A Whole Lot of Shakin' Going On: Movement Disorders Caused by Brain Trauma

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A WHOLE LOT OF SHAKIN’ GOING ON:
MOVEMENT DISORDERS CAUSED BY BRAIN TRAUMA

JACK E. HUBBARD* AND SAMUEL D. HODGE, JR.**

ABSTRACT

There has been a lot of publicity directed to the consequence of brain trauma, such as headaches forgetfulness, irritability, and depression. That is only part of the sequelae. A little known but challenging result of brain trauma is the development of or aggravation of a movement disorder such as a tremor, dystonia, a tic, or Parkinson’s Disease.

A movement disorder is an all-encompassing term that refers to a constellation of neurological issues that cause involuntary or voluntary movements or abnormal positioning of a body part. Various regions of the brain interact with each other to control movements of the body. If an injury occurs to a part of the brain that affects movement, it can trigger mobility problems and change the established line of communications. This can result in a host of unwarranted issues from a simple tic to a progressive neurological disorder that can lead to significant motor impairment over the years.

Very little has been written about the medical-legal aspects of movement disorders and brain trauma. This article attempts to fill that void. It will discuss the medical aspects of post-traumatic movement disorders with a focus on the physiology of the brain and how the resultant movement maladies develop. The second section will examine the legal cases where this neurological problem has become an issue.

CONTENTS

I. INTRODUCTION ................................................................. 288
II. A MEDICAL EXPLANATION OF MOVEMENT DISORDERS ... 289
   A. Anatomy ........................................................................ 290
   B. Pharmacology ............................................................... 292
   C. Post-Traumatic Movement Disorders ......................... 293
      1. Tremor ....................................................................... 294
      2. Dystonia .................................................................... 295
      3. Chorea ....................................................................... 295
      4. Tics .......................................................................... 296
      5. Myoclonus ................................................................. 297
      6. Parkinson’s Disease .................................................. 298

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I. INTRODUCTION

Muhammad Ali is considered one of the greatest boxers and celebrated heavyweights of all time. He is credited with some of the most renowned moments in the ring, and his devotion to his craft was unmatched. There was one opponent, however, that he could not defeat: a movement disorder known as Parkinson’s disease. This degenerative process was diagnosed three years after he retired from boxing. The many punches to the head that Ali absorbed in the ring are believed to have played a role in the development of his slowness of movement, tremors, and speech problems. Physicians are unable to say that boxing was the definite cause of his declining health, but all agree that repeated head trauma increased the risk.

Much attention has been devoted to concussion and related forms of brain trauma that produce symptoms, such as headaches, memory difficulty, and pain in various parts of the body. A little known but very problematic consequence of brain trauma is that of movement disorders—i.e., impairment of movement that is not paralysis, but rather involves difficulty with carrying out voluntary actions. Very little has been written about the medical-legal aspects of these disorders. This article attempts to fill that void. Section II discusses the medical aspects of post-traumatic movement disorders. Section III then discusses the legal cases where this neurological impairment has become an issue.

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5 Bob Tedeschi, Muhammad Ali and Parkinson’s Disease: Was Boxing to Blame?, STAT (June 4, 2016), https://www.statnews.com/2016/06/04/muhammad-ali-parkinsons-disease/.
6 The connection between trauma and movement disorders was recognized in the nineteenth century. SOTIRIOS A. PARASHOS, CURRENT CLINICAL PRACTICE 427 (Charles H. Adler & J. Eric Ahlskog eds., 2000).
II. A MEDICAL EXPLANATION OF MOVEMENT DISORDERS

Movements of the body are controlled by various parts of the brain that interact with each other.  

If an injury occurs to a part of the brain that affects movement, it can trigger mobility problems and change the established motor lines of communication. 

All body movements are initiated and controlled by the motor system. This is the part of the nervous system that allows for voluntary, conscious movements, such as raising an arm, moving a leg, and turning the head, as well as repetitive, learned voluntary movements that are more complex but that we do not think about, such as running, bicycling, and typing. 

The motor system is further divided into two subparts—the pyramidal and extrapyramidal systems.

To understand the interaction of these two motor subsystems, think of a car. For a car to operate properly, the vehicle must have an engine and transmission. The engine starts and stops the car’s motion as well as controls how fast the car can go. The transmission allows for smoothness in acceleration and deceleration of the car. Similarly, the pyramidal system can be likened to the engine of the car. Originating from the surface of the cerebral cortex, the pyramidal system initiates and stops movement through its connections with the spinal cord via the corticospinal tract.

Like the transmission of the car, the extrapyramidal system provides smoothness and accuracy of movement. Centers controlling the functions of the extrapyramidal system are located deep within the cerebral hemispheres. Just as both the car’s engine and transmission need to be in good working order for the car to function properly, the pyramidal and extrapyramidal systems are necessary for

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10. Id. at 3-5.

11. The pyramidal system is the primary pathway for voluntary movement. Each of the motor signals that start in cortex of the brain travel through the pyramidal system. This conduit supplies the voluntary muscles of the head, neck, and limbs. *Neural Pathways*, http://pegasus.cc.ucf.edu/~cwatts/spa3101/Neuro4.html (last visited Oct. 25, 2016).

12. The extrapyramidal system is also a motor system that helps control movement. Neuronal activity starts in the cerebral cortex and influences the lower motor neurons. The basal ganglia are the primary extrapyramidal nuclei. See id.


14. The cerebral hemispheres are the largest part of the brain, and each forms one half of the cerebrum. They are separated by a large fissure or indentation, and each half controls muscle function, speech, thought, emotions, reading, writing, and learning. *What is the Cerebral Hemisphere*, Sharecare, https://www.sharecare.com/health/nervous-system-parts/what-is-the-cerebral-hemisphere (last visited Oct. 25, 2016).
graceful, accurate body movements. Damage to the pyramidal system results in paralysis\(^{15}\) of the affected part of the body. On the other hand, an injury to the extrapyramidal system produces a movement disorder, and not paralysis.\(^{16}\)

Movement disorders caused by disturbance in the extrapyramidal system are a collection of neurological problems that result in impaired or disrupted voluntary movement, collectively termed dyskinesias (dys = abnormal; kinesia = movement).\(^{17}\) Some dyskinesias produce slowness of movement termed hypokinesias (hypo = diminished; kinesia = movement), as in the slowness seen in Parkinson’s disease.\(^{18}\) Other forms of dyskinesias produce abnormally excessive or involuntary movements termed hyperkinesias (hyper = increased; kinesia = movement), as in Huntington’s chorea.\(^{19}\)

Dyskinesias are caused by many factors, such as certain medications and toxins.\(^{20}\) Some movement disorders are due to neurodegenerative diseases including Parkinson’s disease and Huntington’s chorea.\(^{21}\) Head trauma can also produce dyskinesias, collectively termed post-traumatic movement disorders. The relationship between a head injury and a movement disorder is not fully known. Statistically, however, movement disorders following a severe head trauma is not a rare occurrence.\(^{22}\) While the alterations in movement following a mild or moderate brain trauma may be transient, severe head trauma can have life altering consequences.\(^{23}\) This medical-legal review will focus on those movement disorders that are caused by brain trauma, namely: tremor, dystonia, chorea, tics, myoclonus, and Parkinson’s disease.

**A. Anatomy**

Before addressing these specific movement disorders, it is helpful to understand where the extrapyramidal system, the part of the motor system responsible for involuntary movement,\(^{24}\) is located and how it relates to the pyramidal system. There

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\(^{16}\) *Neural Pathways*, supra note 11.

\(^{17}\) Yousef Hannawi et al., *Abnormal Movements in Critical Care Patients with Brain Injury: A Diagnostic Approach*, 20 CRITICAL CARE 1, 1 (2016).

\(^{18}\) *Id.* at 2.

\(^{19}\) *Id.*


\(^{21}\) Rena K. Mann et al., *Comparing Movement Patterns Associated with Huntington’s Chorea and Parkinson’s Dyskinesia*, 218 EXPERIMENTAL BRAIN RES. 639, 639 (2012).


\(^{23}\) *Id.* at 927.

are two major centers for the extrapyramidal system. These areas are located deep within the cerebral hemispheres, collectively called the basal ganglia, and also within the brain stem located at the base of the brain.25

The basal ganglia, a collection of neurons located deep within the two cerebral hemispheres that are responsible for motor control,26 is divided into several subgroups—caudate nucleus, putamen (these two together are called the striatum), globus pallidus, and subthalamic nucleus.27 These words are foreign to most outside of the medical profession, so brief definitions are in order. The caudate nucleus is present in each hemisphere and, in addition to motor control, plays a paramount role in learning and the storage and processing of memories.28 The putamen is part of a very sophisticated feedback loop that assists in the movement of the limbs.29 It is particularly susceptible to the ravages of Parkinson’s disease.30 The globus pallidus is one of the nuclei that forms the basal ganglia,31 and the subthalamic nucleus is part of the basal ganglia found under the thalamus (hence its name).32 It connects the basal ganglia together and plays a role in inhibiting function in part of the extremities.33 Within the brain stem34 is another important structure: the substantia nigra.35 The Latin translation for this term is “black substance” because of its dark appearance.36 It plays a role in reward and movement.37

26 Jose L. Lanciego et al., Functional Neuroanatomy of the Basal Ganglia, 2 COLD SPRING HARBOR PERSP. MED. 1, 1 (2012).
27 Id.
30 See id.
34 The brain stem is located at the base of the brain, resembles a person’s thumb, and connects the brain with the spinal cord. Robert Joynt, Brainstem, ENCYCLOPEDIA BRITANNICA (July 8, 2015), https://www.britannica.com/science/brainstem.
37 See id.
These strange sounding terms have been presented because they form the centers that interconnect in complex fashions with each other and with other parts of the brain. Their output influences the final common motor pathway to the spinal cord, known as the corticospinal tract, to provide smooth, accurate, and purposeful movements of the body.

B. Pharmacology

The pharmacology of the extrapyramidal system is just as important as the anatomy in understanding movement disorders. Neurons, the basic cells of the nervous system, communicate with each other by means of a chemical, termed a neurotransmitter or chemical messenger. Neurons are not directly connected to each other, but they are separated by a narrow cleft. The contact point between two neurons is called a synapse. Neurotransmitters are released at the synapse to relay information from one neuron to another. Different kinds of neurotransmitters are found throughout the brain, but each neuron only has one kind of neurotransmitter. In this regard, the important neurotransmitters in the extrapyramidal system are dopamine, acetylcholine, gamma-aminobutyric acid (GABA), glutamate.

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38 Harvey Lodish et al., Molecular Cell Biology 1005 (Katherine Ahr et al., eds., 6th ed. 2008).

39 Id.

40 Hubbard & Hodge, supra note 25, at 31-32.

41 Lodish et al., supra note 38, at 1005.

42 Dopamine is a neurotransmitter that assists in the management of the brain's reward and pleasure centers. It also helps control movement and emotional responses. What is Dopamine, Psychology Today, https://www.psychologytoday.com/basics/dopamine (last visited Oct. 28, 2016).

43 Acetylcholine is the most widespread of the neurotransmitter whose job is to activate muscles. Kendra Cherry, Discovery and Functions of Acetylcholine, Verywell (Aug. 24, 2016), https://www.verywell.com/what-is-acetylcholine-2794810.


45 Glutamate is one of the most important neurotransmitters for normal brain activity and more than 50% of the brain transmitters release this substance. Glutamate, Neuroscience, https://www.ncbi.nlm.nih.gov/books/NBK10807/ (last visited Oct. 28, 2016); see generally Krauss & Jankovic, Head Injury and Posttraumatic Movement Disorders, supra note 22.
norepinephrine, and serotonin. When these neurotransmitters do not fire properly, tremors or other types of movement disorders may develop.

One of the reasons why many drugs and substances produce abnormal body movements is because they block or enhance the actions of a specified neurotransmitter associated with the extrapyramidal system. Likewise, enhancing or diminishing the affected neurotransmitter system can often accomplish treatment of movement disorders. For example, Parkinson’s disease results from diminished levels of the neurotransmitter dopamine within the brain. Thus, increasing the available dopamine or enhancing the neurotransmitter’s effectiveness can treat Parkinson’s.

C. Post-Traumatic Movement Disorders

Most movement disorders associated with a traumatic brain injury usually occur after a severe injury. Various studies reveal a wide range in the incidence of post-traumatic movement disorders occurring in 13-66% of patients who suffered severe brain injury. In cases of mild to moderate head injury, the incidence of movement disorders is much lower, occurring only transiently in 7.6% of the cases and persistently in 2.6% of the patients. These movement disorders are not disabling and do not require medical therapy. The most common post-traumatic movement disorders are tremor, dystonia, chorea, tics, myoclonus, and Parkinson’s disease.

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46 Norepinephrine is a stress hormone that affects those parts of the brain that regulate attention and responding actions. It is also part of the flight or fight response. Norepinephrine, RICE U. DEPT’ OF COMPUTATIONAL & APPLIED MATHEMATICS, http://www.caam.rice.edu/~cox/wrap/norepinephrine.pdf (last visited Oct. 28, 2016).


49 Extrapyramidal System, supra note 24.


53 Krauss & Jankovic, Head Injury and Posttraumatic Movement Disorders, supra note 22; see also Joachim K. Krauss & Joseph Jankovic, Movement Disorders After Traumatic Brain Injury, in NATHAN D. ZASLER ET AL., BRAIN INJURY MEDICINE 661, 662 (2d ed., Demos Med. 2013) [hereinafter Krauss & Jankovic, Movement Disorders After Traumatic Brain Injury].

54 Joachim K. Krauss et al., Posttraumatic Movement Disorders After Moderate or Mild Head Injury, 12 MOVEMENT DISORDERS 428 (1997) [hereinafter Krauss et al., Moderate or Mild Head Injury].

55 O’Suilleabhain & Dewey, supra note 52.
1. Tremor

A tremor is an involuntary rhythmic shaking of an extremity, or portion of the extremity, such as the hand or the head.\textsuperscript{56} A tremor is a type of hyperkinetic movement disorder and can result from a variety of causes; some are fairly benign, but some are more serious. A useful way of sorting out causes of tremors is to consider when they happen. Some tremors, termed \textit{intention tremors}, only occur when the extremity is being used, such as the hand shaking while eating.\textsuperscript{57} The most common tremor of this type is called \textit{benign essential tremor}, and it is a rhythmic trembling of the hands, head, voice, legs, or trunk.\textsuperscript{58} Some tremors, termed \textit{resting tremor}, only occur when the extremity is at rest but go away when, for example, the hand is used in some type of activity.\textsuperscript{59} This tremor type is seen in \textit{Parkinson’s disease}.\textsuperscript{60} \textit{Postural tremors} occur with positional changes, such as an \textit{orthostatic tremor} that develops with standing.\textsuperscript{61}

A tremor is the most common movement disorder associated with a brain injury, usually occurring as intention tremors, but they can also be postural tremors.\textsuperscript{62} The time lapse between the trauma and the appearance of a tremor is variable but generally appears from within a month up to a year.\textsuperscript{63} Often the trauma is the result of a motor vehicle accident with deceleration trauma, but it also occurs in pedestrians who are struck by a vehicle.\textsuperscript{64} Typically in these cases, the tremor does not occur as the solitary symptom; in a high percentage of the times, it is associated with cognitive abnormalities, speech problems, and even some degree of weakness.\textsuperscript{65} Consequently, post-traumatic tremors are associated with a significant brain injury with the person being comatose for weeks.\textsuperscript{66} In some cases of a traumatic neck injury, a postural tremor may develop on the side of the injury.\textsuperscript{67}

\begin{itemize}
\item \textsuperscript{57} Id.
\item \textsuperscript{59} \textit{Tremor Fact Sheet}, supra note 56.
\item \textsuperscript{60} Id.
\item \textsuperscript{61} Joseph Jankovic, \textit{Movement Disorders}, in \textit{TEXTBOOK OF CLINICAL NEUROLOGY} 713, 732 (2d ed. 2003) [hereinafter Jankovic, \textit{Movement Disorders}].
\item \textsuperscript{62} HUBBARD & HODGE, supra note 25.
\item \textsuperscript{63} Joachim K. Krauss et al., \textit{The Treatment of Posttraumatic Tremor by Stereotactic Surgery: Symptomatic and Functional Outcome in a Series of 35 Patients}, 80 J. NEUROSURGERY 810, 815 (1994).
\item \textsuperscript{64} Id.
\item \textsuperscript{65} Id.
\item \textsuperscript{66} Id.
\item \textsuperscript{67} T. Hashimoto et al., \textit{Peripheral Mechanisms in Tremor After Traumatic Neck Injury}, 73 J. NEUROLOGY NEUROSURGERY PSYCHIATRY 585 (2002).
\end{itemize}
2. Dystonia

_Dystonia_ is a hyperkinetic movement disorder characterized by sustained, repetitive contractions of muscle groups that can occur either at rest or when carrying out a specific activity. Dystonia can involve the entire body (generalized dystonia) or can be confined to localized muscle groups (focal dystonia). Common forms of focal dystonias are cervical torticollis (“wry neck”) and writer’s cramp. In some cases, the dystonia may also have an associated tremor.

The most common form of dystonia following brain injury is one in which one entire side of the body is affected (hemidystonia), with brain injury resulting in 7-9% of all the various causes for hemidystonia. As with a tremor, most cases of post-traumatic hemidystonia only develop after a severe head injury, but some cases following mild-moderate head trauma have been reported. While the time frame from injury to appearance of symptoms is variable, one study reported that the mean latency is about twenty months. In most cases of post-traumatic dystonia, the injury occurs in the basal ganglia.

3. Chorea

Derived from the Latin term choreus (“dance”), _chorea_ is another hyperkinetic movement disorder that is characterized by involuntary, random, brief, jerking, non-rhythmic movements. The overall appearance is a person who appears restless with constant random movements of the head and extremities. An extreme form of chorea is a flinging movement of an extremity, such as that which resembles an arm throwing a ball termed _ballismus_. This form is associated with damage to the

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71 Jankovic, _Movement Disorders_, supra note 61.
72 Krauss & Jankovic, _Movement Disorders After Traumatic Brain Injury_, supra note 53.
73 Id.
76 Stanley Fahn, _Hypokinesia and Hyperkinesia_, in _TEXTBOOK of NEUROLOGY_ 279, 280 (2d ed. 2003).
78 Id.
If only one side is affected, this condition is termed hemiballismus.\textsuperscript{80} Choreic movements are sometimes associated with slow writhing movements of an extremity of the body termed athetosis; the combined appearance of these two movement disorders is termed choreoathetosis.\textsuperscript{81} Commonly seen in the neurodegenerative disorder Huntington’s disease, choreic movements can occur in other conditions as well, such as Sydenham’s chorea resulting from a bacterial infection, senile chorea beginning after age sixty, hyperthyroidism, and as an adverse reaction to various medications.\textsuperscript{82} Treatment of Parkinson’s disease with excessive amounts of dopamine can also produce choreic movements.\textsuperscript{83} The choreic involuntary movements displayed by the actor Michael J. Fox is such an example.\textsuperscript{84} In cases of severe head trauma, choreic movements can occur within weeks to months following injury.\textsuperscript{85} Traumatic bleeding into the subthalamic nucleus has been reported to produce symptoms on one side within a day of the brain injury.\textsuperscript{86} Choreic movements have also been reported to occur in conjunction with an epidural or subdural hematoma as well.\textsuperscript{87}

4. Tics

Tics are another form of a hyperkinetic movement disorder that occur within a localized muscle group as a motor tic, as part of a vocalization, or a combination of the two symptoms.\textsuperscript{88} With regard to motor tics, they appear as abrupt, sudden, single isolated movements, such as a shoulder shrug, head jerk, eye blink, or darting of the eyes.\textsuperscript{89} Tics can occur repetitively and, unlike choreic movements, can be temporarily suppressed.\textsuperscript{90} The vocalization that occurs with or without the motor tics can simply be a grunt or throat clearing but can also be more complex, sounding like

\textsuperscript{79} Fahn, supra note 76.
\textsuperscript{80} Gonzalez-Usigli, supra note 77.
\textsuperscript{81} Fahn, supra note 76.
\textsuperscript{82} Jankovic, Movement Disorders, supra note 61.
\textsuperscript{83} Id.
\textsuperscript{84} Parkinson’s Disease, HUMAN DISEASES & CONDITIONS, http://www.humanillnesses.com/original/Pan-Pre/Parkinson-s-Disease.html (last visited Jan. 29, 2017).
\textsuperscript{85} Krauss & Jankovic, Movement Disorders After Traumatic Brain Injury, supra note 53.
\textsuperscript{86} Han-Joon Kim et al., Posttraumatic Hemiballism with Focal Discrete Hemorrhage in Contralateral Subthalamic Nucleus, 14 PARKINSON RELATED DISORDERS 259 (2008).
\textsuperscript{87} John R. Adler & Ken R. Winston, Chorea as a Manifestation of Epidural Hematoma; Case Report, 60 J. NEUROSURGERY 856 (1984).
\textsuperscript{88} Fahn, supra note 76.
\textsuperscript{89} Id.
\textsuperscript{90} Nischant Ranjan et al., Tics After Traumatic Brain Injury, 25 BRAIN INJURY 629 (2011).
The most common form of a tic first occurs in childhood as Gilles de la Tourette syndrome (also known as Tourette’s syndrome).\textsuperscript{92}

Usually, tics occur weeks to months following a head injury but sometimes materialize later on.\textsuperscript{93} Also, although it rarely occurs, tics may result from a brain injury. This was the case for a nineteen-year-old who sustained a severe traumatic brain injury.\textsuperscript{94} He had no prior history of tics or vocalization but one year following the brain injury developed both motor and vocal tics.\textsuperscript{95} A magnetic resonance imaging scan (MRI) of his brain revealed damage to the basal ganglia.\textsuperscript{96}

In a series of six patients who developed post-traumatic tics, five of them had mild-moderate injury, and none had basal ganglia abnormalities on their MRI scans.\textsuperscript{97} Because Tourette’s syndrome develops in childhood, a careful history is necessary to assure that the tics are not a prior condition although pre-existing tics may worsen after a head injury.\textsuperscript{98} The inception of tics in adults is rare, and a medical search reveals only four studies that discuss tics in the context of adults with traumatic brain injuries.\textsuperscript{99}

5. Myoclonus

Myoclonus is another example of a hyperkinetic movement disorder that is characterized by sudden, brief, shock-like involuntary movements from a muscle contraction.\textsuperscript{100} They can appear as irregular, non-rhythmic movements or as rhythmic movements, such as those involving the palate or eyes.\textsuperscript{101} At times, it may be difficult to distinguish repetitive tics from myoclonus.\textsuperscript{102} Some forms of myoclonus occur at rest while others are only apparent with a purposeful body movement, such as moving an arm or leg.\textsuperscript{103}

Myoclonus can be produced by many different causes, including metabolic (e.g., kidney failure, hypoxia, liver failure, low blood sugar or sodium levels), poisonings (e.g., bismuth, cocaine, mercury), stroke, infection, medications (e.g., lithium,
amantadine, metoclopramide), and trauma. In a critically injured patient in the intensive care unit who develops myoclonic activity, it is important to sort out whether or not the brain injury is causing the myoclonic movements or if the movements could be a result of one or more of the many other causes listed above. In one series of 221 patients who had severe brain injury followed over four years, 0.5% had persistent myoclonic involuntary movements. In a subsequent study of patients who had mild-moderate head injury followed over four to six years, the same researchers identified only one patient who had mild persistent cervical myoclonic twitches.

6. Parkinson’s Disease

A resting tremor can be associated with Parkinson’s disease, but the latter condition is generally considered a hypokinetic movement disorder. Usually starting around the fifth to sixth decade of life, idiopathic Parkinson’s disease is a progressive disorder characterized by slowness of movement (bradykinesia), rigidity, flexed posture of the neck, trunk, and limbs, episodic freezing of movement, and loss of postural reflexes causing the person to fall easily. A resting tremor of the hands with a characteristic “pill-rolling” appearance is present in most but not all cases. Considered a neurodegenerative disorder leading to significant motor impairment over a matter of years, Parkinson’s disease has no available cure at this time. Treatment of the condition is symptomatic only, restoring the dopamine levels that are lost with the progressive neuronal loss in the pathway between the substantia nigra in the brain stem and the striatum of the basal ganglia.

The term parkinsonism is used when an individual shows signs of Parkinson’s but do not have the true idiopathic Parkinson’s disease. Certain medications, such as antipsychotic drugs, metoclopramide, and lithium, can produce parkinsonian features, making it important to make sure that the person is not taking these drugs when that diagnosis is being entertained. Exposure to toxins such as cyanide, manganese, and carbon monoxide can also produce features of Parkinson’s

104 See id. at 281, 290, 295.
105 See Hannawi et al., supra note 17, at 3, 6.
107 Krauss et al., Moderate or Mild Head Injury, supra note 54.
108 See Fahn, supra note 76, at 291.
109 See id. at 292.
111 See Garrett E. Alexander, Biology of Parkinson’s Disease: Pathogenesis and Pathophysiology of a Multisystem Neurodegenerative Disorder, 6 DIALOGUES IN CLINICAL NEUROSCIENCE 259, 259 (2004).
112 See Dennis W. Dickson, Parkinson’s Disease and Parkinsonism: Neuropathology, 2 COLD SPRING HARBOR PERSPECTIVES IN MED. 1, 1 (2012).
113 See Fahn, supra note 76, at 291.
The development of parkinsonism due to head trauma, however, has led to controversial issues having medical-legal implications.

In considering post-traumatic parkinsonism, one needs to distinguish the consequences after a single head injury versus subsequent to repeated head injuries. With regard to cases of parkinsonism that occur after a single head injury, Krauss and Jankovic state that “[t]he causal relationship in most cases has been largely speculative and the interpretation complicated by medicolegal issues.” Because the history of head trauma can date back twenty to thirty years prior to symptom onset, a “cause-and-effect” relationship is difficult to establish. They further note that the head injury incident may have been caused by the motor impairment of an unrecognized Parkinson’s disease, rather than vice versa.

Having made those statements, the researchers do describe some instances of parkinsonian features occurring following a football injury, as well as cases after a chronic subdural hematoma. An interesting study of ninety-three pairs of twins found that in those cases where there was a twin with a prior head injury with loss of consciousness or amnesia, they had a significantly increased risk for developing Parkinson’s disease.

Muhammad Ali’s case highlights the development of progressive bradykinesia and tremors after repeated head trauma. This clinical picture is one of a chronic encephalopathy termed “pugilistic” parkinsonism affecting between 20-50% of professional boxers. In these cases, imaging studies and neuropathological examinations demonstrate abnormal changes within the basal ganglia and substantia nigra.

III. LEGAL DISCUSSION

A. Movement Disorders

A basic question in a medical and legal context is whether trauma can cause or aggravate a medical disorder. For instance, the connection between multiple sclerosis and trauma has been debated for years. That controversy, however, has

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115 See Krauss & Jankovic, Movement Disorders After Traumatic Brain Injury, supra note 53, at 670.
116 Id.
117 Id.
118 Id.
119 Id. at 671.
120 Id. at 670.
121 See Samuel M. Goldman et al., Head Injury and Parkinson’s Disease Risk in Twins, 60 ANNALS OF NEUROLOGY 65 (2006).
122 Krauss & Jankovic, Movement Disorders After Traumatic Brain Injury, supra note 53, at 670.
123 See id.
been resolved by various scientific studies that show there is no relationship.\textsuperscript{124} Stress can cause a variety of health issues, but the association between stress and cancer is tenuous.\textsuperscript{125} That is not the case with a movement disorder and trauma. It is generally accepted that such a connection exists.\textsuperscript{126}

A movement disorder is an all-encompassing term that refers to a constellation of neurological issues that cause involuntary or voluntary movements or abnormal positioning of a body part.\textsuperscript{127} As has been noted, examples include tics, tremors, dystonia, chorea, Parkinson’s disease, paroxysmal dyskinesias, and other forms of hyperkinesias.\textsuperscript{128}

There is one medical-legal study worth mentioning. A group of researchers started with the proposition that patients with a traumatically induced movement disorder frequently seek the assistance of an attorney and engage in litigation.\textsuperscript{129} This class of patients was used to test the theory that contact with the legal system may impact the prognosis of the disorder.\textsuperscript{130} The results showed that represented patients varied from those unrepresented in that they were not as old and a substantial number more were disabled.\textsuperscript{131}

A plethora of court cases exist involving movement disorders, but most focus on a specific problem like Parkinson’s disease or dystonia.\textsuperscript{132} These lawsuits include obvious issues, such as the failure to diagnose or properly treat, Social Security disability and worker’s compensation benefits, and whether trauma can aggravate the condition.\textsuperscript{133} The more creative cases involve whether medication or vaccines can cause or exacerbate movement disorders, off-label use of medication, abnormal movements causing handwriting and forgery issues, and whether welding fumes play


\textsuperscript{128} See Antonio Siniscalchi et al., Post-Stroke Movement Disorders: Clinical Manifestations and Pharmacological Management, 10 Current Neuropsychology 254, 254 (2012); see also supra Section II.


\textsuperscript{130} Id.

\textsuperscript{131} Id.

\textsuperscript{132} See generally Vickie L. Willard, Parkinson’s Disease and Graphic Disturbances, 10 J. Forensic Document Examination 1 (1997).

\textsuperscript{133} See generally id.
a role in the development of Parkinson’s disease.\textsuperscript{134} This analysis of court cases begins with an overview of the settings in which movement disorders have been litigated. It then focuses on trauma and its relationship to a specific problem like dystonia or tics.

The issue of movement disorders has actually reached the Supreme Court on the question of whether companies that sell generic drugs can be sued for their failure to provide warnings that differ from the labels provided by the brand name manufacturers.\textsuperscript{135} One case involved the taking of a generic drug for heartburn that allegedly caused a movement disorder.\textsuperscript{136} The Court rejected the lawsuits and held that federal law preempted the claims and the defendants did not have to provide labeling that differed from the brand name label approved by the Federal Drug Administration (FDA).\textsuperscript{137}

On the other hand, the parties reached a $20 million settlement in a dispute involving the off-labeling promotion of Xyrem, a medication approved by the FDA for the treatment of daytime sleepiness.\textsuperscript{138} The company admitted that it improperly promoted the medication for the care of movement disorders, including Parkinson’s disease, even though that particular use had not been approved by the government.\textsuperscript{139} Another lawsuit involved Prozac and Lithium.\textsuperscript{140} The facts show that a woman was being treated for a bipolar disorder and had been given Lithium.\textsuperscript{141} Subsequently, the treating physician prescribed Prozac.\textsuperscript{142} The plaintiff then began having trouble walking and developed seizures.\textsuperscript{143} She was hospitalized and diagnosed with a permanent drug-induced movement disorder caused by the mixture of the two drugs.\textsuperscript{144} Both the prescribing psychiatrist and drug manufacturers were named defendants.\textsuperscript{145} The plaintiff alleged that the doctor was negligent in not properly monitoring his patient, and the drug manufactures failed to warn of the dangers of

\begin{itemize}
  \item[134] See generally id.
  \item[136] Id. The cases are PLIVA, Inc. v. Mensing, 564 U.S. 604 (2011), and Actavis, Inc. v. Demahy, 562 U.S. 1104 (2011).
  \item[137] See PLIVA, Inc., 564 U.S. at 626.
  \item[138] See Jazz Pharmaceuticals Settlement Totals $20 Million; Subsidiary Pleads Guilty, 16 No. 7 FDA EN"T MANUAL NEWSL. (Thompson Media Grp., Bethesda, Md.), Sept. 2007, at 12.
  \item[139] Id.
  \item[140] See Suit Arising from Adverse Reaction to Prozac and Lithium Results in Settlement with Drug Manufacturers and Defense Verdict for Psychiatrist, 15 No. 1 VERDICTS, SETTLEMENT & TACTICS NL 21 (1995).
  \item[141] Id.
  \item[142] Id. at 1.
  \item[143] Id.
  \item[144] Id.
  \item[145] Id.
\end{itemize}
combining the medications.\textsuperscript{146} The doctor asserted that he had no duty to monitor the patient and that her movement disorder was psychogenic and not drug-induced.\textsuperscript{147} The drug companies jointly settled their liability for $200,000, and a defense verdict was entered on behalf of the psychiatrist.\textsuperscript{148}

\textit{Rutz v. Secretary of Health and Human Services}\textsuperscript{149} involved a hepatitis B vaccine that allegedly caused the plaintiff to develop an involuntary movement disorder affecting her neck and back, along with twitching and spasms in her left shoulder and cervical spine.\textsuperscript{150} A neurologist who examined Rutz noted that her movement disorder was unusual in its clinical presentation and the patient was able to partially suppress some of the movements.\textsuperscript{151} Another neurologist opined that the plaintiff suffered from an organic tremor disorder that was psychogenic.\textsuperscript{152} Because the claimant was unable to produce a medical expert who would connect the vaccine and the problem, she argued that the vaccine caused her to suffer from a psychological injury which did not appear in the Vaccine Table of compensable claims.\textsuperscript{153} The court noted, “It is well-settled that, although the Vaccine Act relaxes the burden to prove causation in petitions which allege Table injuries, it does not relax the burden for claims based on off-Table injuries,”\textsuperscript{154} and the petitioner must prove causation-in-fact.\textsuperscript{155} The trial court denied the claim and stated that it could not find in favor of the claimant in the absence of proof that the hepatitis B vaccine was causally related or significantly aggravated her psychological condition.\textsuperscript{156} There was also no evidence that described how the vaccine caused the movement disorder.\textsuperscript{157}

\textsuperscript{146} Id.
\textsuperscript{147} Id.
\textsuperscript{148} Id. at 2. A defense verdict was rendered in a case against a doctor who prescribed Risperdal for ten years to treat severe depression and anxiety but discontinued the drug after the plaintiff developed a head tilt during her pregnancy. The medication had to be restarted to control the claimant’s mental disease and movement disorder. In her suit, the woman alleged that the doctor improperly prescribed Risperdal which caused her permanent movement disorder. The psychiatrist argued the drug was appropriate for the plaintiff’s mental illness and her condition was properly monitored. The defense also maintained that the movement disorder only developed after she was started on different medication which caused her abnormal movements. A defense verdict was returned despite very significant specials. \textit{Defense Verdict in Suit Alleging Negligence in Prescribing Risperdal}, 35 No. 5 \textit{VERDICTS, SETTLEMENTS \& TACTICS NL 31} (2015).

\textsuperscript{150} Id. at *2.
\textsuperscript{151} Id.
\textsuperscript{152} Id. at *3
\textsuperscript{153} Id. at *12.
\textsuperscript{154} Id.
\textsuperscript{155} Id.
\textsuperscript{156} Id.
\textsuperscript{157} Id. at *14.
In *Nail v. Farmers Insurance Exchange*, the court denied a claim for medical benefits stemming from a car accident. The plaintiff was rear ended at a stoplight and diagnosed with a movement disorder involving the basal ganglia. The matter proceeded to trial, and the plaintiff received more than $60,000 in allowable medical expenses. The court, however, denied the plaintiff’s request for attorney’s fees based upon the testimony of the independent medical examination (IME) physician who stated that he “observed several inconsistencies in the plaintiff’s test performance” and that she had “multiple indicators that non-neurological factors had significant bearings on these results.”

In *Kraft v. Colvin*, the plaintiff brought a claim for Social Security disability benefits based on her difficulty speaking and involuntary movements. The Administrative Law Judge (ALJ) found that Kraft suffered from a psychogenic movement disorder that involved spasms, shaking, and jerking of different body parts, as well as balance issues. The problem was that there was no physical reason for her abnormal movements, and they were generally considered stress related. When her claim was denied, Kraft appealed, claiming that the judge improperly concluded that “psychogenic” is the same thing as “malingering,” and she was able to control her symptoms. In rejecting the claim, the court relied upon the opinion of a doctor who noted that Kraft showed evidence of embellishment. The plaintiff countered by arguing that her symptoms may have a psychological basis, but her somatic complaints were not intentional or under her control. On appeal, the court rejected these arguments and stated that there was no evidence that the trial judge rejected the idea that the symptoms could be psychological. To the contrary, the ALJ found that there was a psychological component to her symptoms that were aggravated by stress. The judge was also entitled to discount the plaintiff’s testimony based upon the doctor’s opinion that she was embellishing her symptoms. However, the appellate court reversed the denial of benefits because the ALJ failed to consider the doctor’s testimony that the claimant had a serious

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159 Id. at *1, *2.

160 Id.

161 Id.


163 Id.

164 Id.

165 Id. at *3.

166 Id.

167 Id.

168 Id. at *3, *4.

169 Id. at *4.

170 Id.
impairment as the result of her pain, depression, and physical limitations.\textsuperscript{171} The court noted that the trial judge could not rely on one portion of the physician’s testimony while ignoring other critical parts.\textsuperscript{172}

\textit{Hewitt v. Union Oil Co.} involved a claim under the Americans with Disabilities Act (ADA).\textsuperscript{173} The question was whether a sleep and movement disorder could result in a disability under federal and state law.\textsuperscript{174} The facts demonstrated that Hewitt was discharged by his employer.\textsuperscript{175} However, he suffered from a sleep and limb movement disorder that caused sleep deprivation, which allegedly constituted a physical impairment under the law.\textsuperscript{176} The trial court dismissed the claim, but the Court of Appeals reinstated the complaint and ordered the judge to ascertain whether the plaintiff’s condition constituted a claim.\textsuperscript{177}

\textbf{B. Dystonia}

Dystonia is “a movement disorder characterized by involuntarily muscle contractions that contort certain body parts into abnormal and painful postures.”\textsuperscript{178} Dystonia has a connection to trauma and may follow an injury to the head or another body part.\textsuperscript{179} It has also been seen following a visit to the dentist, surgery, injury to the eyes, and whiplash injuries.\textsuperscript{180} Proximate cause issues may also arise because the symptoms may not surface for months or years following the traumatic event.\textsuperscript{181} Counsel should be aware that following a head injury, the dystonia symptoms may affect the opposite side of the brain injured by trauma.\textsuperscript{182} Terminology applied to depict trauma-induced dystonia includes: “injury-induced, peripherally-induced, post-traumatic dystonia, causalgia-dystonia syndrome, reflex sympathetic dystrophy with dystonia.”\textsuperscript{183}

\begin{itemize}
\item \textsuperscript{171} Id. at *5.
\item \textsuperscript{172} Id. at *6.
\item \textsuperscript{173} See Hewitt v. Union Oil Co. of Cal., 44 F. App’x 827, 827 (9th Cir. Aug. 16, 2002).
\item \textsuperscript{174} Employee Gets Chance to Show Sleep Disorder is Disability, 17 No. 2 ANDREWS EMP. LITIG. REP. 8 (2002).
\item \textsuperscript{175} Id.
\item \textsuperscript{176} Id.
\item \textsuperscript{177} Id.
\item \textsuperscript{180} See More Info: Trauma-Induced Dystonia, DYSTONIA MED. RES. FOUND., https://www.dystonia-foundation.org/what-is-dystonia/forms-of-dystonia/secondary-dystonia/trauma/more-on-trauma (last visited Feb. 6, 2017) [hereinafter More Info: Trauma-Induced Dystonia].
\item \textsuperscript{181} See \textit{Trauma—Quick Facts}, supra note 179.
\item \textsuperscript{182} Id.
\item \textsuperscript{183} More Info: Trauma-Induced Dystonia, supra note 180.
\end{itemize}
From a defense point of view, counsel should be aware that the precise cause of dystonia is unknown. The best that can be said is that this condition was caused by an irregularity in or an injury to the basal ganglia or other brain areas that control movement. The “unknown etiology” defense, however, cannot be used in isolation. It must be buttressed by the appropriate scientific articles and expert opinions and be considered along with the other evidence.

*Hedrick v. PPG Industries* provides an example of the need for additional evidence to prove dystonia. This was an appeal from a worker’s compensation determination in favor of an employee. The evidence demonstrated that the plaintiff was injured at work when she fell on her buttocks and lower back. A couple of months later, she started to have involuntary tremors of her head, trunk, and extremities. A neurologist determined that her movement disorder was not psychological but was an unspecified type with dystonia qualities. The employee went on medical leave and stopped working. She was awarded temporary, total disability and medical compensation, and the defendant-employer appealed. The defendant argued that there was no scientific proof that the trauma caused the dystonia, and the evidence merely raised a possibility that the condition was causally related to the fall. A second neurologist stated that the initial injury, along with the surgery, caused the dystonia or triggered a pre-existing dormant condition. The defense expert testified that there was little data to support the principle that peripheral trauma can cause dystonia and many people have neck surgery and never develop this problem. The court dismissed the defense’s arguments and stated that the textbooks on movement disorders and medical literature widely accept the idea that trauma can cause dystonia.

A similar result occurred in *Smith v. Dillard’s Dept. Stores, Inc.*, although the plaintiff’s physicians testified that “there are many causes of dystonia, and in some instances, a definite cause cannot be found.” The doctor admitted that “because of the complexity of the brain, the experts ‘don’t know exactly how to explain

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184 *Dystonias Fact Sheet*, supra note 178.
187 *Id.* at 854.
188 *Id.*
189 *Id.*
190 *Id.*
191 *Id.* at 855.
192 *Id.*
193 *Id.* at 856.
194 *Id.*
195 *Id.*
196 *Id.*
197 *Smith v. Dillard’s Dep’t Stores, Inc.*, 2000-Ohio-2689 (8th App. Dist.).
peripheral trauma causing dystonia.\textsuperscript{198} The expert for the defense opined that the plaintiff’s dystonia could have been caused by peripheral trauma but did not believe that it was possible.\textsuperscript{199} In denying the defendant’s motion for summary judgment, the court did not rule on whether dystonia can arise from trauma and did not rely solely on the diagnosis of various physicians.\textsuperscript{200} The thrust of the defendant’s argument was that the scientific community is unable to explain how trauma can cause the movement disorder.\textsuperscript{201} The court resolved the conflict by ruling that the challenge merely went to the weight of the evidence and not to the reliability of the evidence.\textsuperscript{202}

Courts have reached dystonia verdicts or settlements in excess of $1 million in a number of cases. Donnellan v. First Student, Inc. resulted in a $6 million award as the result of a school bus accident.\textsuperscript{203} The plaintiff was a thirty-one-year-old man who claimed that he sustained a number of permanent injuries, including a cranial nerve injury and dystonia.\textsuperscript{204} The defense expert testified that he was not able to properly evaluate the plaintiff because the claimant gave an insufficient effort during the exam, and he thought the claimant had a somatization disorder or preoccupation with his health without a physical reason.\textsuperscript{205} On appeal, the court upheld the verdict.\textsuperscript{206}

The court rendered a $7 million verdict in Mac Morris v. Rides-R-U’s.\textsuperscript{207} This unusual fact pattern involved a kiddie ride that was not properly grounded and caused the plaintiff to be shocked when she brushed against it, causing her to suffer traumatic dystonia to the hand.\textsuperscript{208} She claimed that her injury caused extensive problems with the ability to engage in everyday tasks, including taking care of her young children.\textsuperscript{209} The defense expert questioned the relationship between dystonia and electrical shock and argued that the plaintiff merely developed a hysterical conversion that would get better with therapy.\textsuperscript{210} The jury awarded the plaintiff $7

\begin{thebibliography}{99}

\bibitem{198} {Id. at *2.}
\bibitem{199} {Id. at *3.}
\bibitem{200} {Id.}
\bibitem{201} {Id. at *1.}
\bibitem{202} {Id. at *9; see also Gordon v. Trumbull Memorial Hosp., 56 N.E.3d 1013 (Ohio Ct. App. 2016).}
\bibitem{203} {Donnellan v. First Student, Inc., 891 N.E.2d 463 (Ill. App. Ct. 2008).}
\bibitem{204} {Id. at 471.}
\bibitem{205} {Id. at 472.}
\bibitem{206} {Id.}
\bibitem{208} {Id.}
\bibitem{209} {Id.}
\bibitem{210} {Id.}

https://engagedscholarship.csuohio.edu/clevstlrev/vol65/iss3/5
million, but the parties had entered in a high/low agreement, so the award was reduced to the agreed upon higher amount.211

*Kneile v. Montefiore Medical Group* dealt with a medical malpractice claim involving an ulnar nerve injury that developed after the insertion of contraceptive implants into the plaintiff’s left arm.212 The defense claimed that the injury occurred when the plaintiff underwent additional surgery to have the implants removed.213 Kneile eventually developed atrophy in the extremity and was diagnosed with dystonia. A verdict was rendered in favor of the patient in the sum of $2.25 million.214

Not all lawsuits involving dystonia are so dramatic, and defense verdicts have been rendered on a number of occasions. *Lawton v. Coker* involved a sixty-eight-year-old man who claimed that he developed cervical dystonia as the result of a rear end car accident.215 The defendant admitted that he caused the collision but denied that the dystonia was traumatically induced because it did not develop immediately after the incident.216 The plaintiff demanded $300,000 during trial, and the defense countered with a $10,000 settlement offer that was rejected.217 The jury returned with a defense verdict, and a motion for a new trial was denied.218

*Hansen v. Walmart Stores, Inc.* is a middle-of-the-road case that involved a Walmart customer who slipped and fell on a wet floor.219 The plaintiff alleged injuries that included cervical focal dystonia.220 The defendant denied liability, and the fact finder determined that both parties were 50% at fault, awarding the plaintiff $40,481.221

211 Id.


213 Id.

214 Id.; see also Verdict and Settlement Summ., Adams v. The Imported Car Store, No. 05-2009-CA-54698 (Fla. Cir. Ct. Nov. 13, 2013), in which a $28.5 million verdict was rendered in favor of the plaintiff who was assaulted by the manager of a car agency. The defendant head butted the claimant causing him to hit his head on the cement. Id. The plaintiff developed dystonia which caused painful muscle contractions and a distorted posture. Id. The defense argued that the plaintiff merely sustained a minor concussion and that a head butt did not cause the dystonia. Id.


216 Id.

217 Id.

218 Id. A defense verdict was also entered in *Samuels v. Winn-Dixie Midwest, Inc.* See Verdict and Settlement Summ., Samuels v. Winn-Dixie Midwest, Inc., No. 99-CI-928 (Ky. Cir. Ct. May 1, 2001).


220 Id.

221 Id. The defendant offered $75,000 in settlement, which was rejected.
C. Tics

A Westlaw search uncovered only a small number of cases involving tics.222 In one example, *Fattal v. Leye*, the passenger in a cab claimed an accident aggravated a pre-existing tic disorder.223 The defense filed a motion for summary judgment to dismiss the claim for failure to demonstrate a serious injury.224 The defense stated that an IME showed the plaintiff had a normal neurological exam with full range of motion.225 She also only missed ten days of work.226 Fattal’s medical expert countered that the plaintiff informed him that her Tourette’s had become worse since the incident, and medical articles contain “multiple cases of tics that start or worsen after trauma.”227 The court granted the defendant’s motion because the plaintiff presented no triable issue of fact to show a serious injury.228 The court stated that the plaintiff

[had] not raised an issue of fact regarding her claimed exacerbation of Tourette’s because there [was] no indication that the doctor she saw reviewed a single record, had any objective assessment of her pre-accident Tourette’s and only concluded that any increase in tics may be related to the accident—which [was] speculative and conclusory.229

Another example, *Ex parte Rhea*, involved a worker’s compensation claim arising out of two separate car accidents.230 In the first accident, the worker began to have spontaneous muscular contractions in the nature of facial tics but did not seek worker’s compensation benefits.231 Six years later, he was involved in a second accident and applied for benefits on the basis of spontaneous muscle spasms resulting in the inability to work.232 The Civil Court of Appeals dismissed the claim, finding the problem was related to the first accident and barred by the statute of limitations.233 The appellate court reversed this determination after finding it significant that the worker had never applied for a loss of earning capacity as the result of the first accident.234 While the first incident predisposed the claimant to the


224 *Id.* at *2.

225 *Id.* at *2-3.

226 *Id.* at *3.

227 *Id.*

228 *Id.*

229 *Id.*

230 *Ex parte* Rhea, 807 So.2d 541 (Ala. 2001).

231 *Id.* at 542.

232 *Id.* at 542-43.

233 *Id.*

234 *Id.* at 545.
second injury, there was no loss of earning capacity claim until the second event. Therefore, the suit was not time barred.\textsuperscript{235}

In \textit{Rogers v. Moody}, a twenty-year-old man was in a car accident, struck his head on the windshield, and cut his face on the glass.\textsuperscript{236} He also severed muscles in the face that left him with a facial tic that accentuated his facial scars.\textsuperscript{237} In this case, the court found that the jury’s award of $40,000 was not excessive.\textsuperscript{238}

The courts are not always impressed with the development or aggravation of a tic. That was the case in \textit{Thomas v. Encompass Insurance}, which involved a motor vehicle accident that exacerbated nervous tics related to Tourette’s syndrome.\textsuperscript{239} While the court noted the accident was a stressful event, it found that medication resolved the tics.\textsuperscript{240} Likewise, in \textit{Schramm v. Long Island Railroad Company}, the plaintiff was hit in the back of the head by a large tree branch and sustained head and neck injuries.\textsuperscript{241} He was diagnosed with a concussion, neck spasms, and a facial tic and missed five months of work.\textsuperscript{242} The jury returned a verdict of $250,000, but the court granted a new trial on the basis that the verdict was excessive.\textsuperscript{243}

\textbf{D. Tardive Dyskinesias}

\textit{Dyskinesias} is a neurological problem that has been the subject of many lawsuits. As for a definition, \textit{Jenkins v. Bristol Myers Squibb} offers the following: “Tardive dyskinesia is a neurological disorder that causes irregular, involuntary muscular movements and can affect the limbs, upper extremities, trunk and facial muscles.”\textsuperscript{244} Most cases involving this disorder are related to taking drugs that affect the chemicals in the brain, and trauma does not seem to play a role in the disorder.\textsuperscript{245} This opinion, however, is not unanimously accepted, and at least one medical text notes that “brain injury is a risk factor for tardive dyskinesia.”\textsuperscript{246} Nevertheless, the

\begin{quote}
\begin{footnotesize}
\begin{enumerate}
\item \textsuperscript{235} Id.
\item \textsuperscript{236} Rogers v. Moody, 242 A.2d 276, 277-78 (Pa. 1968).
\item \textsuperscript{237} Id. at 278.
\item \textsuperscript{238} Id. at 279.
\item \textsuperscript{240} Id.
\item \textsuperscript{242} Id. at 258.
\item \textsuperscript{243} Id. at 259.
\item \textsuperscript{245} Mark Hallett, \textit{Blepharospasm and Tardive Dyskinesia}, 31 \textit{Benign Essential Blepharospasm Res. Found. NewsL.} 4 (2012).
\item \textsuperscript{246} Robert P. Granacher, Jr., \textit{Traumatic Brain Injury: Methods for Clinical and Forensic Neuropsychiatric Assessment} 394 (2d ed. 2007).
\end{enumerate}
\end{footnotesize}
\end{quote}
reported cases focus on the relationship between medication and tardive dyskinesia.\textsuperscript{247}

\textbf{E. Parkinson’s Disease}

Scientific studies have shown a link between Parkinson’s disease and head or brain trauma resulting from such things as motor vehicle accidents, sports injuries, and falls.\textsuperscript{248} This risk increases with more severe or repetitive head injuries.\textsuperscript{249} In fact, people over fifty-five years old who have been seen in the hospital for a traumatic brain injury have a 44% chance of developing Parkinson’s disease over the next six years.\textsuperscript{250}

Litigation regarding Parkinson’s disease includes the need for reasonable accommodations,\textsuperscript{251} the connection between arsenic poisoning and the disease,\textsuperscript{252} environmental factors and pesticides,\textsuperscript{253} medication causing Parkinson’s disease,\textsuperscript{254} loss of employment because of Parkinson’s,\textsuperscript{255} exposure to welding fumes as the cause for developing Parkinson’s,\textsuperscript{256} manganese fume exposure,\textsuperscript{257} a Parkinson’s

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\textsuperscript{249} \textit{Id}.

\textsuperscript{250} \textit{TRAUMATIC BRAIN INJURY LATE IN LIFE INCREASES PARKINSON’S RISK}, Parkinson’s Disease Found. (JUNE 5, 2015), http://WWW.PDF.ORG/EN/SCIENCE_NEWS/RELEASE/PR_1433778887 (DISCUSSING RAQUEL C. GARDNER ET AL., TRAUMATIC BRAIN INJURY IN LATER LIFE INCREASES RISK FOR PARKINSON DISEASE, 77 ANNALS NEUROLOGY 987, 991-93 (2015)).

\textsuperscript{251} James Boyan, \textit{Employers are Not Required to be Mind Readers}, 20 N.J. EMP. L. LETTER 3, 3 (2012).

\textsuperscript{252} \textit{Film Processing Worker Petitions California Supreme Court to Review Toxic Exposure Case: Waterman v. Carlton Communications/Technicolor}, 18 ANDREWS TOXIC CHEMICALS LITIG. REP. 9, 9 (2000).

\textsuperscript{253} 110436 Parkinson’s Disease, 8 OCCUPATIONAL MED. DIG. 13, 13 (1996).


\textsuperscript{257} \textit{Court Ok\'s Testimony Linking Welding Fumes to Parkinson’s Disease: In re Welding Fume Prods. Liab. Litig.}, 27 ANDREWS ASBESTOS LITIG. REP. 9, *1 (2005).
drug causing a gambling habit,\textsuperscript{258} Parkinson’s and the ADA,\textsuperscript{259} the link between carbon monoxide poisoning and Parkinson’s,\textsuperscript{260} and negligence in the failure to diagnosis and treat the disease.\textsuperscript{261}

The court cases linking trauma and Parkinson’s disease over the past fifty to seventy-five years have gone through a metamorphosis. At one time, the medical community did not believe that a connection existed, so lawsuits trying to establish such a link were unsuccessful. \textit{Brown v. Los Angeles Transit Lines} was a 1955 case involving a patron who was injured in an accident while on a street car.\textsuperscript{262} The plaintiff alleged that her Parkinson’s disease was caused by the accident, but the defense countered that her condition was due to an emotional disturbance.\textsuperscript{263} A physician from the University of Chicago testified that Parkinson’s does not result from trauma, and another physician noted that in the more than 10,000 patients he has seen only one case was related to an accident.\textsuperscript{264} Under the circumstances, the judge concluded that there was insufficient evidence to warrant finding a link between trauma and Parkinson’s syndrome.\textsuperscript{265} The court reached a similar result in the 1944 case of \textit{Aluminum Co. v. Industrial Commissions}, in which an injured person was not able to return to work because the accident aggravated his pre-existing Parkinson’s condition.\textsuperscript{266} The medical testimony from the parties was remarkably similar, and they both agreed that the plaintiff had Parkinson’s disease for some time before the accident and that it was virtually impossible for the disease to be aggravated by trauma.\textsuperscript{267} As a result, the lower court’s grant of compensation was set aside.\textsuperscript{268}

A shift in position was evident several decades later in \textit{Mancuso v. Mancuso}.\textsuperscript{269} The plaintiff was involved in an accident and sustained what appeared to be

\begin{itemize}
  \item Harper Gerlach, \textit{Appeals Court Reverses Disability Bias Decision Involving Miami Police Officer with Parkinson’s}, 23 \textsc{Fla. Emp. L. Letter} 6, 6 (2011).
  \item $3.7 Million Verdict in Suit Arising from Carbon Monoxide Poisoning from Motel Boiler, 19 \textsc{Verdicts, Settlements & Tactics} 548, 548 (1999).
  \item \textit{Id.} at 811.
  \item \textit{Id.} at 812-13.
  \item \textit{Id.} at 816.
  \item Aluminum Co. of Am. v. Indus. Comm’n, 152 P.2d 297, 299 (Ariz. 1944).
  \item \textit{Id.}
  \item \textit{Id.} at 302; \textit{contra} Moffett v. Bozeman Canning Co., 26 P.2d 973, 978 (Mont. 1933); Barkhurst v. Dep’t of Labor & Indus. of Wash., 274 P. 105, 105 (Wash. 1929); Natalini v. Riefler & Sons, 133 A. 547, 548 (Pa. 1926); Hartford Accident & Indem. Co. v. Indus. Comm’n of Utah, 228 P. 753, 754 (Utah 1924).
\end{itemize}
superficial injuries.\textsuperscript{270} Two years later, she started to develop neurological issues and was diagnosed with Parkinson’s disease.\textsuperscript{271} One year after the original diagnosis, a neurologist confirmed the disease and noted that the condition was aggravated by the accident.\textsuperscript{272} The treating physician opined that while it was not possible to demonstrate that the impact of the accident was the actual cause of the disorder, there was no doubt that the incident aggravated her Parkinson’s disease.\textsuperscript{273} The plaintiff filed suit two years and one month after the incident and eight months subsequent to when she learned that the disease was related to the accident. As such, the defense raised the two-year statute of limitations as a bar to the claim,\textsuperscript{274} but the court allowed the lawsuit to proceed, noting that the connection between Parkinson’s disease and trauma is not common knowledge and requires a highly specialized medical awareness to connect the two.\textsuperscript{275} Therefore, the statute did not start to run until the plaintiff was made aware of that link.\textsuperscript{276}

The cases involving Parkinson’s and trauma have had mixed results. For instance, a $500,000 verdict was rendered in a case involving an automobile accident, \textit{Kiska v. Deleon},\textsuperscript{277} where the plaintiff sustained a brain hematoma that led to the plaintiff’s Parkinson’s disease.\textsuperscript{278} On the other hand, a zero verdict was returned in \textit{Jones v. Cervantes},\textsuperscript{279} where the plaintiff claimed that his Parkinson’s disease had become progressively worse since a car accident. A chiropractor testified that the patient had suffered permanent injuries and would need $20,000 in future medical care.\textsuperscript{280} But on cross-examination, the plaintiff’s expert admitted that Parkinson’s disease becomes progressively worse with time.\textsuperscript{281} The jury returned with a verdict within thirty minutes, demonstrating that they did not believe the aggravation of the disease was related to the accident.\textsuperscript{282}

\begin{itemize}
  \item \textsuperscript{270} \textit{Id.} at 1254.
  \item \textsuperscript{271} \textit{Id.}
  \item \textsuperscript{272} \textit{Id.}
  \item \textsuperscript{273} \textit{Id.}
  \item \textsuperscript{274} \textit{Id.}
  \item \textsuperscript{275} \textit{Id.} at 1257.
  \item \textsuperscript{276} \textit{Id.} at 1255.
  \item \textsuperscript{277} \textit{Kiska v. Deleon}, No. 85L-154, 1986 WL 311502 (Ill. Cir. Ct. Feb. 18, 1986).
  \item \textsuperscript{278} \textit{Id.} Examples of other cases in which the fact finder linked the aggravation of a person’s Parkinson’s disease to trauma include: No. 9833905, 1998 WL 35215218, at *1 (BVA Nov. 17, 1998); No. 9600674, 1996 WL 33636504, at *2 (BVA Jan. 22, 1996); No. 9516951, 1995 WL 17181950, at *2 (BVA Aug. 29, 1995) (case titles redacted by Department of Veterans’ Affairs).
  \item \textsuperscript{280} \textit{Id.}
  \item \textsuperscript{281} \textit{Id.}
  \item \textsuperscript{282} \textit{Id.} For examples of other cases in which the fact finder did not believe trauma aggravated a person’s Parkinson’s disease, see Goldstein v. Prudential, 117 A.D.3d 1368,
F. Chorea

Very few cases discuss trauma and chorea. Surprisingly, there is a case that mentions chorea as early as 1909. In McCarthy v. Philadelphia Rapid Transit Co., a child was involved in a collision between a car and bus.\(^{283}\) McCarthy was treated at a hospital and by his family doctor after sustaining a laceration to his cheek and was nervous for a period of time.\(^{284}\) The plaintiff recovered within three months, but developed chorea one and a half years later.\(^{285}\) No one attempted to link the chorea to the accident, but the testimony demonstrated that chorea in children could be caused by injury, fright, and disease.\(^{286}\) The court did not allow the jury to consider the cause of the chorea because it was too remote.\(^{287}\) It was not established until later that post-traumatic chorea can have an acute origin or it can have a considerable delay in development.\(^{288}\)

Sloan v. Original Stage Line, Inc. reached a contrary result.\(^{289}\) Sloan was a passenger on a bus and claimed that she struck her neck against the window and hit her head on the floor when the bus was in an accident.\(^{290}\) As a result, the plaintiff developed a constant twitching and jerking of the muscles and was diagnosed with traumatic chorea.\(^{291}\) This condition was described as a “disorder of the motor nerves [that] causes a kind of jerking or contraction of the nerves.”\(^{292}\) A verdict was rendered in favor of the plaintiff and was upheld on appeal.\(^{293}\)

In Smith v. Reeves, a passenger in a car accident claimed that she sustained a brain injury resulting in a “chorea-like” movement disorder of her extremities.\(^{294}\) The plaintiff did not seek medical care for ten months following the accident, thereby making any link between the injury and the accident speculative.\(^{295}\) All diagnostic studies failed to show any abnormality, and no testimony was offered to adequately


\(^{284}\) Id.
\(^{285}\) Id.
\(^{286}\) Id.
\(^{290}\) Id.
\(^{291}\) Id.
\(^{292}\) Id.
\(^{293}\) Id. at 465-66. The court awarded plaintiff $10,000, which in 1932 would have been considered a lot of money.
\(^{295}\) Id.
explain how the chorea was related to the accident. On appeal, the court noted that the trial judge should have granted the defense’s motion for summary judgment on the basis that the plaintiff did not sustain a serious injury, a threshold requirement under New York law.

In Gomez v. United States, the plaintiff brought a claim under the Federal Torts Claims Act (FTCA). The facts show that the plaintiff was rendered unconscious in an accident and was diagnosed with brain damage and involuntary chorea. A neurologist examined Gomez and noted that his chorea “could be related to the traumatic head injury; however, an underlying genetic disorder, namely Huntington’s chorea could be present.” The plaintiff’s expert stated that Gomez had the gene mutation for Huntington’s disease, but 50% of the patient’s symptoms were a “direct residual of the head injury.” The defendant countered that the court should bar the expert’s testimony, as it was not premised upon sufficient facts or data; however, the court allowed the testimony because the expert relied upon studies and the patient’s history.

G. Tremors

The majority of tremor cases appear to arise in a Social Security context and are unrelated to trauma. Nevertheless, tremors have found their way into the courtroom in a variety of other ways with varying success. Moore v. Bi-State Development Agency involved a minor who was struck by car while leaving a bus. He sustained multiple injuries, including a traumatic brain injury, multiple fractures, and voice tremors. As a result, he was left wheelchair bound and in need of total assistance with activities of daily living. The case was defended on liability, and the jury returned with a $7.75 million verdict, which was reduced by 49% due to the plaintiff’s contributory negligence.
Bartlett v. Snappy Car Rental, Inc. resulted in a six-figure award for injuries that included tremors, but the verdict was reduced on appeal. The plaintiff was involved in a car accident that required her to be hospitalized. She suffered from pre-existing tremors that were significantly aggravated to the extent that they became debilitating and prevented her from leading an active life. The defendant asserted that it was not liable and that the evidence was insufficient to support the finding of liability. The jury awarded $700,000 in damages, but the verdict was found to be excessive and reduced to $385,000.

In Brown v. Shimkin, the plaintiff—an eighty-year-old woman—was struck while crossing the street and claimed injuries to her neck and back, as well as tremors and paresthesia. The physician who performed an IME noted that the plaintiff had a normal neurological exam and her limitation in movement was age appropriate. The court was critical of the physician’s testimony because he failed to consider an electromyography (EMG), which was relevant to the plaintiff’s claim of tremors and paresthesia. In denying the defendant’s motion for summary judgment, the court noted that the defendant failed to meet the burden of proof and the IME doctor did not discuss what objective tests he used in forming his conclusions.

On many occasions, the courts have been unimpressed with claims involving tremors. Rubenstein v. Senkier offers such an example. The plaintiff in Rubenstein was involved in a motor vehicle accident that fractured his sternum and caused soft tissue injuries to his neck and lower back. The plaintiff asserted that these injuries exacerbated a pre-existing hand tremor that prevented him from continuing his job as a pediatrician, and his allegation was supported by the testimony of a neurologist. The defense countered that the tremor was nothing more than a pre-existing condition related to a genetic medical syndrome and the accident merely caused a transient worsening of the tremors. Further evidence was produced that the plaintiff had been in an earlier accident that made his tremors worse, and a verdict

308 Id.
309 Id. at 597.
310 Id.
311 Id. at 596, 598.
313 Id.
314 Id.
315 Id.
317 Id.
318 Id.
319 Id.
was returned in favor of the defendant.\textsuperscript{320} The jury explained that it believed the hand tremors were a longstanding problem unrelated to the accident.\textsuperscript{321}

The defense achieved a similar result in \textit{Mavrides v. Allstate}.\textsuperscript{322} This matter involved a rear end collision in which the plaintiff, a man in his seventies, claimed that he developed a tremor in his hand as the result of a brain injury sustained in the accident.\textsuperscript{323} The plaintiff’s expert stated that the brain trauma related to the accident caused the tremor.\textsuperscript{324} The defendant maintained that the tremor was not related to the incident, and photographs of the plaintiff’s car showed only a minor impact. A verdict for the defense was returned.\textsuperscript{325}

\textbf{IV. Conclusion}

The above is a discussion of post-traumatic movement disorders often resulting from head trauma that is usually severe in degree. The specific types of hyperkinetic movement disorders (excessive abnormal movements) include tremor, dystonia, tics, myoclonus, and chorea. Parkinson’s disease is the only hypokinetic disorder (diminished abnormal movements). These conditions occur as the result of either trauma or pharmacological damage to structures of the basal ganglia and the brain stem.\textsuperscript{326} In some cases, imaging studies such as the MRI scan demonstrate the injury; in other cases, no pathological changes are seen within the brain. While trauma can be the cause of these movement disorders, utmost care must be made to obtain a detailed history prior to the trauma to ascertain that the abnormal movements were not a pre-existing condition.

There is a clear connection between brain trauma and movement disorders, so counsel should not overlook this link when handling a personal injury claim. However, as the cases demonstrate, an aggravation of a pre-existing movement disorder by itself does not guarantee the awarding of compensation.

\textsuperscript{320} \textit{Id.}

\textsuperscript{321} \textit{Id.}


\textsuperscript{323} \textit{Id.}

\textsuperscript{324} \textit{Id.}


\textsuperscript{326} See generally Krauss & Jankovic, \textit{Head Injury and Posttraumatic Movement Disorders}, \textit{supra} note 22.