MEDICAL MALPRACTICE

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PACIFIC MEDICAL LAW

CAUSATION IN MEDICAL MALPRACTICE ACTIONS: AN ILLUSTRATION PROVIDED BY *GOODMAN V VILJOEN*

The general principles of causation in medical malpractice claims have long been established. Medical malpractice actions are subject to the same requirements as other types of personal injury torts; the plaintiff must prove, on a balance of probabilities, that the defendant caused the injury. The generally applicable test is the "but for" test which requires the plaintiff to show that '"but for" the defendant's negligent act, the injury would not have occurred.'[1] The difficulty in medical malpractice claims arises from the complexity of proving cause and effect in a medical context. Proof of causation almost always requires expert evidence.

Compounding the difficulty of proving causation in medical malpractice claims is the difference between medical and legal causation. In the legal context, causation need only be established on a balance of probabilities. The '"but for" test is to be applied in a robust common-sense fashion.[2] This robust and pragmatic approach to the causation analysis is applicable in cases where the defendant leads no evidence to the contrary *and* in cases involving conflicting evidence for the judge to weigh.[3] Legal causation does not demand scientific precision or scientific evidence of the precise contribution the defendant's negligence made to the injury.[4] It is "essentially a practical question of fact which can best be answered by ordinary common sense".[5] Although the burden of proof remains with the plaintiff, in some circumstances a common sense inference of causation may be drawn from the evidence without positive scientific proof.[6] In a medical context, causation is subject to stricter requirements. The conclusions that can be drawn from studies require a high level of precision to establish statistical significance. Cause and effect in medicine involves scientific proof. When medicine is an integral part of a legal claim, it is important to maintain a clear divide between these two understandings of causation. As was observed by Sopinka J. in *Snell v Farrell*:

> "It is not therefore essential that medical experts provide a firm opinion supporting the [p]laintiff's theory of causation. Medical experts ordinarily determine causation in terms of certainties whereas a lesser standard is demanded by the law."[7]

In *Goodman v Viljoen*[8], this medical/legal causation dynamic was central to the outcome of the case. It provides an excellent example of the different standards of proof in the medical and legal contexts. The case also illustrates how inferences of causation can support a legal claim despite a complex medical backdrop and medical uncertainty.

Mrs. Goodman was pregnant with twins when she developed a urinary infection for which she was prescribed antibiotics. The following week, Mrs. Goodman experienced a leakage of fluid and made a call to the office of her obstetrician, Dr. Viljoen. She never spoke directly with Dr. Viljoen but reported the fluid leakage to his secretary who subsequently called back to advise that this was related to her infection and that Mrs. Goodman should continue to take her antibiotics. Two days later, Mrs. Goodman went into premature labour. Upon attendance at hospital, her physician confirmed that her membranes had ruptured. Her twins were born at 29 weeks gestation via caesarean section. They subsequently developed cerebral palsy.

In order to succeed in the action, the plaintiffs had to prove that the twins' cerebral palsy was caused by the defendant's negligence. The defendant acknowledged that if Mrs. Goodman reported a leakage of fluid, the standard of care required him to see her or send her immediately to hospital. Whether this report was made was at issue at trial but on appeal the only issue was causation. The difficulty the plaintiffs faced was in proving that, had the defendant properly treated Mrs. Goodman, the twins would not have developed cerebral palsy despite their premature birth. The prematurity itself was unrelated to the defendant's negligence. There was no suggestion that anything the defendant should have done would have allowed Mrs. Goodman to carry the twins to

term. Consequently, the plaintiffs needed to establish that it was the inadequate treatment, and not the prematurity, that resulted in the twins developing cerebral palsy.

There was no dispute amongst the experts that the twins' cerebral palsy was caused by diffuse periventricular leukomalacia (PVL). PVL is a known risk of prematurity. The Ontario Court of Appeal described the process of injury in this way:

> "PVL involves the inadequate blood supply to an area of the premature baby's brain referred to as the watershed zone. Arterial blood supplies to the brain meet in the watershed zone. In premature infants, the arterial membranes may not develop fully. The brain cells affected by PVL are unstable and vulnerable. The expert evidence suggested that the damage to the affected areas of the brain caused by PVL occurs during delivery or in the first few days following birth.
> …
> PVL is associated with the later onset of [cerebral palsy]. The descending nerve tracts to the legs and arms pass through the area of the brain adversely affected by PVL.
> PVL may be caused by hypoxia, that is reduced oxygen in the blood flow circulating to the affected area of the brain, or ischemia, that is a deficiency in the blood supply to the affected area of the brain due to reduced blood pressure. Either hypoxia or ischemia results in the death of brain cells in the affected area of the brain. The twins' PVL was likely caused by ischemia."[9]

Babies born earlier than 34 weeks gestation are more susceptible to PVL than term babies and PVL is the most common cause of brain injuries in premature infants.

It was undisputed at trial that if Mrs. Goodman had seen a doctor on the day she reported the fluid leakage, she would have received two doses of antenatal corticosteroids. Antenatal corticosteroids induce the production of enzymes throughout the fetus within 24-48 hours of administration that serves as a substitute for the surge of hormones that happens in full term babies immediately before birth. This hormone surge accelerates the maturation process and assists in the transition by the fetus to life outside the womb, but it does not occur in premature babies. Administration of antenatal corticosteroids is known to have short term and long term benefits in premature babies. Since Mrs. Goodman never saw a doctor the day she reported the fluid leakage, she was not given antenatal corticosteroids until the day of the twins' birth, so close to the time of birth that it was probably ineffective, and there was only time for a single dose. The discrete causation issue became whether "but for" the failure to receive a full dose of antenatal corticosteroids, the twins would not have developed cerebral palsy, or the severity of their cerebral palsy would have been materially reduced.

The defence position was that causation could not be proven in the absence of any direct scientific proof of a cause and effect relationship between the administration of antenatal corticosteroids and a reduction in a premature baby's risk of developing PVL. The defence argued that since there were no clinical studies or any other form of direct scientific evidence showing such a correlation, the plaintiffs' claim must fail. The plaintiffs argued that causation could be established on the balance of probabilities without the kind of evidence required for the scientific standard of proof. Two experts (a neonatologist/ pediatrician and an obstetrician/maternal fetal medicine specialist) opined that had Mrs. Goodman received a full dose of antenatal corticosteroids two days before her babies were born, the twins would not have developed cerebral palsy, or, alternatively, their disabilities from cerebral palsy would have been less severe.

The plaintiffs structured their case around "biological plausibility." There was evidence showing that antenatal corticosteroids had a maturation effect on tissues in various parts of the fetus, including the lungs and intestines. This maturation process reduced the risk of other conditions known to cause cerebral palsy. The plaintiffs' experts opined that one could infer that the maturation of membranes known to occur in other parts of the fetus would work in a similar way in the parts of the brain adversely affected by PVL (the arteries located in the watershed areas of the premature baby's brain) and reduce the risk and the severity of PVL. While there were no studies showing a correlation between administration of corticosteroids and reduction in PVL, the plaintiffs' experts pointed to other studies to support their opinion. Studies had shown that administration of antenatal corticosteroids had a positive effect on neonatal blood pressure and precipitous drops in blood pressure is a known cause of PVL. Administration of antenatal corticosteroids had a known correlation with reduction in a different type of leukomalacia than the twins had. An animal study showed a correlation between administration of corticosteroids and fetal brain development. Finally, an analytical review of 21 controlled studies reported a reduction in the risk of cerebral palsy through the administration of antenatal corticosteroids. However, this reduction was not statistically significant for scientific purposes, it did not distinguish between full term and preterm babies, and it did not differentiate between the causes of cerebral palsy. On the other hand, the review also did not consider those babies who developed cerebral palsy but suffered fewer disabilities as a result of antenatal corticosteroid administration.

The trial judge accepted the biological plausibility theory. She found:

> "On the totality of the evidence before me, it is reasonable to infer that since PVL results from the immaturity of the pre-term infant's brain and vascular system, and [antenatal corticosteroids] have a maturational effect beyond lung function to mature these systems, that it is more likely than not that the administration of [antenatal corticosteroids] would reduce the risk of PVL. Thus, but for the defendant's negligence, the twins would not have suffered from PVL, and consequently would not have suffered from [cerebral palsy].
> …

> Even if I am incorrect in my conclusion that the PVL (and therefore the [cerebral palsy]) would not have occurred but for the failure to receive a full course of [antenatal corticosteroids], this is not fatal to the plaintiffs' claim. I am satisfied that the evidence before me establishes on a balance of probabilities that [antenatal corticosteroids] reduce the risk and severity of [cerebral palsy] in general, that is, no matter what the cause of the [cerebral palsy]."[10]

Despite the fact that the most applicable scientific analysis did not reach a level of statistical significance, and the fact that none of the literature specifically found a connection between the use of antenatal corticosteroids and PVL, the trial judge was able to draw logical inferences from the recognized effects of antenatal corticosteroids in humans and in animals.

The trial judge's findings were upheld on appeal. The dissenting judge ultimately concluded that causation was not proven in this case but did not dissent on the legal question of whether the evidence had to reach a level of statistical significance before it could prove causation on a balance of probabilities:

> "Scientific evidence revealing a trend suggestive of a causal connection between fact A and fact B is not discarded at trial because it does not reach the level of scientific proof. Instead, that evidence is considered along with any other evidence that is relevant to the question of causation."[11]

Where the dissenting judge differed from the majority was on the matter of quantification of the risk. The majority judgment held that it was not necessary for the court to have experimental evidence that quantified the reduction in the risk of PVL to prove causation on a balance of probabilities. The experts were able to use their clinical experience to fill in the gaps in the scientific evidence.

The Ontario Court of Appeal in this case specifically recognized the complexities of proving causation in this and other medical malpractice cases where, not uncommonly, there are no specific scientific studies that are determinative of the issues before the court. The courts analyzed the evidence, which did not reach a level of scientific certainty, considered the opinions of experts able to supplement the studies with their clinical experience and used logical inferences to find proof of causation. *Goodman v Viljoen* provides a helpful and important illustration of the legal principles of causation. It highlights the differences between medical and legal proof and offers an example of how common sense can be used to infer causation in the appropriate circumstances. V

1 *Clements v Clements*, 2012 SCC 32, [2012] 2 SCR 181 at para 8 [*Clements*]
2 *Clements, supra* note 1 at para 9
3 *Snell v Farrell*, 1990 CanLII 70 (SCC), [1990] 2 SCR 311 at para 33 [*Snell*]
4 *Clements, supra* note 1 at para 9
5 *Athey v Leonati,* 1996 CanLII 183 (SCC), (1996), 140 DLR (4th) 235 at para 16
6 *Athey, supra* note 5 at para 16; *Clements, supra* note 1 at paras 10, 11
7 *Snell, supra* note 3 at para 34
8 2012 ONCA 896, [2012] OJ No 6332
9 *Goodman, supra* note 8 at paras 17, 19, 20
10 *Goodman, supra* note 8 at paras 51, 53
11 *Goodman, supra* note 8 at para 85