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**PARKINSONISM AND
TRAUMATIC BRAIN INJURY¹**

I. INTRODUCTION

Proving a causal relationship at trial between trauma and Parkinson's disease or parkinsonism² is difficult, as evidenced by the fact that only three cases have proceeded to trial in Canada. This difficulty stems in part from medical science's

limited understanding of the process responsible for the condition, and to physicians' natural desire to attain a high level of certainty before concluding that trauma has a causal role in parkinsonism. While there is still controversy in the medical literature concerning the role of trauma in the etiology of parkinsonism, there seems to be little doubt among the majority of medical experts that a traumatic brain injury can cause parkinsonism. The controversy is over the nature and degree of brain injury required.

II. HISTORICAL PERSPECTIVE

The idea that trauma can have a bearing on the occurrence of Parkinson's disease is rooted in the history of medicine. This suggested relationship dates back to the first description of the condition as a separate clinical entity in 1817 by the eminent London physician, Dr. James Parkinson. In his "Essay on the Shaking Palsy", Parkinson coined the Latin phrase "paralysis agitans" to describe the onset of a set of symptoms in his patient, the Count de Lordat,³ who had been injured when his coach overturned. In the accident the patient struck his head and suffered bruising to his left shoulder, arm and left hand. Symptoms of the disease were first reported one year later when the patient noticed rigidity in his left arm.

During the next fifty years trauma was never considered to be an important factor in the etiology of this condition as it was rarely mentioned in patients' histories, possibly for the reason that the cause was rarely of consequence to the injured party. This changed in the latter part of the 19th century with the introduction of worker compensation legislation. Reported cases of trauma preceding the diagnosis of "paralysis agitans" became common.

In the 1860's the renowned French neurologist, Pierre Marie Charcot, examined a number of trauma cases that later developed classic symptoms of "paralysis agitans", which he renamed "Parkinson's disease". Charcot felt that in true cases of Parkinson's disease caused as a result of trauma, the site of the injury must also be the site of the first outward manifestations of the disease.⁴ The majority of cases presented in the literature over the next 50 years considered only peripheral trauma, and not injury to the central nervous system, as a causal factor.⁵ Thereafter, in the early part of the 1900's the prevailing view came to be that trauma must somehow involve the brain and that the peripheral trauma as proposed by Charcot was not sufficient to cause the disease.⁶

III. WHAT IS PARKINSON'S DISEASE?

A. Definition

Parkinson's disease belongs to a group of neurological conditions called movement disorders. The four primary symptoms are:

1. **tremor** or trembling in hands, arms, legs, jaw, and face;

2. **rigidity** or stiffness of the limbs and trunk;
3. **bradykinesia** or slowness of movement;
4. **postural instability** or impaired balance and coordination.

Parkinson's disease is a neurological disorder that affects specific areas of the brain that are involved in the control of voluntary movement. The exact biochemical cause of the disease remains unknown. It is known that with aging there is a progressive loss of a class of dopamine-producing neurons in a small midbrain nuclei called the substantia nigra ("black substance") but it is not understood why this cell loss is accelerated in those people who develop Parkinson's disease.

B. Neurological Locus of the Disorder

Dopamine is a neurotransmitter produced by a small cluster of neuronal cell bodies in the substantia nigra. These cells project their axons to an area of the brain known as the striatum where the release of dopamine affects the activity of the striatal neurons. Thus, this pathway is called the nigrostriatal pathway. The striatum is a major part of the basal ganglia, a network of brain structures that are of major importance in the control of smooth, coordinated muscular activity.⁷

Among its many functions, dopamine is known to play a major role in two activities of the central nervous system: one that helps control movement, and a second that is strongly associated with emotion-based behaviors. Thus, emotional symptoms such as depression are commonly seen with the development of Parkinson's disease.

In addition to the nigrostriatal pathway involved in movement control, the second major dopamine pathway in the brain is the mesocorticolimbic pathway. Via this pathway, dopamine neurons project to other neurons in various parts of the brain's limbic system, which regulates emotion and motivation.

There is no cure for Parkinson's disease, but there are a variety of medications that provide symptomatic relief. In the mid-1960s, the drug L-dopa (levodopa) was found to relieve Parkinson's symptoms. Its mechanism of action is that it causes the surviving neurons in the nigrostriatal pathway to increase production of dopamine. Over time, however, levodopa becomes less effective in relieving symptoms and produces very significant side effects, which will eventually become more debilitating to the patient than the parkinsonian symptoms. For this reason, it is now the generally accepted approach with younger patients to use dopamine agonists, such as bromocriptine, before introducing levodopa.⁸

C. Diagnosis

The diagnosis of Parkinson's disease is based on clinical judgment and there is no specific laboratory test to confirm the diagnosis. The initial clinical diagnosis is based on the identification of some combination of the cardinal motor signs of tremor, rigidity, bradykinesia, and postural instability.

The average age of onset of the symptoms is 60. It is very unusual for Parkinson's disease to be diagnosed in a person under 40 years of age.

D. Early Symptoms and Signs

Early symptoms of Parkinson's disease are subtle and occur gradually. Patients may report feeling tired or that they notice a general malaise. Some may feel a little shaky or have difficulty getting out of a chair. They may notice that they speak too softly or that their handwriting looks cramped and spidery. They may lose track of a word or thought, or feel irritable or depressed for no apparent reason.

Friends or family members may be the first to notice changes. They may observe the person's face to lack expression and animation ("masked face"), or find the person to be clumsy, or to appear stiff, unsteady, and unusually slow.

As the disease progresses, the shaking or tremor that affects the majority of Parkinson's patients may begin to interfere with daily activities. Patients may not be able to hold utensils steady or may find that the shaking makes reading a newspaper difficult. For most patients, it is

tremor that causes them to seek medical help.⁹

Until the patient recognizes that something is wrong and seeks medical attention, they are said to be in a "sub clinical" phase. Studies indicate that the sub clinical phase may persist for 3 to 15 years. This makes it extremely difficult for the medical expert to distinguish Parkinson's disease that is trauma-related from that due to another cause. The diagnosis will depend on the quality of the evidence that can be presented in regards to the patient's level of functioning before and after the trauma.

E. Additional Symptoms

The nature and the intensity of various other parkinsonian symptoms vary from person to person. Depression and emotional changes are common. Memory loss and slower thinking processes may occur, but dementia usually does not occur until the later stages of the disease. Eventually problems with speech and swallowing will occur. Bladder and bowel problems can occur due to the improper functioning of the autonomic nervous system, which is responsible for regulating smooth muscle activity. Sleep may become disrupted. These symptoms will progress to the point that the patient will require full-time care.

IV. PARKINSONISM

Parkinsonism is the umbrella term for a group of disorders in which there are features of the four primary classes of symptoms described above.

Idiopathic Parkinson's disease (often referred to as "Parkinson's disease") is the most common form of parkinsonism. Idiopathic is a term describing a disorder for which no cause is known. In the other forms of parkinsonism, either the cause is suspected or the disorder is known to have occurred as a secondary consequence of another primary neurological disorder. Some examples of secondary parkinsonism are Postencephalitic parkinsonism (a virally-caused syndrome which caused an epidemic of cases after World War I), Wilson's disease, Huntington's disease, Alzheimer's disease, head trauma, and dementia pugilistica or Parkinson pugilistica (more commonly known as "punch drunk syndrome").¹⁰

V. TRAUMATIC BRAIN INJURY AND PARKINSONISM

A. The Medical Literature

There have been many reported cases of Parkinson's disease or parkinsonism developing following head trauma yet the subject remains controversial within medicine. Many reviewers are reluctant to draw any conclusions about cause and effect as the majority of these cases are based on anecdotal information as opposed to having been followed in controlled scientific studies. Only in cases where post-mortem examination of Parkinson patients has confirmed nigrostriatal injury following injury do reviewers such as Schwab,¹¹ Goetz,¹² Factor,¹³ and Koller¹⁴ reluctantly concede the existence of a causal relationship between trauma and Parkinson's disease or parkinsonism. In the remaining cases they argue that the trauma simply precipitated symptom onset, that is, aggravated a sub clinical form of Parkinson's disease. Jankovic¹⁵ makes the point that the interpretation of data is inevitably colored by personal experience and bias. His review of the evidence for post-traumatic movement disorders published in 1994 in *Neurology*, was his effort to "provide an objective and balanced view of this controversial but important topic".¹⁶

B. Diagnostic Criteria

In 1929 Crouzon published the first review of head trauma and parkinsonism, "Le Parkinsonisme Traumatique" and established the following criteria for the diagnosis of posttraumatic parkinsonism:¹⁷

1. Trauma must be sufficiently violent to produce a destruction of some area within the brain.
2. There should be a definite interval following the trauma and before the onset of Parkinson's disease.
3. The interval should be neither too short nor too long.

4. There should be a definite progression of neurological symptoms.
5. Trauma must be of sufficient severity to produce the condition known as *commotio cerebri*.

In an article published in 1932 in the *Journal of Nervous and Mental Disease*, Kulkov reviewed a number of cases of trauma and Parkinson's disease and arrived at similar diagnostic criteria.¹⁸ He stated:

In order to establish a causal connection between the trauma and the paralysis agitans, Bing advances the following points, which are concerning parkinsonism as well:

1. The trauma must be severe enough to be able to cause some cranial lesion or a least call forth symptoms of a *commotio cerebri*.
2. The patient who suffered from the trauma must not have been previously subject to any cerebral symptoms.
3. The parkinsonian symptoms must not follow immediately the trauma: their development ought to be preceded by some prodromal symptoms (cerebral symptoms) which would then gradually develop a characteristic clinical picture. Bing believes that, should these points be strictly observed, the number of cases of traumatic parkinsonism would be reduced to a minimum.

But we think that if we could add here an absolutely clear and precise picture of an objective history showing us the course of the disease from the very moment of the accident (trauma) to the onset of parkinsonism, tracing it point by point... the number of cases of a "pure," unquestionable traumatic parkinsonism would be still more reduced.

C. Is Severe Brain Damage Required?

In a later review article, Schwab and England¹⁹ stated that the number of "published papers on the origin of Parkinson's disease from trauma is unquestionably large enough to justify lawyers claiming before judges and juries that an accident, which was followed some months or years later by Parkinson's disease, was causally related":

In 1964, Lindenberg wrote a careful report on 3 patients with traumatic Parkinson's disease who subsequently died and on whom complete autopsies were performed. Their brains were sufficiently damaged by the injury so that hemorrhagic, traumatic lesions were identified in the vicinity of the substantia nigra. These cases of a postmortem examination to confirm this etiological factor are beyond dispute. This would be the traumatic Parkinson cases that anyone could accept.

These authors seem willing to acknowledge trauma as a cause of Parkinson's disease only in cases of severe brain damage where the damage to the substantia nigra has been confirmed on post mortem examination. They conclude their review of the literature with the following observation:

...trauma as a cause of Parkinson's disease is exceedingly rare, when it is verified by specific evidence of traumatic hemorrhage and destruction to the substantia nigra and the midbrain. Not more than 2 per cent of the cases could possibly be attributed to trauma. The majority of the reported cases are probably those which had an incipient, subclinical, early form of the disease which was aggravated, brought to the surface, and increased in extent by the injury, just as can be caused by a pure emotional stress.

D. Mild Traumatic Brain Injury and Parkinsonism

In an article published in 1934 in the *Journal of Nervous and Mental Disease*, Grimberg published a review of 86 cases in which a causal

relationship between trauma and Parkinson's was suggested. Grimberg came to the conclusion that only 2 of the 84 cases could be considered true cases of posttraumatic parkinsonian syndrome and reformulated the Crouzon criteria:²⁰

First, the trauma must be of sufficient severity to produce definite damage to the brain;

Second, the trauma must be directly to the head or, if not to the head, of such a nature as to indirectly involve the brain, (Barkman's case):

Third, there must be a clear and definite developmental connection between trauma and disease.

Barkman's case was one of the two cases in which Grimberg accepted the causal connection. Grimberg described the clinical history:

Barkman's case No. 86(52), was a miner, 42 years old, giving a past history of epidemic influenza in 1918. In 1922, while descending into the mine, the cable holding the cage in which he was descending broke, and he fell 75 feet landing in the dirt at the bottom of the pit. He lost consciousness for a moment, was disoriented and confused. He recovered his senses rapidly, and he was able to walk up a number of stairs. On his way home, he was shaking with the entire body. He remained in bed for three weeks complaining of pains and numbness. No dizziness, no vomiting. When he first left his bed, he could not walk properly, but dragged his right foot. His right leg was weak. The hhhpains in the right knee and spine were increased when walking. The examination about five months later, showed: Mentality normal, memory poor only for events following immediately after the accident, easily fatigued, slight tremor of the right arm, and occasionally of the left, but under exertion, it spread over the entire body, cranial nerves were all normal, power was diminished in the right upper limb, slight atrophy of the muscles of the right thigh, no rigidity, no sensory changes, no cerebellar symptoms, reflexes were more pronounced on the right than on the left.

Four months later: "The symptoms resembled those of myotonia congenita, especially when beginning to walk." Tremor of the right arm was present, and one month later it developed on the left arm.

Four years after the trauma, an examination showed: Tremor of both arms, tonic contraction on movement of the right hand, pains in the left half of the body and right thigh and chest, rigidity of the right arm and leg, loss of associated movements of the arms when walking, also fixation rigidity of the right arm."

Based on currently accepted diagnostic criteria, it is clear that Barkman sustained a mild traumatic brain injury (he lost consciousness for a moment, was disoriented and confused).^{21, 22, 23, 24}

Even with cases of severe head trauma followed by Parkinson's disease, very few cases come to post-mortem examination. Autopsy results are rarely available in cases of concussion or mild traumatic brain injury, as these are not life threatening injuries. However, Breutsch published one such case study in the 1935 *Journal of Nervous and Mental Diseases*.²⁵ This is the first reported case of posttraumatic parkinsonism following a concussion or mild traumatic brain injury in which the patient's brain was examined. The clinical history was reported as follows:

On February 6, 1931, F.B.K., a laborer age 60 fell a distance of about eight feet from a pile of rubber, landing on his hip and shoulders, and striking the right occipital portion of his head on the concrete floor. He was dazed for a few minutes, but was able to get up without assistance. The skin of the

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scalp was not broken. In walking upstairs to the dressing room, he noticed that he was unable to raise his left foot. He dressed and walked to the streetcar, dragging slightly the left foot. In riding home and later while eating supper, he had a tendency to fall over to one side. In the following night he suffered from intense headache, which persisted for three months. He never vomited and at no time was he unconscious. The following day he returned to work. He did not mention the accident because he was afraid of losing his job. In order to obtain a few days rest, he consulted a physician for a cold, which he had had for several days. In leaving the office, the doctor said to the patient: "what makes you walk so stiff?" He was ordered to bed, where he stayed for ten days. Then he resumed his work as a laborer until April 10, 1931, when he noticed that the muscular rigidity, which had begun in the left side, had spread over the whole body.

Following the patient's death two and one-half years later, an anatomical study of the brain was conducted to determine first, if residual lesions could be found which were traceable to the trauma, and second, if those lesions were in the region of the brain associated with the parkinsonian syndrome. Macroscopic examination of the brain did not reveal any lesions, however microscopic examination revealed lesions in the area of the basal ganglia and the substantia nigra. These lesions were attributed to the trauma to the head suffered in the fall.

The diagnosis of Parkinson's disease is based on a clinical assessment because there is no diagnostic test that is sensitive to the destruction of the cells in the substantia nigra. The cell loss occurs at a microscopic level, well below the resolution of CT scans and MRI scans. The damage to the midbrain is inferred from the symptoms and the clinical presentation. The ability to visualize the actual cellular brain damage

only becomes apparent in those few cases in which the brain has been microscopically examined at autopsy.

In the courtroom, linking trauma to Parkinson's disease would be easier if it could be demonstrated to the trier of fact that microscopic damage can occur in and around the substantia nigra and basal ganglia in concussion or mild traumatic brain injury cases. The medical literature is helpful. Oppenheimer²⁶ examined the brains of five patients each of who had sustained a concussion and died shortly thereafter of unrelated causes. He found microscopic lesions involving the midbrain, the region of the brain that contains the substantia nigra. Similarly, Blumberg reported corroborative findings in his article published in 1994 in *Lancet*.²⁷ He examined the brains of five patients who sustained mild concussive injuries and died of other causes 2-99 days post injury. Two cases were pedestrians struck by cars, two were occupants of cars in accidents, and one was a bicyclist struck by a car. All five patients showed evidence of microscopic lesions in various areas of the brain including the midbrain. None of the patients in either study suffered anything more than an altered state of consciousness or a transient loss of consciousness.

VI. PROOF OF CAUSATION

A. The Medical Standard

The standard applied by medical experts to establish a relationship between trauma and Parkinson's disease is substantially more stringent than that required to meet the legal standard. Medical experts often apply a test that approximates proof beyond a reasonable doubt. Accordingly, in the absence of a post mortem examination confirming midbrain damage, it is hardly surprising that their opinions usually fall into one of three categories:

1. The plaintiff was in a sub clinical phase of Parkinson's disease and the onset of symptoms was merely coincidental with the trauma.

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2. The patient had pre-existing Parkinson's disease and the accident aggravated symptoms for a short period of time.
3. The trauma of the accident triggered Parkinson's disease, but it merely accelerated the disease or the symptom onset by 1 or 2 years.

B. The Legal Standard

The legal standard of causation is whether in all likelihood the trauma was a *material (more than de minimus) contributing factor* in the development of Parkinson's disease or parkinsonism. In *Athey v. Leonati*, 1996 CarswellBC 2295, the Supreme Court of Canada set out the following general principles to guide the trier of fact in the determination of causation:

General principles

Causation is established where the plaintiff proves to the civil standard on a balance of probabilities that the defendant caused or contributed to the injury: *Snell v. Farrell*, [1990] 2 SCR 311; *McGhee v. National Coal Board*, [1972] 3 All ER 1008 (HL).

The general, but not conclusive, test for causation is the "but for" test, which requires the plaintiff to show that the injury would not have occurred but for the negligence of the defendant: *Horsley v. MacLaren*, [1972] SCR 441.

The "but for" test is unworkable in some circumstances, so the courts have recognized that causation is established where the defendant's negligence "materially contributed" to the occurrence of the injury: *Myers v. Peel (County) Board of Education*, [1981] 2 SCR 21, *Bonnington Castings Ltd. v. Wardlaw*, [1956] 1 All ER 615 (HL), *McGhee v. National Coal Board*, supra. A contributing factor is material if it falls outside the de minimis range: *Bonnington Castings Ltd. v. Wardlaw*, supra; see also *R. v. Pinsky* (1988), 30 BCLR (2d) 114 (CA), affirmed [1989] 2 SCR 979.

In *Snell v. Farrell*, supra, this Court recently confirmed that the plaintiff must prove that the defendant's tortious conduct caused or contributed to the plaintiff's injury. The causation test is not to be applied too rigidly. Causation need not be determined by scientific precision; as Lord Salmon stated in *Alphacell Ltd. v. Woodward*, [1972] 2 All ER 475 at 490 (HL), and as was quoted by Sopinka J. at p. 328, it is "essentially a practical question of fact which can best be answered by ordinary common sense". Although the burden of proof remains with the plaintiff, in some circumstances an inference of causation may be drawn from the evidence without positive scientific proof.

It is not now necessary, nor has it ever been, for the plaintiff to establish that the defendant's negligence was the *sole cause* of the injury. There will frequently be a myriad of other background events that were necessary preconditions to the injury occurring. To borrow an example from Professor Fleming (*The Law of Torts* (8th ed. 1992) at p. 193), a "fire ignited in a wastepaper basket is ... caused not only by the dropping of a lighted match, but also by the presence of combustible material and oxygen, a failure of the cleaner to empty the basket and so forth". As long as a defendant is *part of the cause* of an injury, the defendant is liable, even though his act alone was not enough to create the injury. There is no basis for a reduction of liability because of the existence of other preconditions: defendants remain liable for all injuries caused or contributed to by their negligence.

This proposition has long been established in the

jurisprudence. Lord Reid stated in *McGhee v. National Coal Board*, supra, at 1010:

It has always been the law that a pursuer succeeds if he can show that fault of the defender caused or materially contributed to his injury. There may have been two separate causes but it is enough if one of the causes arose from fault of the defender. The pursuer does not have to prove that *this cause would of itself have been enough to cause him injury*.

The law does not excuse a defendant from liability merely because other causal factors for which he is not responsible also helped produce the harm: Fleming, supra, at p. 200. It is sufficient if the defendant's negligence was a cause of the harm: *Assiniboine South School Division No. 3 v. Greater Winnipeg Gas Co.*, [1971] 4 WWR 746 (Man. CA), at p. 753, affirmed [1973] 6 WWR 765, [1973] SCR vi. Ken Cooper-Stephenson, *Personal Injury Damages in Canada* (2nd ed. 1996), at p. 748.

The so-called "crumbling skull" rule simply recognizes that the pre-existing condition was inherent in the plaintiff's "original position". The defendant need not put the plaintiff in a position *better than his or her original position*. The defendant is liable for the injuries caused, even if they are extreme, but need not compensate the plaintiff for any debilitating effects of the pre-existing condition which the plaintiff would have experienced anyway. The defendant is liable for the additional damage but not the pre-existing damage: Cooper-Stephenson, supra, at pp. 779-780 and John Munkman, *Damages for Personal Injuries and Death* (9th ed. 1993), at pp. 39-40. Likewise, if there is a measurable risk that the pre-existing condition would have detrimentally affected the plaintiff in the future, regardless of the defendant's negligence, then this can be taken into account in reducing the overall award: *Graham v. Rourke*, supra; *Malec v. J.C. Hutton Proprietary Ltd.*, supra; Cooper-Stephenson, supra, at pp. 851-852. This is consistent with the general rule that the plaintiff must be returned to the position he would have been in, with all of its attendant risks and shortcomings, and not a better position.

The plaintiff may have been predisposed to develop Parkinson's disease for a variety of reasons including previous concussions or traumatic brain injuries (the true "thin skull scenario"). Accordingly, it is sufficient if the medical expert provides an opinion that the injuries sustained in the accident were a material contributing factor in the triggering of Parkinson's disease or parkinsonism even though the accident was not the sole contributing factor. The diagnosis then becomes posttraumatic parkinsonism.

C. Case Law

While there may have been a number of cases in Canada where trauma was alleged to be a factor in the onset of Parkinson's disease, there are only three cases that have proceeded to trial.

I. Trauma Accelerating Parkinson's Disease

In *Kamis v. Oaks*, 1988 CarswellAlta 25 (Alta. CA), a 58 year old plaintiff was stopped in his vehicle and was rear-ended causing his vehicle to rollover. He suffered abdominal, chest, hip and shoulder injuries. He was discharged from the hospital after five days. He returned 30 days later with some neurological symptoms including notably slowed speech and reduced motor abilities. A month later, he was walking slowly with the use of a cane. His neurological symptoms worsened and one year later he was diagnosed with the early onset of Parkinson's disease. Four neurologists testified at the trial. One of the plaintiff's experts offered the opinion that the onset of Parkinson's disease was attributable to the

accident. The two defence experts, Dr. West and Dr. Sucherowsky, were of the opinion that the Parkinson's disease was not caused by the injuries suffered in the accident. The condition was either there at the time in a subclinical phase or else it was accelerated as a result of the injuries suffered in the accident. The trial judge made the following findings:

On the basis of all of the medical evidence, I conclude then that the plaintiff's early neurological deficits result directly from the accident and that the Parkinson's disease superimposed on those early symptoms was aggravated and accelerated by the accident. I have no basis whatsoever on the evidence from which to determine the rate of acceleration of the advent of Parkinson's disease. It is impossible for me, therefore, to say that the plaintiff was incapacitated a year earlier or five years or ten years earlier, because the evidence I have is that Parkinson's disease is a very insidious disease and may take anywhere from four or five years up to fifteen years before its debilitating effects are fully felt.

The trial judge awarded the plaintiff \$210,854 including non-pecuniary damages of \$100,000 and discounted the future loss of earnings by 50 per cent on the basis that the debilitating effects of the Parkinson's disease would have rendered the plaintiff unable to work before the age of 70. The Court of Appeal found that there was evidence to support the finding that the onset of the Parkinson's disease had been accelerated by the accident, but reduced the award on the basis that damages should be limited to the period of acceleration which on the evidence was one or two years. The non-pecuniary damages were reduced to \$40,000 and the future loss of earnings claim was dismissed.

2. Trauma Aggravating Parkinson's Disease

In *McAfee v. Hartman*, [2000] BCJ No. 190 (BCSC) the plaintiff had suffered from tremors in her left arm and left leg which were attributed to Parkinson's disease. The plaintiff was involved in a motor vehicle accident and almost immediately noticed an increase in her tremors. The issue was whether the plaintiff's pre-existing Parkinson's disease was aggravated by the accident. Medical evidence from her treating neurologist suggested that the stress of the accident exacerbated the plaintiff's Parkinson's symptoms but did not accelerate the disease process. The trial judge accepted this evidence and found that the accident aggravated the symptoms for a period of two years and awarded non-pecuniary damages of \$30,000 and past income loss of \$36,561.

These two cases involved an acceleration or aggravation of Parkinson's disease by peripheral trauma. In order to establish a causal relationship between trauma and parkinsonism, it would appear that on the basis of the currently accepted medical diagnostic criteria, the trauma must cause damage to the brain.

3. Posttraumatic Parkinsonism: a Mild Traumatic Brain Injury Case

The recent case of *Heringa v. Mah* 2000 BCCA 490 involved a finding by a jury that the injuries sustained in an accident (a mild traumatic brain injury) caused Parkinson's disease or parkinsonism. The facts of the case are summarized by Rowles, J.A.:

[5] On January 12, 1995, the plaintiff was struck by a motor vehicle while crossing the street in a crosswalk. He was 38 years of age at the time and worked as a professional photographer. About three months after the accident, he developed early signs of Parkinson's disease. The plaintiff alleged that the accident caused various soft tissue injuries and a head injury which, in turn, caused his Parkinson's disease. Evidence was led at trial to show that he was a skilled photographer, and that had it not been for the accident, he would have had a lucrative career as a professional photographer.

[6] The position of the defendants was that the trauma and stress from the accident served only to accelerate the onset of the symptoms of Parkinson's disease which had been present but asymptomatic before the accident.

[7] There was a marked difference of opinion between the expert witnesses. The plaintiff's experts gave evidence that the trauma from the head injury triggered the onset of the disease. The experts called by the defendants opined that such a head injury could not have caused the disease. The defence experts were of the opinion that the accident, combined with other stressful events in the plaintiff's life, accelerated the onset of the symptoms of the disease by one or two years.

[8] The trial, which lasted four weeks, was heard by Justice Shaw and a jury. In their verdict rendered June 27, 2000, the jury assessed damages as follows:

Non-pecuniary loss	\$ 253,000.00
Past income loss	\$ 588,035.00
Future income loss	\$3,865,840.00
Cost of future care	\$1,292,512.00
Special damages	\$ 19,510.45
Total	\$6,018,897.45

In the accident, Heringa was struck on his left arm and leg by the van and propelled 12 feet through the air but did not hit his head. He sustained a brief period of posttraumatic amnesia. He did not lose consciousness but was slightly confused. At the scene of the accident he was "shaking like a leaf". Two days later his family doctor diagnosed a concussion. He was complaining of headaches, dizziness, nausea, forgetfulness, distractibility and difficulty with concentration. He was a professional photographer and could take hand held, clear photographs at a shutter speed of 1/15 of a second. When he returned to work following the accident, he noticed a subtle tremor in his left arm. He could no longer take hand held photographs and had to use a tripod. He was initially diagnosed with a traumatic brain injury and an "essential tremor", a benign condition characterized by a slight tremor of one or more limbs. Seven months after the accident the plaintiff was referred to a neurologist who specialized in Parkinson's disease. The diagnosis was posttraumatic parkinsonism triggered by the closed head injury suffered in the accident.

It is important to note in the *Heringa* case that the plaintiff had sustained two concussions or mild traumatic brain injuries approximately 20 years prior to the accident. He recovered from those injuries after a few months and was symptom free until he was struck by the van.

(a) Physiotherapy Records

The evidence of a very experienced physiotherapist who had been trained as an orthopaedic nurse was of particular significance in this case. She had examined and treated the plaintiff on 78 occasions beginning the month following the accident. The following notations from her clinical records document the symptoms of both the traumatic brain injury and the parkinsonism:

Feb. 20, 1995

Numbness L thumb – could not feel over the thumb
Reduced strength upper and lower extremities
Hyperexcitability and clonus in quads
Involuntary contraction/relaxation muscles
Assessing motor integrity – unable to shrug L shoulder
Unilateral weakness very unusual

Feb 22, 1995

Absence sensation outer side L thumb
Posture – incredibly rigid
Neurological examination – unable to lift L shoulder
Memory difficulty

March 9, 1995 [symptoms noted]

Headaches	Dizziness
Poor memory	Fatigue
Irritability	Balance
Sensitivity to noise	Squint
Problem solving	Depression
Emotional instability	Sleeplessness
Loss of strength	Diminished interest

March 16/95 L thumb absence of sensation, L shoulder girdle, quads, hamstrings, extreme irritability

Mar 28/95 L thumb numbness, L palm

April 4/95 weakness L arm

April 10/95 fine tremor hamstrings, L forearm

April 20/95 STM [short term memory] poor, emotions labile, L side different, L arm swing

May 8/95 decreased sensation L thumb, now coin size area numbness L palm, decreased L arm swing

May 19/95 tremor L arm, L leg after exertion, reduced L arm swing, not balanced L to R, STM poor, unable to retain info

(b) Defence Experts

The defence called three neurologists (all specialists in Parkinson's disease) and a neuropathologist who all agreed that the mild head injury sustained by the plaintiff could not have caused or triggered the parkinsonism. Several of the defence neurologists did agree that parkinsonism could be caused by a single severe traumatic brain injury or by multiple mild traumatic brain injuries. All the defence experts were of the opinion that the plaintiff must have been in a sub clinical phase of Idiopathic Parkinson's disease prior to the accident and that the trauma or emotional stress of the accident accelerated the onset of the symptoms by one or two years.

In the *Heringa* case there was substantial cross-examination of the defence experts on the medical literature. The jury was able to examine all of the evidence in the case and decide which opinions they were going to accept. Based on the nature of their verdict it seems clear the jury members rejected the opinion of the defence experts and accepted the opinion of the plaintiff's neurologist that the plaintiff was not in a sub clinical phase of Parkinson's disease prior to the accident and that the parkinsonism was triggered by the traumatic brain injury suffered in the accident.

It was an important factor in this case that the plaintiff was young (38) and that his occupation prior to the accident required the use of fine motor skills. It would be difficult to design a more sensitive test for the existence of a tremor than the ability to take crystal clear photographs with a handheld camera at a shutter speed of 1/15th of a second. A further factor of some significance was that the plaintiff had sustained two prior concussions or mild traumatic brain injuries, which may have made him more vulnerable to developing parkinsonism following the accident.

VII. SUMMARY

The majority of cases of posttraumatic parkinsonism will likely involve motor vehicle accidents in which the plaintiff has sustained a traumatic brain injury. The *Heringa* case demonstrates the necessity to document the emergence of the physical, cognitive, and emotional symptoms of both traumatic brain injury and Parkinson's following the trauma. This will require a careful review of all the records including the ambulance crew report, hospital records, and clinical records of all medical practitioners. Success may depend largely on the observational insights of the treating professionals, including physical therapists. These records will provide the necessary foundation for the medical expert to correlate

the relationship of traumatic brain injury to the onset of parkinsonian symptoms to support a diagnosis of posttraumatic parkinsonism.

1. The author gratefully acknowledges the input of his colleague, David N. Osborne, to this paper. This paper was originally prepared for the CLE seminar, Personal Injury: Medical Issues held September 2000 in Vancouver BC.
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