlation between head trauma and Parkinson disease (Godwin-Austen et al 1982). Ward and colleagues postulate a genetic association to trauma and movement disorders and found head trauma to be an independent risk factor for developing Parkinson disease in an affected twin (Ward et al 1983). A case-control study in Olmsted county, Minnesota, revealed that the frequency of head trauma overall was significantly higher in patients with Parkinson disease than in controls (odds ratio=4.3; 95% CI=1.2 to 15.2), suggesting an association between head trauma and the later development of Parkinson disease (Bower et al 2003). Another case-control study in British Columbia, Canada also found that head injury, particularly at work, is associated with increased risk for Parkinson disease (Harris et al 2013). However, in 2 population-based, case-control studies in Denmark and Sweden, hospital contact for head injury seemed to be a consequence of Parkinson disease rather than its cause, as they usually occurred within 3 to 12 months before diagnosis of Parkinson disease (Rugbjerg et al 2008; Fang et al 2012). Another interview-based case-control study in Denmark failed to demonstrate that head injury increases the risk of Parkinson disease (Kenborg et al 2015). A systematic review and metaanalysis of 22 studies demonstrated a pooled odds ratio for the association of head injury and Parkinson disease of 1.57 (95% CI, 1.35-1.83), with a higher risk in subjects with a history of concussion (Jafari et al 2013). However, another systematic review from the International Collaboration on Mild Traumatic Brain Injury Prognosis group did not find evidence to support the causal association between mild traumatic brain injury and Parkinson disease (Marras et al 2014).

Many factors might confound the association of head trauma and Parkinson disease, such as differences in childhood and adolescent lifestyles or genetically determined risk-taking behaviors. A twin study may control these variables and provide a more precise association. A case-control study in 93 twin pairs from the National Academy of Sciences/National Research Council World War II Veteran Twins Cohort demonstrated an association between a prior
head injury with amnesia or loss of consciousness and an increased risk for Parkinson disease (odds ratio, 3.8; 95% confidence interval, 1.3-11; p = 0.014). Risk increased further with a subsequent head injury and with head injuries requiring hospitalization (Goldman et al 2006).

A population-based analysis quantified the risk for developing Parkinson disease after traumatic brain injury compared to nontraumatic brain injury trauma in California. It demonstrated that among patients aged 55 and more, traumatic brain injury was associated with a 44% increased risk of developing Parkinson disease over 5 to 7 years that was not likely from confounding factors or reverse causation (Gardner et al 2015).

A population-based study in Spain demonstrated that serious head injury was significantly more common in patients with essential tremor than in controls (Benito-Leon et al 2015). This new finding is interesting but requires additional studies to confirm the association.

**Prevention**

No known preventive strategies following trauma that may decrease the occurrence of posttraumatic movement disorders.

**Differential diagnosis**

**Nondystonic torticollis.** It is important to differentiate nondystonic torticollis that may occur following trauma from the “true” dystonic torticollis. Nondystonic torticollis may result from atlantoaxial subluxation or fixation, fourth nerve palsy, and hemianopsia causing head posturing, nasopharyngeal infection, and psychogenic dystonia. Acute posttraumatic dystonic torticollis begins 1 to 4 days after the neck injury and differs clinically from idiopathic torticollis by marked limitation of range of motion, lack of improvement after sleep ("honeymoon period"), and absence of geste antagonistique. Onset of pain immediately after the trauma and marked spasms of the paracervical muscles are other predominant features. Anticholinergic therapy is without benefit; however, some improvement occurred with botulinum toxin injection (Truong et al 1991).

**Primary (idiopathic torsion) dystonia.** In contrast to action dystonia and mobile dystonic posture, seen in patients with primary dystonia, the posttraumatic, peripherally-induced dystonias are often characterized by a fixed posture present at rest often with underlying contracture, limitation of range of motion, absence of sensory tricks, and presence of complex regional pain syndrome.

**Psychogenic movement disorders.** Many authors argue that the relationship between trauma and movement disorder is coincidental. This is often reinforced by the perception that patients with posttraumatic movement disorders have a psychological component to their disorder and the severity of disorder is influenced by pending litigation. A majority of abnormal movements of psychogenic cause is classified in the dyskinesia categories. Psychogenic movement disorder is suggested by a sudden onset, spontaneous remissions, changing frequency, amplitude, and pattern, paroxysmal occurrence, distractibility, and a character of the movement that is not consistent with a typical movement disorder (Monday and Jankovic 1993). In addition, other features such as false (“give-way”) weakness, false sensory symptoms and pseudoseizure may be present. A dramatic improvement after suggestion, physical therapy, and psychotherapy argues against an organic etiology of movement disorders. Finally, clinicians should consider the possible combination of an existing movement disorder with a psychogenic dyskinesia or the exaggeration of a posttraumatic movement disorder on a psychological basis.

**Diagnostic workup**

Diagnosis of posttraumatic movement disorders is based on clinical history and physical examination. Brain imaging such as CT or preferably MRI may show contralateral basal ganglia (particularly caudate-putamen) or thalamic lesions in patients with hemidystonia following severe head injury. Functional studies such as SPECT, PET, or functional MRI are currently used for the research purpose.

**Management**

**Parkinsonism.** The treatment of posttraumatic parkinsonism is the same as for Parkinson disease, but the response to medications is less predictable. In patients with established lesions in substantia nigra, the benefit from L-dopa therapy may be dramatic; however, in some cases it may be complicated by motor fluctuation and dyskinesia. In
peripherally induced parkinsonism, levodopa is usually not effective as a result of postsynaptic damage from central reorganization associated with aberrant peripheral input. Emerging surgical therapies, such as functional stereotactic surgery or, preferably, deep brain stimulation in the thalamus, may be effective in selected cases (Krauss and Jankovic 2002; Ruzicka et al 2005). The postsynaptic component of peripherally-induced parkinsonism is supported by poor response to bilateral stimulation of the subthalamic nucleus (Baizabal-Carvallo and Jankovic 2014).

**Tremor.** The postural and intention tremor occurring after mild to moderate head injury usually do not require therapy and subside spontaneously. In contrast, only a small number of patients with tremor following severe head injury improve spontaneously within 1 year of onset. In these cases, posttraumatic tremor is difficult to treat. Beta-blockers such as propranolol may decrease kinetic tremor and improve motor control (Ellison 1978). Carbamazepine, primidone, levodopa/carbidopa, anticholinergics, benzodiazepines, and new antiepileptic drugs such as topiramate may be used for postural and action tremors as an initial therapy. Botulinum toxin injection to affected limb is helpful in some cases that fail oral therapy, but the high doses that must be administered to both proximal and distal arm muscles limit the use of this treatment. In severe, medically refractory cases, surgical intervention such as deep brain stimulation or ablative stereotactic surgery with targets in the thalamus or the subthalamic nucleus should be carefully considered. Despite the excellent efficacy of ablative stereotactic surgery, many patients develop permanent cognitive/language side effects and secondary dystonia. Therefore, deep brain stimulation to the ventral intermediate nucleus of thalamus, ventro-oralis posterior/zona incerta region, or globus pallidus internus is generally recommended as the preferable procedure, although it has been used in only limited number of patients with posttraumatic tremor and the benefit is variable (Reese et al 2011a; Reese et al 2011b; Issar et al 2013; Sitsapesan et al 2014; Rojas-Medina et al 2016). This approach should be considered no earlier than 1 year after the onset of tremor (Krauss and Jankovic 2002).

**Dystonia.** Medical therapy for posttraumatic dystonia is often ineffective with minimal response to anticholinergic drugs. Botulinum toxin is the treatment of choice for posttraumatic torticollis and other focal dystonias. Functional stereotactic surgery targeting the thalamus, the subthalamic nucleus, or the internal globus pallidus is an alternative option for patients with disabling hemidystonia. Benefit is usually seen in the early postoperative period; however, the long-term outcome is still unknown. The data for deep brain stimulation in posttraumatic hemidystonia or peripherally-induced dystonia remain limited (Capelle et al 2006). Reports demonstrated long-term efficacy of globus pallidus stimulation in patients with posttraumatic hemidystonia and dystonic tremor (Kang et al 2010; Carvalho et al 2014). In patients with generalized dystonia and posttraumatic hemiballism, chronic intrathecal administration of baclofen via an implanted pump may provide symptomatic relief (Meythaler et al 1999; Francisco 2006). Other surgical interventions such as local peripheral and central denervation may be considered in refractory cases. In patients with significant psychiatric comorbidities and pending litigation, multidisciplinary treatment including physiotherapy and psychotherapy should be considered (Schrag et al 2004).

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**References especially recommended by the author or editor for general reading.

**ICD and OMIM codes**

**ICD codes**

ICD-9:
- Myoclonus: 333.2
- Secondary parkinsonism: 332.1
- Spasmodic torticollis: 333.83
- Essential tremor: 333.1

ICD-10:
- Myoclonus: G25.3
- Secondary parkinsonism: G21
- Spasmodic torticollis: G24.3
- Essential tremor: G25.0

**Profile**

**Age range of presentation**

0-01 month
01-23 months
02-05 years
06-12 years
13-18 years
19-44 years
45-64 years
65+ years

**Sex preponderance**

female > male in peripherally-induced movement disorders

**Family history**

Family history may be obtained

**Heredity**
Heredity may be a factor

**Population groups selectively affected**

None selectively affected

**Occupation groups selectively affected**

None selectively affected

**Differential diagnosis list**

nondystonic torticollis
primary (idiopathic torsion) dystonia
psychogenic movement disorders

**Associated disorders**

Complex regional pain syndrome
Head injury
Reflex sympathetic dystrophy

**Other topics to consider**

Blepharospasm
Childhood movement disorders
Complex regional pain syndrome
Electrical injuries: neurologic complications
Hemiballism
Psychogenic movement disorders
Severe closed head injury
Sports activities: neurologic complications
Whiplash injuries