
CANADA / JUDGMENT

Taylor v. Liong

2008 BCSC 242

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Supreme Court of British Columbia

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2008 BCSC 242 Taylor v. Liong

IN THE SUPREME COURT OF BRITISH COLUMBIA

Citation:

Taylor v. Liong,

2008 BCSC 242

Date: 20080228

Docket: B992886

Registry: Vancouver

Between:

Harris Clark Anais Taylor

Plaintiff

And

Linda Kua Liong and Timothy Garth Liong

Defendants

Corrected Judgment: The front page of this judgment was amended on March 5, 2010 to correct the name of counsel.

Before: The Honourable Mr. Justice Cullen

Reasons for Judgment

Counsel for the Plaintiff:

R. Nairne

Counsel for the Defendants:

R. Hartshorne

K. Mehl

Date and Place of Hearing:

October 16, 2007

Vancouver, B.C.

INTRODUCTION

- [1] This is an action for damages for injuries arising out of a motor vehicle accident which occurred on May 22, 1998. At issue on this application for judgment brought by the defendants under Rule 18A of the Rules of Court, B.C. Reg. 221/90 is whether the evidence proves on a balance of probabilities that trauma can make multiple sclerosis ("MS") symptomatic or otherwise alter the natural course of the disease. I ordered this issue of causation to be severed and tried separately on application of the defendants brought on September 7, 2007. This followed a voir dire held to determine the admissibility of expert evidence directed at the issue of whether there is a causal connection between trauma, including mild head trauma or whiplash injury, and the onset of symptomatic MS.
- [2] In the result, I held that the evidence of two of the three expert witnesses called by the plaintiff was admissible on the issue and that the evidence of all three of the expert witnesses called by the defence was admissible.
- [3] My reasons for that ruling are reported in *Taylor v. Liong*, 2007 BCSC 231, 70 B.C.L.R. (4th) 284 [*Taylor v. Liong* (2007)] and provide an essential context for my consideration and determination of the issue on this application.
- [4] The plaintiff resists the defendants' application for judgment, both on the basis that this issue is not suitable for determination by Rule 18A, and on the substantial issue of whether the evidence before the Court proves on the balance of probabilities that trauma (such as mild head trauma or whiplash) can or is capable of triggering or exacerbating MS in some circumstances.
- [5] For the reasons that follow, I conclude that this is an appropriate application to be heard under Rule 18A.
1. The Appropriateness of the Application
- [6] The relevant provisions of Rule 18A read as follows:
- (1) A party may apply to the court for judgment, either on an issue or generally, in any of the following:
- (a) an action in which a defence has been filed;

- (8) On an application heard before or at the same time as the hearing of an application under subrule (1), the court may
 - (b) dismiss the application under subrule (1) on the ground that
 - (i) the issues raised by the application under subrule (1) are not suitable for disposition under this rule, or
 - (ii) the application under subrule (1) will not assist the efficient resolution of the proceeding.
 - (11) On the hearing of an application under subrule (1), the court may
 - (a) grant judgment in favour of any party, either on an issue or generally, unless
 - (i) the court is unable, on the whole of the evidence before the court on the application, to find the facts necessary to decide the issues of fact or law, or
 - (ii) the court is of the opinion that it would be unjust to decide the issues on the application
 - [7] The evidence before the Court on this application consists of the evidence of Drs. Poser and Kastrukoff for the plaintiff, and Drs. Sibley, Ebers and Goodin for the defendants, together with approximately 100 exhibits consisting of the witnesses' expert reports and various materials and articles referred to in the list of exhibits appended to the affidavit of Kristen Mehl sworn August 22, 2007, and filed in support of the defendants' application of September 7, 2007.
 - [8] Included as evidence on the voir dire was an agreed statement of facts which reads as follows:
 - 1. On May 22, 1998, Harris Taylor was driving a 1992 Eagle Vista hatchback westbound on 10th Avenue, quite slowly preparing to make a right hand turn into the Sky Train Parking lot (XFD October 29, 2003 QA 686).
 - 2. The motor vehicle collision occurred when the Timothy Liong [sic], driving a 1986 626 Mazda, turned left out of the parking lot on the south side of 10th Avenue intending to travel in the same direction as Harris Taylor when he collided with the front part of the vehicle she was driving.
 - 3. Both vehicles were driven from the scene and neither police nor ambulance attended.
- Copies of the Insurance Corporation of British [sic] CL14 Repair estimates for both the Paula McDonald vehicle and the Linda Liong vehicle are attached together with a photograph of each vehicle taken at ICBC Claim Centre.
- 4. According to Harris Taylor's statement dated May 27, 1998, she:
 - strained the muscles in her right hand and up her arm
 - she had a twinge in her mid lower back just under her rib cage

- she had a bit of agitation to the top of her right shoulder
- she had a history of lower back problems.

Her evidence at her examination for discovery conducted on October 29, 2004 was:

736 Q Did you hit your head on anything in the vehicle?

A No

737 Q And was your head facing forward -

A Yes

738 Q -straight? Okay. And did you stop your vehicle as soon as the collision had occurred?

A Yes

5. At the time Harris Taylor made her statement, she had not yet been to the doctor but said she was going to make an appointment by weeks end. (May 27, 1998 Statement of Harris Taylor).
6. Harris Taylor's first recorded medical or para medical treatment was with Dr. Sheikh, her Chiropractor, on May 27, 1998. (MSP print out and Clinical Records).
7. Harris Taylor's first recorded attendance with a medical physician was on August 31, 1998, with her General Practitioner, Dr. Joan Robillard. (MSP Print Out and Clinical Records).
8. On September 8, 1998 Harris Taylor was diagnosed with Multiple Sclerosis at Vancouver General Hospital. (VGH Clinical Records).
9. Harris Taylor's MRI of the head taken at Vancouver General Hospital on September 10, 1998 confirmed the diagnosis of Multiple Sclerosis and found a "high burden of disease". (MRI Scan September 10, 1998).
- [9] Neither party sought to adduce any further evidence on the Rule 18A application which was heard on October 16, 2007. There was evidence that the "high burden of disease" supported the conclusion that the plaintiff had the disease for some years before the accident.
- [10] The position of the plaintiff respecting the appropriateness of this application is summarized in her counsel's written submissions in paras. 7 and 8 as follows:

The Court is asked to decide if trauma can trigger or exacerbate MS under any circumstances in isolation, divorced from the unique circumstances of the Plaintiff. The question posed is a scientific rather than legal question, which is inappropriate for the Court to answer.

The only question that the Court should address is whether the Plaintiff in this case developed MS following trauma. To do so requires examination of the Plaintiff's unique circumstances, including her medical history. Evidence of the Plaintiff, her family doctor, and the clinical records is critical to answering this question. Once all evidence is heard, the Court can weigh the likelihood the trauma played a role in the onset of the Plaintiff's MS against the Defendants' evidence that such a result is unlikely.

[11] In resisting this application, the plaintiff relied on the factors the court should consider in deciding whether to give judgment under Rule 18A set forth in *Inspiration Management Ltd. v. McDermid St. Lawrence Ltd.* (1989), 36 B.C.L.R. (2d) 202 (C.A.) and cited in *Canadian Imperial Bank of Commerce v. Charbonnages de France International S.A.* (1994), 95 B.C.L.R. (2d) 104 (C.A.) at para. 101 as follows:

1. the amount involved;
2. the complexity of the matter in issue;
3. the urgency of the matter;
4. the likelihood of prejudice arising from delay;
5. the cost of proceeding to a conventional trial in relation to the amount involved;
6. the course of the proceedings;
8. any other matters which arise for consideration.

[12] The plaintiff argued the amount involved in this matter is potentially significant, amounting to \$2.5 million to \$3 million, that the matter is highly complex, and that given the passage of time since the accident to the present trial date of May 26, 2008, there is no urgency requiring this application. The plaintiff conceded the cost of proceeding to a full trial will be considerable, but points out that both parties have already expended considerable resources on this matter.

[13] The plaintiff submitted she should be entitled to a full trial on the merits, having already gone through a voir dire to test the admissibility of the evidence at issue. Further, the plaintiff contended at para. 19 of her counsel's written submissions that the evident purpose of the defendants to treat this law suit as a "test case" to "deny all plaintiffs who develop MS subsequent to a motor vehicle accident any remedy as a matter of law", is at odds with the court's function, which is to "settle disputes between parties on a case by case basis."

[14] Counsel for the plaintiff also relied on a judgment of Gerow J. in *Dahl v. Royal Bank* 2005 BCSC 1263, 46 B.C.L.R. (4th) 342, aff'd 2006 BCCA 369, 56 B.C.L.R. (4th) 58, in which she collated additional factors to be taken into account in determining the suitability of proceeding by Rule 18A from other decisions rendered since *Inspiration Management*, supra. Those factors include the following, set out at para. 12:

- is the litigation extensive and will the summary trial take considerable time;
- is credibility a crucial factor - and have the deponents of the conflicting affidavits been cross examined;
- will the summary trial involve a substantial risk of wasting time and effort, and producing unnecessary complexity; and
- does the application result in litigating in slices.

[15] Counsel for the plaintiff contended that those factors were "particularly important" in the case at bar submitting, in part, at para. 23 of his written argument as follows:

The litigation involved in this matter is extensive. Most of the evidence obtained at the voir dire must be reconsidered. Most importantly, the issue for consideration ought not to be assessed in isolation. As will be addressed more fully below, the determination of the theoretical relationship between MS and trauma is a question for science, not the Court, which should restrict itself to whether the facts show trauma was a material contributing factor to the emergence of MS in this Plaintiff.

[16] The plaintiff relied on *Parsons v. Finch*, 2003 BCCA 409, 16 B.C.L.R. (4th) 86 ["Parsons"] and *Prevost (Committee of) v. Vetter*, 2002 BCCA 202, 100 B.C.L.R. (3d) 44 ["Prevost"] which held, in effect, that where contrary views of evidence which overlaps separate issues - in those cases the duty and standard of care and causation - may be a risk, those issues ought not to be separately determined and hence a Rule 18A application would not be suitable to resolve one of them.

[17] The plaintiff submits at para. 24 of her counsel's written submissions that the issue of the suitability of Rule 18A determination is similar in the present case as follows:

By isolating this issue, the Defendants pose a scientific rather than legal question. It need not, and indeed should not be answered by the Court. The question of whether trauma triggered or exacerbated the Plaintiff's MS overlaps with and is inextricably interwoven with the issue of whether there is a general relationship between MS and trauma.

[18] The plaintiff submits, having admitted the impugned expert evidence, the role of the Court is to consider if the plaintiff has proved, on a balance of probabilities, that in her case, trauma triggered her MS. The plaintiff submitted this issue requires consideration of all of the evidence including that of the plaintiff, her family physician, and the doctors who treated her MS. The plaintiff submits at para. 27 of her counsel's written argument that it would be "unjust to make any conclusions based on general theories alone as it requires the Plaintiff to prove a general scientific theory."

[19] The plaintiff further contended that this application will only shorten the trial if the defendants succeed. If the plaintiff succeeds, the trial will not be substantially shortened and in those circumstances this proceeding would be a waste of the court's resources.

- [20] The plaintiff relied on *North Vancouver (District) v. Lunde* (1998), 60 B.C.L.R. (3d) 201 (C.A.) at para. 33, in submitting that the onus is on the defendants to demonstrate "that the administration of justice, as it effects not just the parties to the motion, but also the orderly use of court time, will be enhanced by dealing with the issue as a separate issue."
- [21] The plaintiff further referred to the caution of Southin J.A. in *Bacchus Agents (1981) Ltd. v. Philippe Dandurand Wines Ltd.*, 2002 BCCA 138, 164 B.C.A.C. 300 ["Bacchus Agents"] in which she observed at para. 7 that 'litigating in slices' may become a hindrance to the 'just, speedy and inexpensive determination' of the dispute 'on its merits'.
- [22] As I see it, I am not being asked through this application to determine a scientific rather than a legal issue. The question before me is whether the evidence adduced proves on the balance of probabilities that trauma, including mild head trauma or whiplash, is capable of triggering or exacerbating MS symptoms. That is a question that is necessary to answer to reach a meaningful conclusion in this law suit. In other words, it is a legal question, the answer to which is neither designed nor conducive to resolving any lingering scientific debate on the issue within the medical or scientific world. As I noted in ruling on the admissibility of the parties' expert evidence, Lord Rodger, the Lord President, similarly characterized the same issue before the court in *Dingley v. The Chief Constable, Strathclyde Police*, [1998] Sess. Cas. (C.S.I.H.) 548 (Scot.), ["Dingley"], *aff'd* [2000] Sess. Cas. (H.L.) 77, when he said at p. 551 as follows:

At present, despite many years of research, the cause of the onset of symptomatic MS has not been scientifically established. That being so, to succeed in his claim for damages against the defender, the pursuer requires to establish two things on the balance of probabilities: first, that the onset of symptomatic MS can be triggered by an injury of the type which the pursuer suffered in the accident; secondly, that not only could the pursuer's injury have triggered the onset of his condition, but it actually did so. So far as the first of these matters is concerned, I agree with the Lord Ordinary [in the court below appealed from] when he says that he was solely concerned with the evidence which the parties led before him: his task was to decide the case on that basis, rather than to settle the scientific debate which divides the medical world.

- [23] I thus do not accept the plaintiff's underlying precept that the Court in addressing itself to this general issue is undertaking anything other than a determination of whether an essential element of the tort claim - causation - is made out on the basis of the cogent evidence.
- [24] Moreover, while I appreciate that the ultimate issue of causation is whether the plaintiff's MS symptoms were triggered or exacerbated by the accident, the general issue - whether trauma can trigger MS symptoms - dominates the ultimate or specific issue to such an extent that it must be proved on a balance of probabilities to engage even a remote possibility of a connection between the accident and the plaintiff's ensuing symptomatic MS. In other words, if the Court does not accept on the balance of probabilities that MS can be triggered by trauma, how can the plaintiff prove on a balance of probabilities that her MS symptoms

were exacerbated by the trauma of the accident? If the plaintiff's specific circumstances, condition, and course of treatment constituted meaningful evidence of a causal connection between trauma and the onset of MS, those issues would have been cogent considerations on the issue of the reliability and hence admissibility of the impugned expert evidence. They were not.

- [25] Hence, it would simply not be logical to conclude that the ultimate issue of the causation of the plaintiff's MS onset could be proved on a balance of probabilities while concluding the general proposition necessary to establish could not be proved.
- [26] Such an illogical result would be amplified in the present case in which, on the plaintiff's own theory, the MS symptoms induced by trauma occur in something less than 5% of all cases.
- [27] I therefore conclude that what is at issue is not a general scientific theory, but rather an essential element of the plaintiff's case insofar as she asserts a causal connection between the accident and the onset or exacerbation of her MS symptoms.
- [28] As to the other issues raised by the plaintiff in opposing resolution of this matter by Rule 18A, I similarly conclude that they are not dispositive of the issue. It is common ground that the trial of this matter in addition to the voir dire will consume six weeks. It will be expensive and will consume considerable court time. This application, despite the complexity of the underlying issue and the comprehensiveness of the evidence in the voir dire, took only one day to argue. Insofar as there are issues of credibility or reliability which condition the issue, I have had the benefit of full and skilled cross-examination of well qualified experts on both sides. As indicated, no further evidence was advanced on this application and no evidence, not called on the voir dire, could be considered germane to the issue on this application. While there may not be a significant benefit in terms of the saving of time and resources if I were to dismiss this application, it is common ground that there would be a dramatic narrowing of the issues if I were to determine this issue in favour of the defendants.
- [29] In my view, on balance, these factors weigh in favour of this application as the cost in terms of judicial resources and to the parties is not great, while the possible reduction in cost of this matter particularly if it is determined favourably to the defendants is great.
- [30] As for the plaintiff's concern that this application is being pursued, at the expense of the plaintiff having her day in court, to establish a precedent for other outstanding cases, in my view that is not, in the circumstances, a significant factor. In the first place, whatever precedential value my ruling will have relates to the evidence and the issue before me. Secondly, as I have observed, there is no indication that the evidence of the plaintiff's specific circumstances, condition or course of treatment have probative value on the issue before me. Thus, if some precedential value does emerge from the resolution of this issue, it will not be in the absence of evidence said to be probative, and it will depend on the evidence which the parties elected to put before me.

- [31] Finally, on the issue of litigating in slices, where there is a risk of making findings of fact in relation to issues under consideration from evidence that overlaps with other issues not being determined, I similarly see no bar to proceeding in the present case.
- [32] The issues identified by the plaintiff as overlapping in the present case, as I attempted to explain earlier in these reasons, are not susceptible to the risk identified in *Parsons*, *supra*, *Prevost*, *supra*, or *Bacchus Agents*, *supra*, because they must logically be decided in order, and involve separate evidence. It would not be logically possible to find that the plaintiff's trauma exacerbated her MS symptoms without a prior determination that trauma can exacerbate MS symptoms. It does not follow from a finding that trauma can exacerbate MS symptoms that the plaintiff's trauma caused her MS symptoms. Thus, unlike the circumstances in *Prevost*, *supra*, where the overlapping evidence on the issues of the duty and standard of care and causation required them to be determined together to ensure a coherent and consistent result was achieved, the present case can be determined in logical steps without impairing or impeding a coherent and just final result.
- [33] I therefore decline to dismiss the defendants' application on the basis that it is inappropriate for a determination under Rule 18A.
2. Does the Evidence Prove on a Balance of Probabilities that there is a Causal Relationship between Trauma, including Mild Head Trauma or Whiplash, and the Onset or Exacerbation of MS Symptoms.
- [34] Having found that this is an appropriate application to be heard under Rule 18A, I will now consider the issue of whether the evidence proves on a balance of probabilities that trauma can or is capable of triggering or exacerbating MS in some circumstances.
- [35] In *Dingley*, *supra*, Lord Rodger, after extensively reviewing the evidence before the Lord Ordinary in the court appealed from, much of which parallels the evidence led in this case on the *voir dire*, concluded as follows at pp. 601-02:

I have weighed all the various factors in favour of the pursuer's view and in particular the paper by Oppenheimer, the observations of Gonsette and his colleagues and the theory of Dr. Poser. In the end, however, I do not find it proved, on a balance of probabilities, that trauma in general, or whiplash injury in particular, can trigger the onset of symptomatic MS. In saying this I do not need to go so far as actually to accept the conclusions reached by the defender's witnesses in their evidence. It is enough that, when I examine and weigh the evidence upon which the pursuer relies along with the evidence for the defender, I am left with sufficient doubt about the pursuer's case that I am not satisfied that he has proved it on the balance of probabilities.

Since I am not satisfied that whiplash injury can ever cause the onset of symptomatic MS, I do not need to go on to answer the second question which relates to the particular facts of the pursuer's accident and injury. I merely observe that I have difficulty in seeing how, even supposing that it were possible for trauma to trigger the onset of MS, the evidence establishes that this was a case where the trauma was a trigger rather than a case where the existence of a trauma preceding the onset was coincidental.

[36] The decision of Lord Rodger was upheld on appeal to the House of Lords in: *Dingley v. The Chief Constable, Strathclyde Police*, [2000] Ses. Cas, (H.L.) 77. In that decision, Lord Hope of Craighead concluded at p. 93:

In the end the process of reasoning which led the Lord President to this conclusion seems to me to be quite straightforward, and in my opinion it is unassailable. There is a body of clinical evidence that shows that there are many cases of symptomatic MS which cannot be related to trauma. In a small number of cases the onset of symptoms is preceded by trauma. The epidemiological studies do not support the appellant's case. If anything they tend to support the arguments to the contrary. But for present purposes the important point is that they show that the appellant's case depends on there being an acceptable theory to explain what it is that overcomes the BBB [blood brain barrier] and permits the development of symptomatic MS. A satisfactory explanation would go a long way to supporting the appellant's case. But the evidence, which reaches its highest point with the study by Oppenheimer, does not go far enough to provide that explanation. So the appellant's case fails on a balance of probabilities.

[37] In my reasons for ruling on the admissibility of the expert evidence proffered by the parties I noted as follows at para. 56:

The evidence considered by the court in *Dingley* was adduced before the trial court in 1995. That was before the "debate that divides the medical world" was conditioned by the AAN Article, the published position of the National MS Society in 2003, the approach to the issue taken in McAlpine's 4th ed. published in 2005, and the similar approach taken in Harrison's 16th ed., also published in 2005. Moreover, the court in *Dingley* did not have before it the results of the Goldacre Study published in 2006, or studies published in 1998 and 2000 said to challenge the significance of the role of the breakdown of the blood brain barrier in the pathogenesis of MS symptoms.

[38] As will become apparent from a review of *Taylor v. Liong* (2007), the AAN Article - a special publication of a sub-committee of the American Academy of Neurology - the publication of the National MS Society, the content of McAlpine's 4th ed. and the content of Harrison's 16th ed. all represent an erosion of support for the theory of a causal link between trauma and MS symptom onset within the medical world.

- [39] Additionally, as is apparent from those reasons, the results of the Goldacre Study published in 2006 and the "studies published in 1998 and 2000" - the Filippi Study and the Werring Study respectively - provided additional information adding to the weight of evidence against the prospect of a causal link between trauma and the onset of MS symptoms.
- [40] It is worthy of consideration that, although the Dingley decision is not binding on me, the reasoning of Lord Rodger, so far as it addresses the same or similar evidence as confronts me, is compelling by its lucid, careful and thorough analysis and conclusion. The fact that since that decision was pronounced (but not because of it) the relevant scientific community can be seen as coalescing around the scientific proposition that no causal relationship exists between trauma and MS symptoms in light of additional studies and discoveries, can only lend weight to the guidance implicit in Lord Rodger's extensive judgment.
- [41] In admitting the evidence of Dr. Poser and Dr. Kastrukoff, I analyzed the evidence against the legal test for admissibility which included an assessment of its threshold reliability. In doing so, I attempted to summarize the evidence and the parties' position on that evidence without, of course, making any determination of its ultimate reliability.
- [42] In coming to my conclusion on admissibility, I held as follows at paras. 196-97 respecting the distinction between threshold and ultimate reliability:

I am not satisfied that what I am confronted with here is a case of threshold unreliability. This is not a case where the plaintiff's theory has never been published in peer review publications, nor subjected to peer criticism or testing. It is not a green, untried, and untested theory, although it is a theory that has lost adherents over time, and with the accumulation of new evidence and new studies, it has attracted opposition from a substantial majority in the relevant scientific community. I accept that that opposition is a telling factor against reliance on this theory, certainly in the medical or scientific field, but also in the courtroom. However, I do not find in that substantial opposition, a basis for concluding that the theory or the evidence relied on in its support is in a legal sense, incapable of being considered by a trier of fact.

The evidence amassed in opposition to the plaintiff's theory and her evidence may attenuate her case. Dr. Poser's credibility and his asserted misreliance on studies may compromise it. The logic flowing from a theory espousing a low risk of MS from the cause being asserted as compared to other causes, and the high likelihood of coincidence of trauma and exacerbation, may undermine the case. But, there remains a theoretical and evidentiary base for its consideration by a trier of fact. The issues raised by the defendant's objections and evidence ultimately require consideration, understanding, comparison, analysis, and conclusion concerning contradictory evidence and competing opinions. That is the subject matter of ultimate, not threshold, reliability. Accordingly, I would admit the evidence of the plaintiff's experts' theory and the evidence in support of it, save for Dr. Lehrer's theory and the evidence supporting that.

[43] It was my conclusion that an important logical step in determining the admissibility of the evidence was whether it had any "biological plausibility". I held as follows at paras. 191 and 193:

The plausibility of the mechanism advanced by Dr. Poser is of considerable significance in determining the admissibility of his theory and the evidence in support of it. If the theory remains plausible, it would be difficult to rule it or the evidence indirectly supporting it unworthy of consideration by a finder of fact. If the theory is implausible, then it renders evidence said to be supportive of it without coherence.

Of course the presence of a plausible theory for a cause and effect relationship does not establish such a relationship any more than the presence of a plausible method for building a house establishes the existence of the house. Some evidence must exist that is capable of supporting the existence of the relationship above and beyond its mere plausibility before consideration can be given to the process of determining that ultimate question.

(a) Biological Plausibility

[44] The issue of the biological plausibility requires consideration of the evidence of what is generally known about MS and what is unknown. In paras. 8 - 27 of my reasons on the admissibility hearing, I provided an overview of what the evidence established about MS as follows:

Axons are the neurons or nerve fibres in the central nervous system ("CNS") which extended from the nerve cell and conduct messages throughout the CNS and to the peripheral nervous system by electrical impulses. Myelin is an electrically insulating sheath that surrounds axons. There are spaces along the axons not covered by myelin, and these are called nodes of ranvier. What is known about MS is that it is a disease that attacks and destroys Myelin. Under normal conditions, the electrical impulses comprising the messages travel at speeds of approximately 100 metres per second by jumping along the axon from one node of ranvier to the next. When the myelin sheath is destroyed by the disease, the electrical impulses are slowed by being forced to travel along the axon and there can be a corresponding impairment of the physical abilities of the individual experiencing the condition. The process of myelin destruction is called demyelination. The process of demyelination leaves plaques or lesions along the axon.

There are portions of the CNS that are largely composed of bundles of myelinated axons which are comprised of almost 80% lipids (fats) and are known as the white matter of the brain. These portions of the brain are found in the inner layer of the cortex, the optic nerves, the central and lower areas of the brain (notably the brain stem), and surrounding the central shaft of grey matter in the spinal cord. About 95% of all MS lesions occur in the white matter.

The portions of the brain known as the grey matter consist of nerve cell bodies and astroglia consisting of about 80% water.

Multiple sclerosis is believed to be an autoimmune disease. There are various autoimmune diseases. An autoimmune disease refers to a body's immune system failing to recognize its own constituents and attacking them as they would attack a foreign invader. In rheumatoid arthritis for example, which is an autoimmune disease, the immune system will attack the body's joint's causing pain and inflammation.

The immune system is a separate system within the body which has evolved to identify and attack foreign invaders such as viruses which might otherwise cause illness. The immune system operates at a cellular and molecular level and involves various constituent elements with various functions. Each cell in the body has a marker on its surface which identifies the cell as belonging in the body. The markers are molecules called major histocompatibility complex (MHC). In blood cells, human leukocyte antigens (HLA) help identify cells as belonging to the self or as foreign.

In the event that foreign invaders enter the body, the immune system reacts by activating certain cells including B Cells, T Cells, and macrophages which respond to the threat represented by the foreign bodies.

B Cells are a type of white blood cell originating in bone marrow. B Cells are turned into plasma cells which multiply and form a cloned army of cells which produce proteins called antibodies. The antibodies respond to antigens which are molecules identifying the foreign invaders as appropriate targets for the antibodies. The antibodies then bind to the antigen and, working with blood proteins called the complement system, destroy the antigen or organism.

T Cells are also a type of white blood cell which are modified when presented with a specific antigen (by other, presenter cells) to produce a cloned army of cells which attack the offending substances by either secreting cytokines which tell other cells to attack the offending substances or by attacking directly themselves. Cytokines are small protein molecules released by cells to communicate with and direct the activity of other cells in the vicinity. They help the immune system to function by activating cells and directing the processes of inflammation, tissue breakdown and cell death. A subset of cytokines called interleukins or lymphokines act as messengers between cells. They are largely secreted and received by cells called leukocytes to stimulate a variety of activities depending on what is required by the context.

Macrophages are versatile. They can engulf (eat) foreign substances, dead cells, or other debris, and can also act as antigen presenters to B Cells and T Cells to engage their immune response.

An autoimmune reaction refers to some abnormality in the process by which the body's immune system becomes activated, and involves immune cells harming the body's own central nervous system.

It is generally accepted that in North America, approximately one out of one thousand people are diagnosed with multiple sclerosis and a further one out of one thousand people are undiagnosed despite having the condition. Women were historically at risk at a rate of 2 to 1, compared to men. Surprisingly and somewhat alarmingly, that ratio has changed dramatically (by an increase in incidence among women, rather than a decrease among men) in the past 50 years. This strongly suggests environmental factors in the initiation of the disease.

Magnetic resonance imaging ("MRI") is a non-invasive method used to render images of pathological or other physiological alterations of living tissues. MRI scans can be used to detect lesions in the myelin that covers axons. Gadolinium compounds can be injected into the body before scanning to enhance the MRI images.

Medical practitioners and scientists draw a distinction between biological symptoms (lesions that can be seen on MRI scans but cause no physical consequence) and clinical symptoms (in which the development of lesions manifest ascertainable physical consequences). Scientists estimate that for every one clinical symptom, there are five biological symptoms.

In one of his medical legal reports dated April 9, 2001, Dr. Sibley explained the mechanics of the disease thus:

MS is characterized by the appearance of patches of inflammation associated with demyelination (loss of fatty insulation of nerve fibres) in the white matter of the brain, brain stem, optic nerve and spinal cord. These patches, also called plaques or simply lesions, often occur in crops and they cause the common symptoms of the disease (visual loss, double vision, numbness and/or weakness of the limbs). They have become much easier to detect and monitor since the advent of the magnetic resonance imaging (MRI) scan in about 1985.

Serial MRI studies during the early years of the disease indicate that the average patient gets two to six new patches of MS in the brain each year, but clinical symptoms are much less common.

There is believed to be a strong genetic influence on the development of MS as studies have shown a relatively high correlation among family members grading upwards from half siblings to identical twins.

Environmental factors are also believed to be involved but those factors are as yet largely unidentified by studies.

As to the triggers of the disease, epidemiological studies implicate viral infections as potential triggers and it is presently generally accepted that approximately 25% of multiple sclerosis onset and exacerbation is triggered by viral infection.

There is no general acceptance of what other triggers may be responsible for the remaining 75% of MS onset or exacerbation. Interestingly, it appears that vaccinations do not precipitate MS attacks despite introducing antigens into the body which provoke an immune response. On the current state of knowledge it appears that vaccinations have a salutary effect on MS exacerbation.

Biological symptoms (the lesions that form in the myelin that covers the axons) are believed to start developing between the ages of about 5 and 15 in genetically susceptible people. The condition may have no consequences unless an onset is precipitated by some environmental factor. There is evidence that an alteration or disturbance of the blood brain barrier is implicated in the context of the formation of lesions in the course of the disease, although there is presently controversy whether the alteration or disturbance is a cause or effect of the lesion formation process.

The blood brain barrier ("BBB") refers to the protection afforded the brain and spinal cord (the CNS) by endothelial cells that are tightly bound together in what are referred to as tight junctions. The BBB prevents the easy passage of certain substances and cells into the CNS unless they are transported across by specific transport systems. The CNS endothelial cells are distinctive from other cells throughout the body which are not tightly joined and allow easy passage of substances or white blood cells to the surrounding tissues. Not all areas of the brain are covered by the BBB. The area postrema which is a sensory area for toxins located on the brain stem is not subject to the BBB, for example.

[45] Following that, I set out, in paras. 28 - 50, a summary of the evolution of the theory of a causal relationship between trauma and MS as follows:

The idea that trauma could trigger the onset or exacerbation of MS symptoms is not a novel theory in the sense of being new.

In McAlpine, D., "The Problem of Disseminated Sclerosis" (1946) *Brain* 69: 233 - 250 at 241, Dr. McAlpine, one of the then leading experts on MS, noted:

A number of observers, notably Allison (1931) and Von Hoesslin (1934) have made surveys in this disease with the purpose of discovering any facts which might throw light on its causation. The results of such surveys, in the main, confirm the occasional role of trauma, pregnancy and other factors precipitating the disease.

He went on to assert at p. 245:

After excluding those cases in which the disease itself was responsible for the alleged trauma, there remain a few cases of disseminated sclerosis in which there can be little doubt of the close relationship between peripheral trauma and the appearance of signs.

In McAlpine, D., and Compston, N., "Some Aspects of the Natural History of Disseminated Sclerosis" (1952) *Quarterly Journal of Medicine, New Series* XXI. No. 82, Dr. McAlpine and Dr. Nigel Compston, another leading expert in the field, wrote at p. 152 as follows:

In 31 patients, out of a total of 146 who suffered injury more than 3 months before the onset of their disease, the site of their initial lesion was related to the site of the injury (21.2%). Even allowing for the greater likelihood that injury, especially when apparently related to the first symptom, will be recalled in patients suffering from disseminated sclerosis, it seems probable that injury does sometimes precipitate the onset of the disease, and may determine the site of the initial lesion.

In Brain, R., and Wilkinson, M., "The Association of Cervical Spondylosis and Disseminated Sclerosis" (1957) *Brain* 80: 456 - 478 [the "Brain and Wilkinson Study"], the authors noted at p. 477:

In five of our series of cases trauma in the neck appears to have either precipitated the first symptoms of, or substantially exacerbated, the disseminated sclerosis and the co-existence of the two disorders has tended to bring about a deterioration more rapid than would have been expected, had either been present alone.

In Oppenheimer, D.R., "The Cervical Cord in Multiple Sclerosis" (1978) *Neuropathology and Applied Neurobiology* 4: 151 - 162 [the "1978 Oppenheimer Study"], the author, D.R. Oppenheimer of the Department of Neuropathology, Radcliffe Infirmary, Oxford, found a greater proportion of lesions in the cervical spine in 18 cases, distributed in such a way and forming a pattern of a sort, which he advanced as supporting the theory that they were caused by mechanical stresses.

In a leading text, Matthews, W.B. et al., *McAlpine's Multiple Sclerosis*, 2nd ed. (New York: Churchill, 1992), the state of acceptance of the association between trauma and exacerbation of the symptoms was summarized as follows:

A hypothesis can be constructed linking onset or relapse of multiple sclerosis up to six weeks after concussive head injury but thereafter with rapidly diminishing probability. Minor injury to the spinal cord could be held similarly responsible. The hypothesis is based on the unproven primary role of breach of the blood brain barrier in the pathogenesis of the plaque.

Breach of the BBB is the mechanism which is at the heart of Dr. Poser's opinion that there is a causative link between trauma to the head or spinal cord and onset or exacerbation of symptoms of MS.

In two articles: Poser, C.M., "Pathogenesis of multiple sclerosis" (1986) *Acta Neuropathol (Berl)* 71: 1 - 10, ["Poser 1986"] and Poser, C.M., "The role of trauma in the pathogenesis of multiple sclerosis: a review" (1996) *Clinical Neurology and Neurosurgery* 96: 103 - 110 (updated version of 1987 article) ["Poser 1994"], Dr. Poser advanced the contention that an alteration in the BBB is an obligatory step in the pathogenesis of the MS lesion and that minor concussive injury to the head, neck or upper back may result in such an alteration. He suggested the logical inference could be drawn that trauma could facilitate the conditions precedent to the formation of new lesions or the enlargement and activation of old ones.

In Poser 1986, Dr. Poser postulated [at p. 1]:

The available data lead to the following hypothesis: multiple sclerosis is a disease which requires the following factors for the production of demyelinating lesions of the central nervous system:

1. a genetically determined susceptibility;
2. an environmental, probably viral, probably immune mediated initiatory event producing a symptomless systemic illness;
3. a subsequent alteration of the blood brain barrier resulting from diverse mechanisms including trauma or a second, immune mediated event; and
4. a myelinoclastic plaque-forming mechanism which is operative only in the central nervous system.

In Poser 1994, Dr. Poser averred as follows [at p. 104]:

In the age of risk of clinical MS, trauma to the nervous system is by far the most common facilitator. In our automobile-oriented culture, motor vehicle accidents are the most common type of trauma, and among those the so-called rear-end collision is the most frequent. Because in so many instances, in particular in the United States, these automobile accidents result in law suits, the significance of trauma in the pathogenesis of MS has become a matter of controversy, with experts arguing either for or against the role of trauma in either producing exacerbations of an already diagnosed condition, or resulting in the appearance of classical manifestations of an hitherto asymptomatic disease. This controversy has veiled the importance of this factor in our understanding of the pathogenesis of MS. Certainly, the courtroom is not the proper venue for discovering the scientific truth.

In both of his articles, Dr. Poser questioned the value of epidemiological studies in affirming or discounting a causative association between trauma and the onset or exacerbation of symptoms. In his Poser 1986 he wrote [at p. 7]:

This is most relevant in understanding why epidemiological studies of the relationship between trauma and MS are unlikely to be fruitful. (Bamford et al 1981). MS patients represent an extremely heterogeneous group suffering from an unpredictable and highly variable illness. In clinical practice, the role of trauma must be evaluated with great circumspection: it is most unlikely that an injury to a limb, to the chest, or to the abdomen has the same effect with regard to producing changes in the permeability of the BBB as an injury to the brain or spinal cord. It is also important to consider a reasonable time relationship between the trauma and the symptoms that follow. No simple rule can be given and all cases must be judged individually.

In Poser 1994, Dr. Poser wrote as follows [at p. 108]:

In conclusion it is clear that dependence upon the "scientific evidence" of epidemiology and biostatistics in these contexts, ignores the fact that such studies can at best only be estimates within a cohort and not generalizable. They assume that MS patients are not only genetically and immunologically identical, but that their disease has reached the same extent of clinical and pathological involvement, and a similar degree of activity at the time of trauma. The reliance on

such studies to establish or deny causal relationships is a disavowal of our clinicopathologic tradition. It denigrates the work of the experimentalists and sweeps aside the experience of clinicians and neuropathologists.

In 1999, the therapeutics and technology assessment sub-committee of the American Academy of Neurology published a "special article": Goodin, D.S., Ebers, G.C., et al., "The relationship of MS to physical trauma and psychological stress" (1999) *Neurology* 52: 1737 - 1745 [the "AAN Article"]. The AAN Article was written by a panel of experts, which was chaired by Dr. Goodin and included Dr. Ebers and Dr. Sibley as well as Drs. Johnson, Rodrigues, and Wolinsky. Dr. Poser was a member of the panel for some time, but resigned before the article was published because he was not permitted to write a dissenting opinion.

The AAN Article did not report on any new study, but rather was a review of existing studies and articles with a view to resolving what had, to that point, become a matter of contention within the medical community: whether there was an association between trauma and psychological stress and either MS onset or exacerbation. The article concluded with respect to physical trauma as follows [at p. 1744]:

On the basis of strong and generally consistent class 2 evidence, any posited association of trauma, especially head trauma, with more than a small effect on either MS onset or MS exacerbation is excluded. Moreover, the preponderance of the class 2 evidence supports no association between physical trauma and either MS onset or MS exacerbation.

Class 2 evidence refers to a classification of scientific studies broken down according to their strength. A definition taken from the Practice Guideline for Clinical Practice in 2002 published in a report of the sub-committee of the academy of neurology and the MS Council for Clinical Practice Guidelines reads as follows:

Class 2 - evidence provided by a prospective study of a narrow spectrum of persons at risk for having the condition or by a retrospective study of a broad spectrum of persons with the condition compared to a broad spectrum of controls. The study measures the prognostic accuracy of the risk factor using an acceptable independent gold standard for case definition. The risk factor is measured in an evaluation that is masked to the outcome.

Dr. Goodin, who chaired the subcommittee which published the AAN Article, testified that the subcommittee was formed to conduct an assessment of the issue in response to a person who "felt that this (the association between trauma and MS onset or exacerbation) was being misused in the courtroom."

Dr. Goodin explained that the AAN Article came to its final form through a 9 step process: First, a review of the relevant literature was done. Second, a document was prepared by the panel of experts and submitted to the full subcommittee for review. Third, the full subcommittee reviewed the document, and provided feedback to the panel of experts. Fourth, the panel of experts modified the document based on the feedback received. Fifth, the document was resubmitted to the subcommittee for further assessment. Sixth, the document was sent to a

further 50 or 60 members of the academy interested in MS such that their input could be obtained. Seventh, that input was considered and the document was again reviewed by the subcommittee. Final approval of the subcommittee was provided at that stage. Eighth, the document was sent to the board of Directors of the academy. Finally, the document was submitted to Neurology, which is a peer reviewed journal, for publication.

Subsequently, in 2003, the National MS Society published its position on the relationship between trauma and MS referencing the AAN Article and two epidemiological studies: Sibley, W.A., Bamford, C.R., et al., "A prospective study of physical trauma and multiple sclerosis" (1991) Journal of Neurology, Neurosurgery and Psychiatry 54: 584 - 589 [the "Sibley Study"] and Siva, A. et al., "Trauma and multiple sclerosis: A population-based cohort study from Olmsted County, Minnesota" (1993) Neurology 43: 1878 - 1882 [the "Siva Study"]. The MS publication, in the National MS Society Information Sourcebook, concluded:

Both studies showed that there are more traumatic events among people with MS than in the healthy control group. Many traumas were caused by MS symptoms such as in-coordination, impaired balance, or abnormalities with gait or vision. These events, however, were not precipitating factors in the onset or exacerbation of the disease.

A similar approach to the issue has been taken in recent editions of the major text concerning multiple sclerosis. In Compston, A., et al., McAlpine's Multiple Sclerosis, 3rd ed. (New York: Churchill, 1998), after quoting the passage from the second edition which endorsed a causal connection between trauma and onset or exacerbation (see para. 33 above) the authors conclude at p. 100:

Our view now is that a complete reading of the literature provides no direct evidence in support of the hypothesis that trauma influences the expression or natural history of multiple sclerosis.

In Compston, A., et al., McAlpine's Multiple Sclerosis, 4th ed. (New York: Churchill, 2005), the same view was expressed somewhat more comprehensively at p. 259:

Our present view is that a complete reading of the literature provides no epidemiological evidence in support of a relationship between trauma and the cause of multiple sclerosis. Trauma does not precipitate latent multiple sclerosis. Nor does it change the activity, course and prognosis in patients with established multiple sclerosis. That position is reached using the following logic:

- The frequencies of trauma and disease activity in multiple sclerosis make it certain that these will occasionally occur together by chance.
- Epidemiological studies do not show an association between trauma and disease activity or the course of multiple sclerosis.
- Colocalization of lesions due to multiple sclerosis and cervical spondylitis, or in patients undergoing direct brain needling, does not establish causality.

· Opening of the blood brain barrier is necessary but not sufficient for establishing the cascade of events that culminates in the pathological features of multiple sclerosis.

Approached from the rigorous attitude of medical science in assessing evidence, there is no evidence for a causal relationship between trauma and multiple sclerosis. That position is not materially altered if the same questions are asked from the perspective of civil law on a balance of probabilities. (underlining added)

In another leading textbook, Kasper, D., et al., *Harrison's Principles of Internal Medicine*, 16th ed. (New York: McGraw-Hill, 2005), the authors make the following assertion at p. 2462:

The relationship of multiple sclerosis to trauma and stress: the existing evidence does not support any association of trauma with either multiple sclerosis onset or exacerbation. Similarly, a relationship between stress and either onset or exacerbation of multiple sclerosis has not been established, although this area is not easily studied because of difficulties in quantifying stress.

Since the publication of McAlpine's 4th ed. and Harrison's 16th ed. in 2005, the results of an additional epidemiological study have been published and are cited by the defendants as support for the position that there is no association between trauma and onset or exacerbation of MS symptoms. The study, Goldacre, M.J., et al., "Risk of multiple sclerosis after head injury: record linkage study" (2006) *Journal of Neurology, Neurosurgery, and Psychiatry* 77: 351 - 353 [the "Goldacre Study"], focussed upon hospital records from the Oxford National Health Service Region Hospitals from January 1, 1963, to March 31, 1999, involving some 110,993 patients admitted to hospitals with head injuries, and some 534,600 patients used as a reference cohort. The study reported that head injury patients at 6 and 11 month periods following the injury appeared slightly less prone to onset or exacerbation of MS symptoms than the reference patients. There was no appreciable difference reported based on the apparent severity of the head injury.

[46] Two significant studies implicating the biological plausibility of a causal relationship between trauma and symptomatic MS onset or exacerbation are those which I referred to earlier, Filippi, M., et al., "Magnetization Transfer Changes in the Normal Appearing White Matter Precede the Appearance of Enhancing Lesions in Patients with Multiple Sclerosis" (1998) *Ann Neurol.* 43: 809 - 814 [the "Filippi Study"] and Werring, D.J., et al., "The pathogenesis of lesions and normal-appearing white matter changes in multiple sclerosis: A serial diffusion MRI study" (2000) *Brain* 123: 1667 - 1676 [the "Werring Study"]. The Filippi Study and Werring Study provide evidence that there is a pathological process in MS lesion formation which occurs before the breakdown of the BBB can be detected on MRI scans. While the studies leave open the possibility that there are earlier, non-detectable disruptions of the BBB, or that there is more than one process involved in lesion formation, they are consistent with the position that detectable disruptions to the BBB are the result of inflammation from the process of lesion formation rather than an initiating cause of it.

[47] Although the plaintiff's expert, Dr. Poser, agreed that the breakdown of the BBB was not the first event in the formation of MS lesions accepting "that there is first of all inflammation that precedes it and maybe there are other immune phenomena" he maintained that the breakdown of the BBB is nevertheless an obligatory event and thus the biological plausibility of his theory was sustained.

[48] In cross-examination on the point Dr. Poser testified at the voir dire, in part, as follows:

Furthermore, these changes that you see on the MR transfer [pre BBB disruption], again they may precede the formation of a plaque, but they don't form a plaque, and the whole point I have made is that in order for a plaque to be formed, there must be a significant alteration of the blood-brain barrier, and I want to stress significant, because these changes that you see by MR transfer are not seen by the usual MRI, they are not gadolinium-positive, so that you can be sure that indeed at that stage there is in fact an alteration of the blood-brain barrier.

[49] Dr. Poser was cross-examined about the Filippi Study, in part, as follows:

Q All right. I'm going to show you another two studies which I'm sure you're familiar with. The Filippi study, which is under G in the documents you've got, you're familiar with this study, Dr. Poser, aren't you?

A Not only that, but I'm very familiar with Dr. Filippi.

Q Okay. The - looking at the headnote, there the conclusion says at the very last line:

These results suggest that changes in the NAWM, which is the normal appearing white matter, of patients with MS occur before lesions become evident on conventional MRI scans.

Right?

A Mr. Hartshorne, with all due respect, I just told you exactly what they're saying here.

Q I understand that, Dr. Poser. I'm - I'm putting these in because the evidence has -

A All right. Okay. I'm sorry I interrupted you.

Q Part-way down on the right-hand side of the page, it says:

In MS, lesions seen to enhance on magnetic resonance imaging (MRI) scans, after gadolinium-DTPA injection represent areas with a damaged blood-brain barrier and intense inflammation. At present, this abnormality is considered the first step of MS lesion formation that is visible with MRI. By using magnetization transfer imaging (MTI), there is, however, evidence that tissue changes may occur in MS lesions and in the normal appearing white matter, independently from enhancement.

You're familiar with that, Dr. Poser?

A Yes, of course I agree with that, but let me point out that with magnetic transfer, it is not possible to know if the involvement is just with the blood vessel or also involves the surrounding tissue. That's the problem, and this is why I say - I fully agree with what they say here, but I don't think it really addresses the problem of the obligatory change in the blood-brain barrier. The gadolinium enhancement absolutely requires an alteration of the blood-brain barrier.

[50] As for the Werring Study, Dr. Poser testified during the voir dire as follows:

Q This is under Tab Number H, Dr. Poser.

A Mm-hmm.

Q That's the Werring study?

A Yes.

Q And on the right-hand side of the summary it says:

These findings indicate that new focal lesions associated with frank BBB leakage are preceded by subtle progressive alterations in tissue integrity beyond the resolution of conventional MRI. The increases in ADC in anatomically matched contralateral regions after lesions have appeared supports the concept that structural damage and lesions causes damage or dysfunction in connected areas of NAWM.

And you're familiar with that result, their findings, Dr. Poser?

A Yes, but again I agree with all of this. There's no problem.

Q All right.

A I've just - I just answered to your previous question applies to this exact, because it's the same situation.

[51] The response of the defendants to the evidence of the plaintiff with respect to the issue of biological plausibility of Dr. Poser's theory was primarily dealt with by Dr. Goodin in response to Dr. Lehrer's expert report and his evidence which I ruled to be inadmissible. Dr. Goodin wrote as follows at p. 2 of his report dated May 4, 2005:

Certainly, trauma, especially severe trauma, can injure the CNS and disrupt the BBB, and the [sic] also certainly, the BBB is often disrupted in MS attacks. This, however, doesn't prove (or even particularly suggest) that the two are connected. Moreover, whether BBB disruption is either the first step or an 'obligatory' step is less clear. As I noted in my earlier report, there is now consistent evidence to suggest that, in the months prior to the occurrence of an area of new gadolinium enhancement (a marker of BBB disruption), the MRI demonstrates changes in what is called the magnetic transfer ratio (MTR). Thus, the BBB disruption is preceded by other focal changes in the brain and BBB disruption would seem to be a late event in the evolution of an MS lesion. Also, contrary to what Dr. Lehrer asserts, activated lymphocytes can cross into the brain through a non-disrupted BBB, so that its breakdown is not an obligatory step. We know this also

from other evidence. Thus, patients in the secondary progressive phase of MS often continue to develop new symptoms, despite not having evidence of BBB disruption.

[52] In his evidence, Dr. Goodin described the Filippi Study, among others, as offering "an incredibly important observation" because it provides evidence that prior to the breakdown of the BBB (measurable by gadolinium enhanced MRI's) "there are focal changes in the brain which are occurring which predict where that lesion is going to happen three months down the line." He regarded the conclusion that "the opening of the blood-brain barrier is not the first step" as being "crucial". Dr. Goodin was also referred to the Werring Study, which concluded as follows at p. 1674:

This longitudinal study provides new information about the pathogenesis of multiple sclerosis lesions and NAWM changes. It suggests that focal inflammatory BBB leakage, indicated by contrast enhancement, is not necessarily the initiating event in multiple sclerosis plaque formation but may be preceded, and perhaps triggered, by subtle progressive alterations in tissue integrity beyond the resolution of conventional MRI.

[53] Dr. Goodin testified he regarded the results of the Filippi Study and Werring Study as inconsistent with Dr. Poser's 1987 article, where he implied the breach of the BBB was a primary event, along with a genetically determined predisposition and a triggering event between the ages of 5 and 15.

[54] Dr. Goodin acknowledged that the Werring Study referenced other studies, which did not observe the changes to tissue integrity preceding BBB disruption, theorizing that the reason for the discrepancy is "unclear" but may imply different pathological processes or a "difference between the methods and the MRI protocols", that is, the means of testing.

[55] In my opinion, despite the limitations of the Filippi Study and the Werring Study, they do weaken the biological plausibility of Dr. Poser's theory, not only as it was originally espoused in his 1987 article, with the BBB breakdown as a primary event in the demyelination process, but also as he has modified it to be an obligatory event in that process.

[56] As I see it, if the breakdown of the BBB is a later rather than a primary part of an ongoing process leading to demyelination, the likelihood that an independent cause of BBB disruption is implicated in the process significantly lessened. Simple logic dictates the increased likelihood of coincidence, rather than causation, in the conjunction of prior trauma and subsequent demyelination in those circumstances.

[57] By way of analogy, if a person has symptoms characteristic of the onset of a cold, before going into the presence of another with a cold, his ultimate development of that condition is less likely, as a matter of logic and inference, to be due to the subsequent contact than it is to a prior cause.

- [58] When that simple analogy is put in the context of the biological plausibility of a link between trauma and symptomatic MS, it means that if there is a process that precedes and predicts BBB disruption integral to demyelination, an independent event which may cause BBB disruption is an unlikely culprit in the end result.
- [59] Accepting that the Werring Study and the Filippi Study are not dispositive of the issue, I nonetheless conclude that they attenuate the biological plausibility of the theory underlying the plaintiff's position of a causal link between trauma and symptomatic MS exacerbation.
- [60] Although the AAN Article accepted the biological plausibility of trauma as a causal link, it was written before the Werring Study was published. Of the Filippi Study, it said at p. 1738:

Also, evidence is accumulating that changes within the normal-appearing white matter precede and predict the occurrence of gadolinium enhancement (a measure of BBB breakdown) on MRI. Observations such as these raise the possibility that the breakdown of the BBB is not the initial event in the development of an MS lesion.

[footnotes omitted]

- [61] The AAN Article also noted at p. 1738:

Clearly, in the absence of such biological plausibility, any epidemiologic evidence of an association, in order to be convincing, would need to be overwhelming.

- [62] I conclude that while there is some degree of biological plausibility to the theory underlying the plaintiff's claim, its relevance or usefulness in the analytical process is attenuated by the Werring Study and the Filippi Study and the accumulating evidence that demyelinating BBB disruption is not an initiating event because it is "predicted" by the onset of a prior process and, therefore, is unlikely to be caused by an autonomous event.
- [63] It is within that context that the evidence for and against the theory must be judged.
- [64] In the course of the evidence on the voir dire, aside from the evidence relating to the biological plausibility of the causal link being advanced, the parties led evidence of, and in relation to, various studies, and as well, the plaintiff's and defendants' experts testified to the nature and effect of their respective clinical experience.

(b) Clinical Experience

- [65] The plaintiff's witnesses agreed that in their clinical experience, the apparent association between trauma and symptomatic MS onset presented only rarely. Dr. Poser confined it to less than 5% of his cases. Dr. Lehrer said he had seen only four or five out of 1,500 patients presenting with such an apparent association, and Dr. Kastrukoff considered that about 3% to 4% of patients had what he considered to be trauma contributing to MS exacerbation. He considered it rare.

- [66] So far as the witnesses for the defendants were concerned, they did not regard clinical experience or "anecdotal evidence" to be evidence of an association, rather it was a starting point for an investigation.
- [67] I accept the thrust of the defendants' evidence and the defendants' submissions on this point. While, of course, it is necessary to view all the evidence in its context, the very rare conjunction of trauma and MS exacerbation testified to by the plaintiff's experts gives a far greater flavour of coincidence than of causality to the association.
- [68] In the circumstances, little weight can be placed on the plaintiff's experts' clinical experience evidence to establish any connection given its rarity. The evidence does illustrate how difficult it would be to conclude, even assuming a causal nexus, that in an individual case, trauma was an operative cause of an MS exacerbation.
- [69] The other studies comprising the foundation on which the parties mounted their respective arguments for and against proof of a causal nexus consist of studies relating to the prevalence of MS lesions on the cervical spine, studies related to the disruption of the BBB, a study linking direct trauma to the brain by means of a thalamotomy (the insertion of a knitting needle shaped instrument called a trocar into the brain to destroy part of the brain to relieve against uncontrollable cerebral tremors) to the formation of lesions along the path of the trocar, and epidemiological studies.

(c) The Studies Relating to the Prevalence of MS Lesions on the Cervical Spine

- [70] At the centre of the evidence relating MS lesions to the cervical spine was the 1978 Oppenheimer Study (referred to in para. 44 above) based on the autopsies of 18 former MS patients, which found an excess of lesions in the cervical spine. The 1978 Oppenheimer Study distinguished the earlier Brain and Wilkinson Study (referred to in para. 44 above) and was itself conditioned by a 1993 study, Kidd, D., et al., "Spinal cord MRI using multi-array coils and fast spin echo; II. Findings in multiple sclerosis" (1993) *Neurology* 43: 2632 - 2637 [the "Kidd Study"].

- [71] The summary of the 1978 Oppenheimer Study reads as follows at p. 151:

The spinal cords were examined in eighteen cases of multiple sclerosis, with special attention to the cervical enlargement. It was found that (1) lesions in the cervical cord are about twice as common as at lower levels, (2) in this region there is a striking preponderance of fan-shaped lesions in the lateral columns. It is argued that both these findings are explicable on the theory that mechanical stresses play a part in determining the site of lesions; that such stresses are commonly transmitted to the cord via the denticulate ligaments during flexion of the spine; and that many of the lesions are attributable to vascular leakages due to tension in the denticulate ligaments. It is concluded that in patients with multiple sclerosis, neck flexion is dangerous

- [72] The earlier 1957 Brain and Wilkinson Study linked cervical spondylosis to exacerbation of MS lesions thus at p. 477:

Cervical spondylosis is predominantly a disorder of the second half of life in which disseminated sclerosis tends to run a less acute and remittent and more steadily and slowly progressive course. The myelopathy caused by cervical spondylosis tends also to be insidious in its onset and, in the absence of trauma, slowly progressive. In 5 of our series of cases trauma in the neck appears to have either precipitated the first symptoms of, or substantially exacerbated, the disseminated sclerosis and the co-existence of the two disorders has tended to bring about a deterioration more rapid than would have been expected, had either been present alone.

[73] The 1978 Oppenheimer Study did not support a direct link between cervical spondylosis and the exacerbation of MS lesions, noting that as the cervical cord was most likely to contain plaques, with or without spondylosis, the conjunction of the two conditions was likely to be coincidental rather than causal, particularly in view of his observation that the lesions did not appear to relate to points of direct impression caused by spondylosis. Oppenheimer hypothesized at p. 161 that if spondylosis plays a role in lesion formation "it is probably not by direct compression, but by the tethering effect on the dura of fibrous adhesions between it and the posterior longitudinal ligament."

[74] The 1978 Oppenheimer Study was confirmed by the later Kidd Study in which MRI's were used to detect lesions in the brain and spinal cord of 88 patients, finding more frequent abnormalities in the cervical cord than the thoracic or lumbar. The Kidd Study concluded as follows at p. 2636:

Spinal cord demyelination may develop at sites of cord compression (for example, with cervical spondylosis). However, although the present study revealed that both MS lesions and degenerative changes occurred with higher frequencies in the midcervical region, the cord lesions were usually not at the actual level of compression. Furthermore, there was no excess of disk protrusion or osteophyte formation in the MS patients compared with healthy controls. These data suggest the lack of a direct causal relationship between the degenerative changes and the site of MS lesions but would be consistent with the hypothesis that the distribution of both is influenced by a common factor such as a mechanical effect, since the midcervical cord is particularly mobile.

[footnotes omitted]

[75] The point of distinction between the plaintiff and the defendants with respect to these studies is not whether something having a "mechanical effect" is postulated by the studies, it is whether drawing a link between trauma and MS exacerbation on a basis of that postulate is more than speculation. In his evidence, Dr. Ebers pointed out that although the 1978 Oppenheimer Study revealed more lesions in the (more mobile) cervical cord than in the lumbar or thoracic area, the study did not correct for the difference in size of the various sections of the spinal cord. He also noted that of the 18 cases studied by Oppenheimer, only 3 had spondylosis, a very small number. He pointed out that the Kidd Study further detracted from the correlation between spondylosis and MS lesion development "because spondylosis is no more common [in MS sufferers] than it is in healthy controls and the

lesions are not at the site of the spondylosis."

[76] Dr. Ebers did not find the evidence of the association of lesions to spinal cord compression "very convincing". He testified:

there [are] excessive lesions in this portion of the spinal cord. It's not clear why that is. There are several other places where MS tends to hit: The optic nerve, for example, the periventricular region, the medial longitudinal fasciculus in the brain stem. None of those are well explained. They're presumably due to some anatomical idiosyncrasy that characterizes the distribution of fibres in the nervous system, perhaps. I mean I just don't know what it is.

[77] In my view, the postulation of a "mechanical effect" emerging from the 1978 Oppenheimer Study and Kidd Study, while suggestive of the possibility that some cause such as trauma may be implicated, goes no further than that. The original theory emerging from the Brain and Wilkinson Study suggesting a link between spondylosis and MS lesions has been contradicted by both Oppenheimer (1978) and Kidd. What is left is an uncertain correlation where an area of the spine amenable to mechanical stress through compression is also amenable to the formation of MS lesions. The Oppenheimer Study and Kidd Study cannot be dismissed as irrelevant, but neither can they be viewed as anything more than a basis for further investigation.

(d) The Studies Related to the Disruption of the BBB

[78] An area of significant contention between the parties was whether studies on animals subjected to trauma said to result in an animal form of MS, experimental allergic encephalomyelitis (EAE), at the site of the injury, and BBB disruption, had some probative value on the issue of a causal link between mild head injury and MS exacerbation.

[79] Dr. Ebers, for the defendants, described EAE as follows:

EAE is an acronym for experimental allergic encephalomyelitis. It's an animal model which has been extensively studied to inform us about MS and it's produced in some strains of mice, rats and guinea pigs by injecting some ground-up central nervous system material as well as a number of other things which stimulate the immune system, and it generates a disease in 10 to 14 days that's characterized by weakness and pathologically by inflammation in the brain and spinal cord.

[80] The defendants submitted at para. 231 of their written submissions on the admissibility of expert evidence that the animal studies involved "very direct and invasive trauma to the brain tissue of the subject animals". The defendants further submitted that although the extent of the injury required to break down the BBB is not precisely known, it is likely to be very substantial. The defendants' experts also relied on studies tending to show that EAE lesions preferred sites where the BBB was intact, rather than disrupted. In particular, Dr. Goodin cited a study, Knobler, R.L., et al., "Distribution of the Blood-Brain Barrier in Heterotropic Brain Transplants and its Relationship to the lesions of EAE" (1992) J. Neuropath. Exp. Neurol. 51: 36 - 39, at p. 3 of his report dated March 11, 2005, for the

proposition that in some circumstances, inflammatory lesions in EAE "have been demonstrated to occur selectively in regions where the BBB is intact, rather than in other locations where the BBB is disrupted".

- [81] Dr. Goodin cited another study, Hofstetter, H.H., et al., "Autoreactive Tcells promote post-traumatic healing in the central nervous system" (2003) *Journal of Neuroimmunology* 134: 25-34, which provided evidence that the development of EAE was not related to traumatic injury disrupting the BBB, but rather was related to the opening of the BBB by the injection of "molecules of microbial origin such as pertussis toxin" (p. 25). Dr. Goodin acknowledged that as the studies related to animals, they "may or may not relate to the human condition". It was his opinion that the studies were inconsistent with the theory proposed by Dr. Poser, which was advanced in Poser 1994 at p. 104 as follows:

Despite the fact that experimental allergic encephalomyelitis (EAE) is not an acceptable model for MS, it does share with the latter the fact that an alteration of the BBB is a necessary step in its pathogenesis. The role of an injury to the BBB as a facilitator was dramatically shown by Clark and Bogdanove, who demonstrated that the usually disseminated lesions of EAE could be 'directed to, and concentrated in, an area of the brain where the BBB had been altered by previous injury'. Levine and Hoenig reported similar observations.

[footnotes omitted]

- [82] A further area of contention between Dr. Poser and the defendants' experts arose from the plaintiff's reliance on the former's article: Poser C.M., "Trauma to the Central Nervous System May Result in Formation or Enlargement of Multiple Sclerosis Plaques" (2000) *Arch Neurol.* 57: 1074-1077. In that article, Dr. Poser wrote, as follows at p. 1074:

For many years, trivial head injuries such as concussion were considered to result only in physiological disruption of neural function without anatomical changes. However, Ommaya et al, Nevin, Oppenheimer, and Hardman pointed out that both spontaneous and experimental concussive CNS injury can cause diffuse microscopic lesions of blood vessel walls that often escape notice, on superficial examination of the brain.

[footnotes omitted]

- [83] The defendants' experts challenged Dr. Poser's assertion that "trivial head injuries" could "cause diffuse microscopic lesions of blood vessel walls" on the basis that the studies he relied on were not dealing with trivial head injuries and said nothing about microscopic lesions of blood vessel walls.
- [84] Dr. Ebers pointed out in his evidence that the Ommaya article - Ommaya, A.K., et al., "Experimental Concussion: A First Report", (1964) *J. Neurosurg.* 21: 249-265, dealt with head injuries causing concussion and "other sequelae". He testified that the Nevin article - Nevin, N.C., "Neuropathological Changes in the White Matter Following Head Injury" (1967), *J. Neuropathol. Exp. Neurol.* 26: 77-84 dealt with a high degree of trauma. Of the 52 subjects autopsied, 22 had a fractured skull and 30 had contusions of the brain. It was Dr. Ebers'

opinion that none of the injuries could be regarded as trivial.

- [85] In Oppenheimer, D.R., "Microscopic lesions in the brain following head injury" (1968) J. Neurol. Neurosurg. Psychiat. 31: 299-306, the autopsied subjects were described as follows at p 301:

Most of the brains examined were from clinically severe cases, dying from the effects of cerebral injury, often combined with cerebral anoxia from chest troubles. It is worth noting, however, that in five of the cases in which microglial clusters were observed the cerebral injury was clinically trivial, consisting of 'concussion', lasting only a few minutes. The cause of death in these was usually fat embolism or pneumonia. A brief account of one such case follows.

- [86] In his summary, Oppenheimer wrote as follows at p. 306:

Following a head injury, diffuse microscopic lesions can be seen in a high proportion of human brains. These consist of (a) capillary haemorrhages and (b) severing of nerve fibres without haemorrhage. There is a local microglial reaction, which can be demonstrated with survival times of less than 24 hours after the injury. This reaction is readily seen with the lowest power of the microscope, in frozen sections stained with a simple silver technique. The lesions are still visible many months after injury, and appear to be identical with those described by Strich (1956) in cases with long survival. They are believed to be mechanical in origin, and can be attributed to (1) surface shearing and contusion; (2) stretching and tearing of small blood vessels; (3) stretching and tearing of groups of nerve fibres; (4) tearing of nerve fibres by a crossing vessel. They are seen, not only after severe trauma, but also in cases of 'concussion'. Detailed studies of their sites and distribution could throw light on the mechanics of acceleration injuries of the brain.

- [87] The defendants contend that the studies are of limited probative value and Dr. Poser's reliance on them is misplaced because they do not deal with mild head trauma, nor do they mention breaches of the BBB.

- [88] In my view, neither the animal studies nor the autopsy studies advance the theory underlying the plaintiff's position in any meaningful way. Regarding the animal studies, EAE is an imperfect analogy for MS and there are studies that cast doubt as to the role, if any, the breakdown of the BBB has in the development of EAE. As to the autopsies, it is simply not clear what degree of trauma is necessary to provoke a disruption of the BBB, and of course, trauma induced breach of the BBB is fundamentally at issue as a cause of MS exacerbation. While the studies relied on by the plaintiff relating to the disruption of the BBB may be a piece of the puzzle regarding the onset and exacerbation of MS symptoms, their significance, if any, in the overall picture yet to be drawn is simply not determinable on the present state of the evidence.

- (e) The Gonsette Study

[89] In my reasons for ruling on the voir dire I summarized the Gonsette Study and the parties' respective submissions in relation to it thus at paras. 157-59:

Dr. Richard Gonsette, a Belgian Neurosurgeon, published a monograph (written in French) regarding the results of two thalamotomies performed on MS patients. A thalamotomie is a medical procedure in which a trocar, a knitting needle shaped instrument, is inserted through a hole in the skull into the brain to destroy part of the brain to relieve against uncontrollable cerebral tremors.

According to Dr. Poser, one of the patients died within a few days of the operation and the other within three weeks. Subsequent autopsies revealed "expression of demyelinating disease at certain levels along the path of the trocar." Dr. Poser testified that according to the Gonsette monograph the demyelination along the trocar's path was fresh, because the acute destruction of myelin produces fatty material which is eventually eaten by microphages, but the fatty material was visible as of the autopsy. The original paper by Dr. Gonsette was written in 1966. A subsequent monograph was written in 1972. Dr. Poser relied on the Gonsette monograph as establishing a link between physical trauma and lesion development.

The defence experts challenged the worth of the Gonsette study on several bases. Dr. Ebers (who is fluent in French) pointed out that the original 1966 paper gave no indication of the freshness of the lesions. He also testified that naturally occurring lesions tend to be symmetrical on each side of the brain and no investigation was done to compare the area at issue with the other side of the brain. In other words, finding symmetry would support the defendants' contention that the lesions pre-existed the thalamotomie. He also testified that there was no basis for Dr. Poser to assert the lesions were fresh and pointed out that the autopsies, and presumably the patient's deaths, occurred months after the thalamotomies, not within days or weeks. He noted that the plaques could relate to the clinical problems for which the procedures were being carried out. Dr. Ebers cited other studies that failed to replicate the Gonsette results, and noted that following biopsies MS sufferers do not appear to have an exacerbation of symptoms.

[90] In my view, the inference of cause and effect from the insertion of a trocar into the brain and the subsequent discovery by autopsy of lesions along its pathway is very tenuous due to the presence of other reasonable explanations for the apparent correlation and the absence of corroboration from other studies or other evidence.

[91] Moreover, Dr. Poser's evidence on the Gonsette Study was unsatisfactory and contradictory. In his evidence in chief, he testified that the two patients involved in the Gonsette Study died "within a matter of a few days and three weeks after the surgery" respectively. In cross-examination he agreed that in an earlier trial in Australia (Edman, October 1, 2003) he testified that the autopsies were conducted more than a year after the surgery. Later in cross-examination he testified that he re-read the study and the patients died two months and seventeen months after the surgery, respectively.

[92] Dr. Poser also testified that he based his conclusions that the lesions were fresh on Gonsette's reference to the presence of fat filled macrophages, which to be considered fresh would have to appear within six to eight months of the formation of the lesions. If that were so, the presence of fat filled macrophages at the time of the autopsy, seventeen months post-thalamotomy, would appear discordant with cause and effect. As well, there is a question whether the presence of lesions only two months post-thalamotomy indicates that the lesions were present before the thalamotomy. The uncertainty of Dr. Poser's evidence, the lack of corroborating evidence, and the evidence of Dr. Ebers concerning the shortcomings of the Gonsette Study substantially weaken its force as indirect evidence of the plaintiff's underlying theory. I would assign it little weight.

(f) The Epidemiological Studies

[93] The difficulty in finding determinative direct or indirect physical evidence addressing the issue of an association between trauma and MS onset or exacerbation has led to reliance upon epidemiological studies designed, through comparison of MS patients with "control" subjects or through following MS patients over a period of time to develop a statistical assessment of the proposed connection.

[94] In opposition to the plaintiff's evidence, the defendants relied upon three epidemiological studies in particular, the Sibley Study and the Siva Study, both referenced in the AAN Article (para. 46 of *Taylor v. Liong* (2007); para. 44 above) and the Goldacre Study. In *Taylor v. Liong* (2007), I summarized those three studies as follows in paras. 166-174:

The Sibley Study was a prospective study, that is a study which follows a targeted group to identify what occurs, rather than a retrospective study which after identifying the target group takes a history from them. Prospective studies are regarded as more valuable than retrospective ones because the latter relies on the memories of the subjects which may be selective and subject to a recall bias, being more prone to recalling trauma if it preceded an exacerbation, for example, than if it did not.

The Sibley Study was conducted through the MS clinic at the University of Arizona. It tracked 170 MS patients and 134 age and sex matched controls over a period of 8 years. Because of drop outs from the study, the average length of time the patients were followed was 5.2 years. The purpose of the study was to attempt to identify environmental factors which may be risk factors with MS onset or exacerbation.

The method of collecting the data involved patients and controls each completing a questionnaire every 30 days to record a broad range of occurrences in the previous month. If the questionnaire was not completed, a nurse would follow up by telephone interview. Each MS patient had a neurological examination every three months and each patient was examined if they experienced what may have been an exacerbation. In relation to trauma, the three month and six month periods after trauma were regarded as "at risk" for the purpose of the study. The study found "no significant correlation between all traumas and disease activity." The study also found in 95 patients a negative correlation between traumatic episodes and exacerbations, "due to less

activity of the disease during a three month period following surgical procedures and fractures."

(i) The Siva Study

The Siva Study was based on records from the Mayo Clinic which served the population of Olmstead County in New York (which had a population of approximately 100,000 in 1990). The Siva Study identified 225 cases of MS from 1905 to 1991 (known as the incidents cohort). The study identified 164 cases involving patients alive as of December 1991 as the prevalence cohort. It also followed a sub-cohort of 819 head injury cases between 1935 and 1984 (known as the head injury cohort).

The study looked at "mechanical trauma" defined as "head and spinal trauma, fractures of the long or small bones of the extremities, and other skeletal fractures." Head trauma meant "evidence of skull fracture, loss of consciousness, focal neurologic signs or post traumatic amnesia." Spinal trauma was trauma to the spine resulting in "compression of vertebrae body or other fractures."

Among the prevalence cohort, 39 out of 164 experienced mechanical trauma. In the result, the study reported no significant difference for the number of exacerbations following the trauma as to the six months preceding the episode of trauma. Of the 819 in the head injury cohort, only two individuals ended up developing MS. One had the first clinical onset three years after the injury, the other 21 years later.

(ii) The Goldacre Study

The Goldacre Study, which was published in 2006, was another records linkage study. It was a study conducted at Oxford University and used data from the Oxford National Health Service Region Hospitals during a period from January 1, 1963, to March 31, 1999. A cohort of 110,993 individuals admitted to a hospital with head injuries was identified and a reference cohort of 534,600 patients not admitted for head trauma was established for comparative purposes.

The study reported that the observed number of MS cases in the head injury cohort at 6 month and 11 month periods following injury were slightly under the expected number based on the MS cases identified in the reference cohort. The study also noted that when the head injury cohort was broken down based on severity of the injury, there was no significant difference in the result.

The abstract of the Goldacre Study reads in part as follows:

Background: The possibility that head injury may influence the development of multiple sclerosis (MS) has been studied inconclusively in the past.

Objective: To determine whether head injury is associated with an increased risk of MS.

Method: Analysis of data base of linked hospital and death records comparing the occurrence of MS in a cohort of people admitted to hospital with head injury and a reference cohort.

Results: The rate ratio for MS after head injury, compared with the reference cohort, was 1.1 (95% confidence interval, 0.88 to 1.36). There was no significant increase in the risk of MS at either short or long time periods after head injury. Using length of hospital stay as a proxy for severity of injury, there was no significant increase in the rate ratio for MS after head injuries with hospital stays of less than two days (rate ratio = 1.1 (0.71 to 1.57)), two or more days (rate ratio = 1.0 (0.68 to 1.45)), or seven or more days (rate ratio = 1.3 (0.64 to 2.34)).

Conclusions: The method used, record linkage, ensures that patients' recollection of injury, or any tendency to attribute MS to injury, cannot have influenced the results. Injuries to the head were not associated with either the aetiological initiation or the clinical precipitation of onset of multiple sclerosis.

- [95] The defendants contend that the Sibley Study, Siva Study and Goldacre Study provide no positive evidence of a causal connection between trauma and MS exacerbation. They rely on evidence that none of the three studies provide evidence of an association between trauma and subsequent MS exacerbation that has any statistical significance supportive of the plaintiff's experts' theory. The defendants assert that because trauma is a relatively frequent occurrence in the general population the probability of a coincidental association is high and the significance of a temporal relationship is therefore significantly reduced.
- [96] In that regard the defendants relied on a study of the US Department of Human and Health Services titled "Types of injuries and impairments due to injuries" (1986) National Health Survey, Series 10; (PHS) 159: 8-26 which was relied on by Dr. Goodin in his March 11, 2005 report. Dr. Goodin reported, at p. 2, based on that study, that "the annual incidence of injuries (of all types) in the United States has been estimated to be 33.2 injuries/100 persons/year; a figure that almost certainly underestimates the true incidence of minor traumatic injuries" [footnotes omitted].
- [97] The position of the plaintiff is that epidemiological studies can neither prove nor disprove a relationship between trauma and MS, and that while the studies in question do not detect such a relationship, they are impaired by methodological flaws and a lack of statistical power sufficient to illuminate the very small proportion of trauma induced exacerbation as posited. The plaintiff relied on evidence from Dr. Ebers that the prevalence rate of MS (the overall number of MS sufferers in a given population) is about 1 in 1,000 and the incidence rate (the number of new cases in a given year) is 5.7 per 100,000. The plaintiff submitted that given those numbers and the associated percentage of MS exacerbations due to trauma as being less than 5%, to be meaningful an epidemiological study would have to incorporate an unmanageably large number of patients and/or controls.
- [98] The plaintiff pointed out that in relatively small epidemiological studies, missing even a single case of a temporal relationship between trauma and MS could be quite significant statistically. The plaintiff submitted at para. 135 of the written submissions on admissibility of expert evidence:

Absence of evidence is not evidence of absence. More specifically, the inability to detect certain conditions (lacking sufficiently powerful tools to do so) does not amount to showing that those conditions do not exist. Nor does the fact that the proposed effect cannot be proved or disproved make the theory unscientific.

- [99] Insofar as the Sibley Study is concerned, there was evidence that Dr. Sibley excluded symptoms that did not persist for at least 48 hours in the face of the then current view that 24 hours was sufficient. The plaintiff submitted this increased the possibility of omitting true exacerbations to guard against the risk of false positives.
- [100] The plaintiff submitted at para. 167 of the written submissions that given the methodology of the Sibley Study, "[t]he study was only as accurate as the patients that [who] participated in it" and could be affected by "poor reporting practices" or a failure on the patient's part to recognize a true exacerbation. The plaintiff asserted that questionnaires were a less effective way of identifying significant events than taking a history and that patients with memory problems were not screened out of the study.
- [101] With regard to the Siva Study, the plaintiff submitted that the most serious shortcoming was that it was severely limited in power, based on only 819 patients with head injury and 39 of 164 in the prevalence cohort, none of whom had serious head injury. The plaintiff further submitted that the Siva Study did not examine the effect of spinal injuries on MS exacerbation which would reduce its utility.
- [102] The plaintiff further submitted, at para. 183 of the written submissions, that methodological problems beset the Siva Study in that it relied on information from medical records of all types of physicians, not just neurologists, relied on records back or went back and reach back in time to periods when "[t]he criteria used to diagnose MS was imprecise". The plaintiff also repeated criticism of the Sibley Study relating to the underreporting of symptoms and the preponderance of undetected lesions. Neither mild concussions nor whiplash injuries were included in the study which, the plaintiff submitted, may not properly define the "at risk group".
- [103] The plaintiff, while acknowledging the large sample size of the Goldacre Study, submitted that nonetheless it was not of sufficient magnitude to determine the issue of a causal link between trauma and MS.
- [104] The plaintiff questioned whether the use of a minimum six month post-trauma window for development of an exacerbation might "mask" a greater than expected incidence of exacerbation within the initial 3.5 months, which was Dr. Poser's temporal criterion for a causal link. The plaintiff also noted that the collected data for the Goldacre Study relied on the first admission to hospital for MS rather than on the first symptoms, and since often diagnosis and hospitalization is delayed by up to four years, it is not possible to conclude with certainty from the study anything definitive about the relationship between trauma and MS onset or exacerbation.

- [105] The plaintiff, at para. 201 of the written submissions, also criticised the methodology of the Goldacre Study in that it was based on "brief statistical abstracts" of hospital records and thus drew "no systematic differentiation between mild and serious head injuries". The plaintiff further submitted that there was evidence that MS would be under reported on death certificates as it is rarely a direct cause of a death. Accordingly, the plaintiff contended, the records may be skewed. (While that may be so, presumably they were similarly skewed for both cohorts).
- [106] Finally, the plaintiff submitted that based on the evidence of Dr. Ebers, the reliability of the data in the Goldacre Study may be undermined by those head injured patients who "would never seek treatment for their injuries" (para. 203) or who would not be hospitalized.
- [107] I accept that the epidemiological studies are neither methodologically flawless nor dispositive of the issue. In my view, however, they cast considerable doubt on the proposition being advanced by the plaintiff, simply because even on the plaintiff's view, they buttress the conclusion that temporal associations between trauma and MS exacerbation are highly anomalous. That being so, logically the relationship lies much more comfortably in the realm of coincidence than it does in the realm of causation.

THE LAW RELATING TO CAUSATION

- [108] In *Athey v. Leonati*, [1996] 3 S.C.R. 458, Major J., speaking for the court, reviewed the general principles respecting causation in paras. 13-16 of his judgment as follows:

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 S.C.R. 311; *McGhee v. National Coal Board*, [1972] 3 All E.R. 1008 (H.L.).

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] S.C.R. 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 S.C.R. 21; *Bonnington Castings, Ltd. v. Wardlaw*, [1956] 1 All E.R. 615 (H.L.); *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 B.C.L.R. (2d) 114 (B.C.C.A.), *aff'd* [1989] 2 S.C.R. 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 E.R. 475, at p. 490, 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.

[109] In *Snell v. Farrell*, [1990] 2 S.C.R. 311, Sopinka J., in addressing the question of proof of causation not susceptible to "be determined by scientific precision", wrote at pp. 328-31 as follows:

I am of the opinion that the dissatisfaction with the traditional approach to causation stems to a large extent from its too rigid application by the courts in many cases. Causation need not be determined by scientific precision. It is, as stated by Lord Salmon in *Alphacell Ltd. v. Woodward*, [1972] 2 All E.R. 475, at 490:

essentially a practical question of fact which can best be answered by ordinary common sense rather than abstract metaphysical theory.

Furthermore, as I observed earlier, the allocation of the burden of proof is not immutable. Both the burden and the standard of proof are flexible concepts. In *Blatch v. Archer* (1774), 1 Cowp. 63, 98 E.R. 969, Lord Mansfield stated at p. 970:

It is certainly a maxim that all evidence is to be weighed according to the proof which it was in the power of one side to have produced, and in the power of the other to have contradicted.

In many malpractice cases, the facts lie particularly within the knowledge of the defendant. In these circumstances, very little affirmative evidence on the part of the plaintiff will justify the drawing of an inference of causation in the absence of evidence to the contrary. This has been expressed in terms of shifting the burden of proof. In *Cummings v. City of Vancouver* (1911), 1 W.W.R. 31 (B.C.C.A.), Irving J.A. stated at p. 34:

Stephens [sic] in his Digest (Evidence Act, 1896) says: "In considering the amount of evidence necessary to shift the burden of proof, the Court has regard to the opportunities of knowledge with respect to the fact to be proved, which may be possessed by the parties respectively."

Hollis v. Young (1909) 1 K.B., 629, illustrates the rule that very little affirmative evidence will be sufficient where the facts lie almost entirely within the knowledge of the other side.

In *Dunlop Holdings Ltd.'s Application*, [1979] R.P.C. 523 (C.A.), Buckley L.J. affirmed this principle in the following terms at p. 544:

Where the relevant facts are peculiarly within the knowledge of one party, it is perhaps relevant to have in mind the rule as stated in Stephen's Digest, which is cited at page 86 of Cross on Evidence [3rd ed.]:

"In considering the amount of evidence necessary to shift the burden of proof, the court has regard to the opportunities of knowledge with respect to the facts to be proved which may be possessed by the parties respectively."

"This does not mean", Sir Rupert continues, "that the peculiar means of knowledge of one of the parties relieves the other of the burden of adducing some evidence with regard to the facts in question, although very slight evidence will often suffice" [emphasis added].

See also *Diamond v. British Columbia Thoroughbred Breeders' Society* (1965), 52 D.L.R. (2d) 146 (B.C.S.C.), at p. 158; *Pleet v. Canadian Northern Quebec R. Co.* (1921), 64 D.L.R. 316 (Ont. S.C., App. Div.), at pp. 319-320; and *Guaranty Trust Co. of Canada v. Mall Medical Group*, [1969] S.C.R. 541 at p. 545.

These references speak of the shifting of the secondary or evidential burden of proof or the burden of adducing evidence. I find it preferable to explain the process without using the term secondary or evidential burden. It is not strictly accurate to speak of the burden shifting to the defendant when what is meant is that evidence adduced by the plaintiff may result in an inference being drawn adverse to the defendant. Whether an inference is or is not drawn is a matter of weighing evidence. The defendant runs the risk of an adverse inference in the absence of evidence to the contrary. This is sometimes referred to as imposing on the defendant a provisional or tactical burden. See Cross, *op. cit.*, at p. 129. In my opinion, this is not a true burden of proof, and use of an additional label to describe what is an ordinary step in the fact-finding process is unwarranted.

The legal or ultimate burden remains with the plaintiff, but in the absence of evidence to the contrary adduced by the defendant, an inference of causation may be drawn although positive or scientific proof of causation has not been adduced. If some evidence to the contrary is adduced by the defendant, the trial Judge is entitled to take account of Lord Mansfield's famous precept. This is, I believe, what Lord Bridge had in mind in *Wilsher* when he referred to a "robust and pragmatic approach to the facts" (at p. 569).

It is not therefore essential that the medical experts provide a firm opinion supporting the plaintiff's theory of causation. Medical experts ordinarily determine causation in terms of certainties whereas a lesser standard is demanded by the law. As pointed out in *Louisell, Medical Malpractice*, vol. 3, the phrase "in your opinion with a reasonable degree of medical certainty," which is the standard form of question to a medical expert, is often misunderstood. The author explains, at p. 25-57, that:

Many doctors do not understand the phrase as they usually deal in "certainties" that are 100% sure, whereas "reasonable" certainties which the law requires need only be more probably so, i.e., 51%.

In *Harvey, Medical Malpractice* (1973), the learned author states at p. 169:

Some courts have assumed an unrealistic posture in requiring that the medical expert state conclusively that a certain act caused a given result. Medical testimony does not lend itself to precise conclusions because medicine is not an exact science.

[110] Recently, in *Resurface Corp. v. Hanke*, 2007 SCC 7, [2007] 1 S.C.R. 333, McLachlin C.J.C., for the Court, restated the basic test for determining causation in the context of distinguishing between the "material contribution" test and the "but for" test. In that case, she held as follows at paras. 20-25:

Much judicial and academic ink has been spilled over the proper test for causation in cases of negligence. It is neither necessary nor helpful to catalogue the various debates. It suffices at this juncture to simply assert the general principles that emerge from the cases.

First, the basic test for determining causation remains the "but for" test. This applies to multi-cause injuries. The plaintiff bears the burden of showing that "but for" the negligent act or omission of each defendant, the injury would not have occurred. Having done this, contributory negligence may be apportioned, as permitted by statute.

This fundamental rule has never been displaced and remains the primary test for causation in negligence actions. As stated in *Athey v. Leonati*, at para. 14, per Major J., "[t]he 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." Similarly, as I noted in *Blackwater v. Plint*, at para. 78, "[t]he rules of causation consider generally whether 'but for' the defendant's acts, the plaintiff's damages would have been incurred on a balance of probabilities."

The "but for" test recognizes that compensation for negligent conduct should only be made "where a substantial connection between the injury and the defendant's conduct" is present. It ensures that a defendant will not be held liable for the plaintiff's injuries where they "may very well be due to factors unconnected to the defendant and not the fault of anyone": *Snell v. Farrell*, at p. 327, per Sopinka J.

However, in special circumstances, the law has recognized exceptions to the basic "but for" test, and applied a "material contribution" test. Broadly speaking, the cases in which the "material contribution" test is properly applied involve two requirements.

First, it must be impossible for the plaintiff to prove that the defendant's negligence caused the plaintiff's injury using the "but for" test. The impossibility must be due to factors that are outside of the plaintiff's control; for example, current limits of scientific knowledge. Second, it must be clear that the defendant breached a duty of care owed to the plaintiff, thereby exposing the plaintiff to an unreasonable risk of injury, and the plaintiff must have suffered that form of injury. In other words, the plaintiff's injury must fall within the ambit of the risk created by the defendant's breach. In those exceptional cases where these two requirements are satisfied, liability may be imposed, even though the "but for" test is not satisfied, because it would offend basic notions of fairness and justice to deny liability by applying a "but for" approach.

[111] Counsel for the plaintiff does not contend that this is a case where the court is entitled to make an inference of causation in the absence of scientific proof. Rather, the plaintiff submitted that the court should not approach the question of causation rigidly, but with common sense following "a robust and pragmatic approach to the facts". Counsel for the plaintiff submitted the following in para. 85 of his written submissions on this application:

Pursuant to Snell, the Court is entitled to take a pragmatic approach to causation even in the absence of supporting medical information. In other words the Court is entitled to consider the Plaintiff's previous health and the temporal connection with the Accident. If no alternative explanation was offered by the Defendants an inference of causation would have been likely, because there would have been no evidence to the contrary. Where, as here, the defence does adduce contrary information, the Court must consider whether it was in the Plaintiff's power to adduce more evidence of causation or whether more evidence contrary to causation could have been adduced by the Defendant. In doing so the Court ought to take a robust and pragmatic approach to the facts.

[112] The issue before me in this case is somewhat similar to that which confronted Osler J. in *Rothwell v. Raeb* (1988), 66 O.R. (2d) 449, 54 D.L.R. (4th) 193 (H.C.J.), aff'd 2 O.R. (3d) 332, 76 D.L.R. (4th) 280 (C.A.).

[113] In that case, Osler J. introduced the problem of causation at p. 457 of his judgment:

It is apparent that all three of the plaintiffs' actions depend for their success upon a finding that there was a causal relationship between the administration of DPTP vaccine and the severe brain damage or encephalopathy suffered by the minor plaintiff. If, on the balance of probabilities, it is found that the administration of DPTP can cause encephalopathy, or permanent, serious brain damage, the actions may succeed. If it is found, on a balance of probabilities, that such a causal relationship can exist, the plaintiffs have the burden of showing, again on the balance of probabilities, that it did exist with respect to Patrick. The issue of causation, or the etiology of Patrick's condition, occupied a major part of the trial. If it is more probable than not that there is no causal connection between the pertussis component and severe, permanent brain damage, the actions must fail.

[114] In concluding that the standard of proof was not met on the evidence before him, Osler J. held as follows at p. 506:

While there are certainly major differences in fact and not all of the evidence was the same, Lord Justice Stuart-Smith, in *Loveday v. Wellcome*, supra, had to decide the precise question of causation that is before me. In dealing with the question of proof and with reference to *Wilsher v. Essex Area Health Authority*, [1988] 1 All. E.R. 871 (H.L.), he had this to say, at p. 16 of the unreported judgment:

In my judgment the decision [Wilsher] made it clear beyond doubt that the Court must decide as a question of fact whether the vaccine can cause permanent brain damage in young children and that the onus of doing so rests on the Plaintiff and the standard of proof is the balance of probability.

That statement accords precisely with our law, as I view it: see, for example, *Buchan v. Ortho Pharmaceutical (Canada) Ltd.* (1986), 54 O.R. (2d) 92, 25 D.L.R. (4th) 658, 32 B.L.R. 285 (C.A.), and *Rogin v. Shannon* (1986), 37 C.C.L.T. 181 (Ont. H.C.J.).

In my view, the plaintiffs have not discharged the onus of showing that it is more likely than not that the pertussis component of the DPTP vaccine can cause serious permanent brain damage. The evidence persuades me that it cannot do so.

[115] In a similar vein, the question before me of whether trauma, including mild head trauma or whiplash injury, can cause the exacerbation of MS symptoms, was the precise question before the court in *Dingley supra*. As did the Lord President and the House of Lords in that case, I conclude that the plaintiff has not proved on a balance of probabilities that such a causal connection exists.

[116] My conclusion is neither intended nor capable of resolving any lingering scientific debate on the effect of trauma on MS. My reference to and reliance on the scientific evidence in this case, as I expressed in my ruling on admissibility, is necessary to understand whether the plaintiff's MS exacerbation could fall within the scope of the risk of injury to which the defendants' tortious behaviour exposed her.

[117] In finding that the evidence falls short of establishing a causal link on a balance of probabilities, I also rely on the fact that a substantial majority of the relevant scientific community has rejected the notion of a causal connection based on developments in understanding the pathogenesis of the disease, epidemiological studies, reanalysis of previous studies said to support the link, and a weakening of the biological plausibility of the theory through studies such as the Werring Study and the Filippi Study. In the result, I have an advantage over the court in *Dingley, supra*, in knowing what the future held for the issue in the scientific community in the years following that judgment.

[118] I do not find, in the evidence before me, persuasive proof of a causal relationship between trauma and MS exacerbation. Dr. Poser, who was the primary witness for the plaintiff, testified as an advocate for a theory that once had currency but has since been eroded by the advance of scientific studies and knowledge. I found in his evidence a tendency to overstate the implications of studies that he relied on, and to be unduly dismissive of those that undermined his theory. While he has clearly achieved the status of a foremost expert in his field of neurology, I find that his commitment to his own theory of causation has to some extent impaired his objectivity and reliability as an expert witness.

- [119] Dr. Kastrukoff, who subscribed to Dr. Poser's theory, did not offer any new or different insights and acknowledged in his evidence that the "literature" on the subject did not positively support linking trauma to MS exacerbation. Dr. Kastrukoff primarily relied on his clinical experience. In that regard, as I earlier noted, I accept the defendants' position that clinical experience, particularly in a context where the asserted relationship is so rare, and the chance of coincidence is so high, is no more than a starting point for further enquiries. I would thus draw no positive inferences from clinical experience in those circumstances, and therefore do not find affirmative support for the proposition in Dr. Kastrukoff's evidence.
- [120] On the other side of the ledger, I find Drs. Goodin, Ebers, and Sibley, although thoroughly and skilfully cross-examined by counsel for the plaintiff, to have a more detached quality to their evidence than did Dr. Poser to his. Clearly all three of the defence witnesses are among the world's leading experts in the field of neurology and the study of MS and in my view, while each advanced opinions based on the shifting sands of scientific study and research, none of them attempted to overstate his respective opinion or the foundations for it. I thus found the evidence of Drs. Goodin, Ebers and Sibley to be more objective and hence more reliable than that of Dr. Poser.
- [121] As to the underpinnings of the plaintiff's theory, for the reasons I have attempted to explain, I do not find in any of the studies or analysis relied on by the plaintiff, taken either individually or collectively, a logical linchpin for proof of the causal relationship being espoused, particularly in view of its asserted rarity, and the growing uncertainty of the role that its asserted mechanism - the disruption of the BBB - has in the demyelination process.
- [122] I find that the likelihood of a causal connection between trauma and MS exacerbation is significantly less than that of a coincidental connection, in light of all the evidence adduced, and the opinion of a substantial majority of the scientific community.
- [123] I thus conclude that even on a robust and pragmatic view of the evidence, it does not support proof of a causal connection between mild trauma, including whiplash, and MS exacerbation, on a balance of probabilities.

"A.F. Cullen J."

The Honourable Mr. Justice A.F. Cullen

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