Trends in Shoulder Dystocia Cases
Non-negligent Causes of Brachial Plexus Palsy

By Barbara A. MacFarlane[i]

Introduction

Shoulder dystocia during delivery is a medical emergency which can result in permanent injury of the baby, including neonatal brachial plexus palsy ("NBPP"), hypoxic ischemic encephalopathy ("HIE") or even death. Previously, it was widely accepted that NBPP injuries were caused by excessive traction by the clinician in delivering the baby, such that standard medical techniques are now used by the physician to avoid injury to the brachial plexus. However, over the last decade, in particular, there has been an increase in the publication of medical literature supporting other causes of NBPP. Some reports suggest that NBPP can occur in the absence of defined shoulder dystocia or following cesarean section. The literature theorizes that the natural forces of labour and maternal expulsion could be responsible for NBPP [Gonick; [1]Sadmire and DeMott[2]], suggesting inevitable injury.

Dr. Henry Lerner, in presenting a case study of one patient who suffered persistent NBPP (Erb's palsy) in the absence of clinician force, suggested that further research was needed to answer: (1) how much force is, on average, required to damage a live infant's brachial plexus; (2) how much force physicians generally use; and (3) whether it is possible to safely deliver shoulder dystocia babies without exceeding the force that may damage the brachial plexus. Respectfully, it would seem an impossible task to conduct a prospective study to answer any of these questions. Regardless of the answers, this theory about inevitable injury has been and will continue to be used by doctors to defend their conduct in NBPP litigation.

In some cases, Judges have been persuaded by the above theory and they have dismissed plaintiffs' claims. For example, in the 2008 decision of *Nessler v. Colliton*, Madam Justice D.A. Sulyma (referencing 1994 medical literature from Baskett and Allen, relied on by the defence expert) found that such an injury may have already occurred by the time the fetal head exits the birth canal and therefore before the clinician touches the head. She reviewed the opinion of the defence expert that[3]: "Considerable indirect evidence favours maternal forces as the most likely cause of Erb's palsy. That evidence includes:

(1) A rapid second stage in 30-40% of Erb's palsy cases;
(2) The absence of associated shoulder dystocia in approximately 50%;
(3) The fact that the posterior arm is the effected on in 33-39%;
(4) the fact that the frequency of the injury is independent of the experience of the clinician."

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[1] Gonik B, Walker A, Grimm M. Mathematic modeling of forces associated with shoulder dystocia: A comparison of endogenous and exogenous sources. Am J Obstet Gynecol 2000;182;689-91.
[2] Sandmire HF, DeMott RK. Erb palsy: Concepts of causation. Obstet Gynecol 2000;95: 940-2.
[3] 2008 ABQB 180 (CanLii), paras. 98-99

In an Ontario case involving a severe BPI (including a nerve avulsion at C-8), Madam Justice Dunnet, found that the injury was consistent with an in utero traction or appropriate traction by the physician on delivery (as there was no evidence to the contrary).[4]

Recently, the American College of Obstetricians and Gynecologists Neonatal Brachial Plexus Palsy Task Force ("ACOG NBPP Task Force") conducted an extensive literature review and drew conclusions from same. A read of the report of the Task Force, suggests a desire to advance these non-negligent causation theories for NBPP. The authors state[5]:

> ...the same biomechanical factors that predispose a fetus to develop NBPP (forces of labor, in utero malpositioning, or failure of truncal rotation) also may predispose a fetus to shoulder dystocia. The presence of both recognized shoulder dystocia and NBPP can lead to an erroneous restrospective inference of causation.
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> Clinician-applied traction and lateral bending of the fetal neck have been implicated as causative factors in some cases of NBPP. However, NBPP also has been shown to occur entirely unrelated to traction, with studies demonstrating cases of both transient and persistent NBPP in fetuses delivered vaginally without clinically evident shoulder dystocia or fetuses delivered by caesarean without shoulder dystocia.

The ACOG NBPP Task Force reported on a summary of 12 reports from 6 countries and concluded that in 46% (0.9/1000 births) of cases NBPP can be seen without documented shoulder dystocia and 26% of those had persistent (>12 months) NBPP. Additionally, they cite the incidence of NBPP ranging from 0.3 to 0.8/1,000 after caesarean section.[6] Relying on these reports, the Task Force suggests that this supports the proposition that clinician applied force is not the only cause of NBPP, even where there is a persistent injury. It should be noted, however, that there were only 2 reports of a persistent injury without documented shoulder dystocia. Furthermore, the reports relating to c-section births do not disclose whether trauma occurred as a result of uterine incision or whether the injury was a transient or persistent one.

The purpose of this paper is to bring attention to an apparent growing trend by the defence bar (with the aid of medical literature) to point to non-negligent reasons for NBPP injuries in shoulder dystocia cases. The defence generally advances the position that shoulder dystocia is a rare medical emergency where, despite best efforts of the physicians, the injuries cannot be avoided. After all they are trying to save a baby's life. In some cases, the Judiciary has already acknowledged the challenge of shoulder dystocia for physicians and accepted "non-negligent" causes for NBPP, even where the injuries are severe. The ACOG NBPP Task Force paper will no doubt be used to advance the "rare" and "non-negligent" defences further. A careful review of the medical literature cited, however, raises questions as to the validity of the conclusions being made by ACOG. For example, it is hard to reconcile how anything other than

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[4] Brown (LG) v Sarraf [1998] O.J. No. 3746
[5] ACOG NBPP Task Force, Chapter 2, p.17
[6] ACOG NBPP Task Force, Chapter 1, p.2

excessive traction on the baby's brachial plexus during a shoulder dystocia delivery could cause severe NBPP injuries (such as ruptures or avulsion of the nerves).

Definition of Shoulder Dystocia

Shoulder dystocia occurs when the baby's anterior shoulder gets stuck behind the mother's pubic bone after the head is delivered, halting the delivery. Historically, the common definition of shoulder dystocia is where the clinician is required to use manoeuvers to deliver the shoulders following a failure to deliver with gentle downward traction.[7] However, there is inherent subjectivity of this clinical definition of shoulder dystocia, dependant on the classification and reporting by the clinician and, perhaps, differences in the study populations used.

In Cunningham et al. Williams Text on Obstetrics, the authors state: "Use of manoeuvres to define shoulder dystocia has been criticized (Beall and associated, 1998; Song and colleagues, 1995). In deliveries in which shoulder dystocia is anticipated, one or more manoeuvres may be used prophylactically, but no diagnosis of shoulder dystocia is recorded. In other cases, one or two manoeuvres may be used with rapid resolution of shoulder dystocia and excellent outcome, and the diagnosis is not identified. Spong and colleagues (1995) attempted to more objectively define shoulder dystocia by witnessing 250 unselected deliveries and timing intervals from delivery of the head, to delivery of the shoulders, and to completion of the birth. The incidence defined by the use of obstetrical manoeuvres was higher than previously reported (11 percent); however, only about half of these were diagnosed by the clinicians. The mean head-to body delivery time in normal births was 24 seconds compared with 79 seconds in those with shoulder dystocia. They proposed that a head-to-body delivery time exceeding 60 seconds be used to define shoulder dystocia."[8]

The ACOG NBPP Task Force acknowledged the studies that suggest a head-to-body interval of >60 seconds or ancillary manoeuvers, or both are more appropriate to define shoulder dystocia. There is a suggestion by the Task Force that adding the >60 seconds interval widens the definition to increase the incidence of shoulder dystocia to about 10% from 0.2-3%.[9] However, the NBPP Task Force made no recommendations to change the aforementioned definition, and prefers to stick with a definition which may include a number of cases where shoulder dystocia did not truly exist (e.g., manoeuvers to enlarge the pelvic outlet were used prophylactically absent a delayed delivery). The "manoeuver definition" arguably, skews the ratio of NBPP in true shoulder dystocia cases.

"There is some evidence that the incidence of shoulder dystocia increased from 1960 to 1980 (Hopwood, 1982). This is likely due to increasing birth weight...It is also likely that the increased incidence of shoulder dystocia is due in part to increased attention to its appropriate documentation (Nocon and co-workers, 1993)"[10] [e.g., reporting by

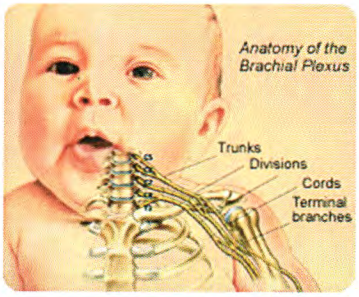
[7] ACOG NBPP Task Force, Chapter 4, p.42
[8] Cunningham et al. Williams Obstetrics. Chapter 19: Dystocia. 21st Edition. 2001
[9] ACOG NBPP Task Force, Chapter 4, p.42
[10] Cunningham et al. Williams Obstetrics. Chapter 19: Dystocia. 21st Edition. 2001

clinicians]. Despite acknowledging an increase in incidence of shoulder dystocia, the ACOG NBPP Task Force suggest that, the incidence of NBPP injuries have remain constant.[11] One could argue that clinician education and training would be the logical explanation for essentially reducing the relative incidence of NBPP injuries. If natural forces of labour or maternal forces were significant causes of NBPP injuries, one might expect the increase in shoulder dystocia cases to be accompanied by a proportionate increase in the absolute number of NBPP injuries.

"Shoulder dystocia may be associated with significant fetal morbidity and even mortality. Gherman and co-workers (1998) reviewed 285 cases of shoulder dystocia and 25 percent were associated with fetal injuries. Transient brachial plexus palsies were the most common injury, accounting for two thirds; 38 percent had clavicular fractures, and 17 percent sustained humeral fractures. There was one neonatal death, and four infants had persistent brachial plexus injuries."[12] Shoulder impaction and prolonged head-to-body delivery interval can lead to birth asphyxia and subsequent neonatal hypoxic-ischemic encephalopathy. There is a risk of acidosis or severe HIE where there is >5 minutes of impaction.[13]

Mechanics of NBPP Injuries

An understanding of NBPP injuries is important to the understanding of causation. The brachial plexus is a network of nerves leading from C-5, C-6, C7, C-8 and T-1 of the spine, through the neck, the axilla (armpit) and into the arm and operates the upper arm, forearm and hand. It is divided into nerve roots, trunks, divisions, cords and branches. Damage to the brachial plexus nerve system responsible for sending signals from the spine to the shoulder, arm or hand can result in severe functional impairment and deformity in a child.



Nerve injury may be caused by either compression or traction of the nerve. Compression can damage the epineurium (the outer sheath of the nerve structure) and axonal tissue. There can be a permanent injury if the compression is maintained for a sufficient period and is of significant magnitude. A traction injury can result in a rupture or avulsion of the nerve if the shoulder is stuck and the head and spine continue to move. "The clinical and biomedical engineering evidence supports the assertion that when a shoulder is restrained either transiently or during a more significant impaction,

[11] ACOG NBPP Task Force, Chapter
[12] Cunningham et al. Williams Obstetrics. Chapter 19: Dystocia. 21st Edition. 2001
[13] ACOG NBPP Task Force, p. 47

both maternal forces and clinician forces, if applied, will stretch the brachial plexus".[14] A rupture is a tear of the spinal nerve root at a point distal to the vertebral foramen [essentially tearing the nerve]. An avulsion is a pulling of spinal nerve root away from the spinal cord, within or proximal to the vertebral foramen [essentially severing the nerve in two]. A lesser injury may be a lesion of the nerve.

Because there is less connective tissue binding the lower nerves (C-8 and T-1) they "are prone to preganglionic injury (avulsion), whereas the nerves comprising the upper trunk tend to sustain post ganglionic injury (rupture). A preganglionic injury results in permanent paralysis of the muscles innervated by the avulsed roots and complete sensory loss of the corresponding dermatomes. Spontaneous nerve regeneration is unlikely. A postganglionic injury allows potential retention of function of the cell body within the ventral horn of the spinal cord, and these neurons may regenerate axons under appropriate conditions."[15]

The ACOG Task Force identifies four groups of nerve injuries in NBPP, including injuries to:[16]

1. C-5 to C-6 (with retained function in elbow, wrist and hand);

2. C-5, C-6 and C-7 (paresis of deltoid, biceps and triceps) but intrinsic muscle of hand being unaffected);

3. C-5, C-6, C-7, C-8 and T-1 (paresis of entire arm) with an unlikelihood of spontaneous recovery [see ACOG NBPP Task Force p. 66]

4. Paresis of entire arm and Horner's syndrome of the ipsilateral eye, implying an injury to all of the nerve roots of the brachial plexus with a proximal injury to the lower nerve roots. This level of injury has little chance for full spontaneous recovery [see ACOG NBPP Task Force p. 66]

An injury to the upper nerve roots (C-5 to C-6 and sometimes C-7) are the most common NBPP cases and are "commonly referred to as Erb's palsy, after William Erb who in 1877 was the first to recognize, diagnose and study brachial plexus injuries. Erb's studies helped determine that these injuries resulted from downward traction to the spinal nerves in the cervical area. It consists of a paralysis of shoulder and arm muscles resulting in a hanging upper arm that may be extended at the elbow."[17]

Involvement of the lower spinal nerves C7 to T1 always includes injury of the upper nerves and results in a palsy including the hand, which can cause a claw-hand deformity. This injury is known as Klumpke's palsy.

Interestingly, the incidence of NBPP reported by the ACOG NBPP Task Force noted that injury occurs every 1.5 per 1,000 total births in USA (1.3 per 1,000 in other countries), stating that the number has varied little in past 20 years (with 1.2 in 1990).

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[14] ACOG NBPP Task Force, p. 34-35
[15] ACOG NBPP Task Force, p. 54-55
[16] ACOG NBPP Task Force, p. 56
[17] Cunningham et al. Williams Obstetrics. Chapter 19: Dystocia. 21st Edition. 2001

These injuries included both transient and persistent (>12 months) conditions. Erb's Palsy represented 1.2/1,000 births and Klumpke palsy were rarest with 0.05/1000 births.[18]

Causation:

Dr. Joseph H. Piatt in his publication, "Birth Injuries of the Brachial Plexus" states: "The mechanisms responsible for brachial plexus injury have been disputed for more than 100 years and modern medicolegal pressures have further roiled these disputations."[19]

The ACOG NBPP Task Force sought to determine the potential mechanics and, therefore, causes of NBPP. A 1979 study by Metaizeau where 9 infant cadavers were tested showed that "[a]s lateral traction was applied, the upper plexus was the first portion of the complex to be damaged – generally resulting in nerve root rupture. With continued lateral loading following C-5-C-6 rupture, avulsion of the C-7 and C-8 roots occurred".[20] This supports the proposition that as more force is applied more significant damage can occur (e.g., avulsion of the nerve root in the lower part of the spine). The Task Force authors call these tests "quite crude by today's standards of biomechanics" but admit that they "lend insight into one of the injury mechanisms of the brachial plexus – the application of lateral bending". They go on, however, to qualify it by stating that the studies "do no provide a complete picture of how and why NBPP may occur during delivery" and it is "inappropriate to conclude that lateral bending is the only cause of the injury..."[21]

The ACOG NBPP Task Force seem to prefer computer models to build a virtual representation of the birth process. They refer to the MADYMO computer model developed to investigate both endogenous [labour and maternal] and exogenous [clinician applied] delivery forces: "...the predicted forces required to achieve delivery were 400N for maternally generated (endogenous) forces and 100N for clinician-applied (exogenous) forces ... [The] contact force at the base of the fetal neck against the maternal symphysis pubis was more than two times higher because of maternal endogenous forces when compared with exogenous forces". [22] It noted that the McRoberts positioning reduced the exogenous force by 50% but did not comment on endogenous force reduction. However, mathematical computer models have been criticized for failing to account for soft tissue resistance, the dissipation of force throughout the uterus or the additive effect of traction and compression forces. This model has received further criticism on the gross assumptions made for the impaction site, the parameters defining the endogenous force distribution, and the wide range of contact pressures between the fetal neck and the symphysis pubis, which includes values that in real life exceed the fatal limits.[23] Moreover, these studies have not

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[18] ACOG NBPP Task Force, p. 1

[19] Piatt JH. Birth Injuries of the Brachial Plexus. Clinics in Perinatology 32:39-59, 2005.

[20] ACOG NBPP Task Force Executive Summary, p. xv

[21] supra

[22] supra

[23] see Gonik et al.: *Mathematical modeling of forces associated with shoulder dystocia: a comparison of endogenous and exogenous sources*, Am J Obstet Gynecol 182:689-691, 2000; and

determined the extent of the NBPP injuries that would be sustained by the force and do not measure the force required to result in an avulsion of the nerve root.

Although the Task Force concluded that forces other than clinician interaction could cause NBPP injuries, it was not able to conclude much about the amount of force necessary to result in a persistent injury or, more importantly, an avulsion injury citing that "because of nonlinear behavior of tissues such as nerve tissue, an estimate of the force needed to cause a nerve rupture cannot be directly established."[24] Instead, it simply focused on factors other than physician error as a potential cause of NBPP. For example, it stated:

> The pediatric neurological community also has reviewed the literature on causation and has similarly concluded that, "The obstetrician's efforts to relieve shoulder dystocia are not the whole explanation for brachial plexus birth injuries. Expulsive forces (ie, endogenous forces) generated by the uterus and the abdominal wall ... may be contributory in many cases."

In Summary, the Task Force concludes:

> Neither high-quality nor consistent data exist to suggest that NBPP can be caused only by a specific amount of applied force beyond that typically used by health care providers and experienced during a delivery without NBPP. Instead...NBPP is a complex event, dependant...on the constellation of forces...that have been acting on the fetus during the labor and delivery process, as well as individual fetal tissue characteristics..."[25]

H. Gordon, Editor of the Journal of Obstetrics and Gynaecology in 2008 published an editorial in which he states: "While it must be accepted that some cases of brachial plexus trauma do involve excessive force (especially with lateral traction and lateral flexion of the head), it is reasonable to suspect that BPI (brachial plexus injury) is due to the normal forces of labour where there has been prolonged labour, persistent occipitoposterior position, instrumental delivery or maternal diabetes. Damage to the posterior shoulder is unlikely to be caused by excess force and strongly suggests that it is the result of forces of labour."[26]

As such, one might say that in cases where there was no uterine malformation, no prolonged labour; there is no medical record of occiput position; there was no instrumental delivery and there was no maternal diabetes, the anterior arm is affected and not the posterior arm, it would be difficult to attribute an alternative mechanism for brachial plexus injury. Furthermore, the medical literature does nothing more than speculate that other causes may be responsible for persistent NBPP injuries. It could be argued that there is no evidence that severe injuries (such as nerve rupture or avulsion injuries, the rare Klumpke's palsy or HIE) could be caused by natural forces of labour or maternal expulsion forces.

<u>Management: Standard of Care</u>

Doumouchtsis et al.: *Are All Brachial Plexus Injuries Caused by Shoulder Dystocia?* CME Review Article, Obstetrical and Gynecological Survey. Volume 64. Number 9, pages 615-623, 2009.

[24]ACOG NBPP Task Force, p. 35

[25]ACOG NBPP Task Force, p. 37

[26]Gordon H. Shoulder Dystocia. Journal of Obstetrics and Gynaecology, May 2008;28(4):371-372.

In most shoulder dystocia litigation there is an element of credibility. Often the clinician or nurses do not fully document the details of the delivery and yet provide a "text-book" explanation during an examination for discovery. Limited documentation leaves little, if any room, to permit an expert to give a prospective review on the medical record. It is useful, however, to understand what the literature suggests is the standard of care in the circumstances of shoulder dystocia.

It is acknowledged that shoulder dystocia is a rare medical emergency and can be a frightening experience for both mother and clinician. It is for that reason that the physician must remain calm and take control to effectively communicate the plan to the nursing staff to ensure safe delivery of the baby. "A pragmatic approach in all deliveries involving shoulder dystocia would be to minimize delay in delivery as much as possible by implementing appropriate manoeuvers once shoulder dystocia is recognized." [27]

Management of Shoulder Dystocia should be a collaborative team effort and it is "important for the delivering clinician to give instructions to the nursing staff regarding the performance of manoeuvers such as McRoberts positioning, the application of suprapubic pressure, and the need for a resuscitation team. The woman in labor should be instructed to refrain from pushing during an attempted manoeuver. She can then be instructed to resume pushing following performance of a manoeuver to allow determination of whether the shoulder dystocia has been successfully relieved".[28]

A variety of techniques have been described to free the anterior shoulder from its impacted position beneath the maternal symphysis pubis: [29]

1. Moderate suprapubic pressure is applied by an assistant while gentle downward traction is applied to the fetal head.

2. The McRoberts manoeuvre, named for William A. McRoberts, Jr., who popularized its use at the University of Texas at Houston. The manoeuver consists of removing the legs from stirrups and bringing them up to the abdomen. It results in a straightening of the sacrum relative to the lumbar vertebrae, along with accompanying rotation of the symphysis pubis toward the maternal head and a decrease in the angle of pelvic inclination and is found to reduce fetal extraction forces.

3. Rotating the posterior shoulder 180 degrees in a corkscrew fashion, the impacted anterior shoulder could be released. This is frequently referred to as the Woods corkscrew manoeuvre.

4. Delivery of the posterior shoulder by carefully sweeping the posterior arm of the fetus across the chest, followed by delivery of the arm. The shoulder girdle is then rotated into one of the oblique diameters of the pelvis with subsequent delivery of the anterior shoulder.

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[27] ACOG NBPP Task Force, p. 47
[28] ACOG NBPP Task Force, p. 43
[29] Cunningham et al. Williams Obstetrics. Chapter 19: Dystocia. 21st Edition. 2001.

Studies have shown that the following combination of manoeuvers is associated with the highest delivery rate (84.4%) and a 71% decrease in anterior nerve stretch and 80% decrease in delivery force[30]: (1) McRoberts positioning; (2) applying subrapubic pressure; (3) delivery of the posterior arm. The ACOG NBPP Task Force goes on to state that "[a]lthough no specific sequence of manoeuvers has been shown to be superior, a standardized sequence of manoeuvers may be valuable within a given institution, as with any medical emergency requiring coordination among multiple health care providers".[31] There is a suggestion by the Task Force that hospitals develop policies and procedures for the proper management of babies with shoulder dystocia. Additionally, they encourage training and proper documentation and recommend the use of a checklist for to assist with early recognition, management and documentation.[32] It stresses the need for contemporaneous documentation of the management of shoulder dystocia to record significant facts, findings, and observations about the shoulder dystocia event and its sequelae. The medical record should include a careful accounting of the events leading up to delivery of the infant whose course was complicated by shoulder dystocia.[33]

Identifying potential risk factors for shoulder dystocia, including (among other things) macrosomia or prior history of shoulder dystocia could include prophylactic strategies. For example, early induction of labour, McRoberts positioning, suprapubic pressure, etc. However, the ACOG NBPP Task Force suggest that these strategies have not been proven to affect the incidence of NBPP.[34] The ACOG Practice Bulletin Number 40, *Shoulder Dystocia*, states "Planned caesarean delivery to prevent shoulder dystocia may be considered for suspected fetal macrosomia with estimated fetal weight exceeding 5,000 g in women without diabetes and 4,500 g in women with diabetes".

Conclusion:

With the publication of the ACOG NBPP Task Force findings, we can anticipate that defence counsel in medical malpractice litigation will vigorously advance non-negligent causes for NBPP injuries. The trend that we have seen in the Courts accepting this proposition may continue to grow. There are, however, significant flaws in the medical literature in support of this position. It may be that NBPP injuries that are less severe (such as transient nerve injuries that resolve or persistent injuries with minimal functional impairment) may be the result of non-negligent causes inherent in the birth process. However, there is little, if any, support that HIE or ruptured or avulsed nerve injuries could be caused by anything other than prolonged impaction or excessive force by the clinician. For example, there is just no scientific literature that confirms that a baby in utero can have nerves ripped out of the spine due to natural forces or maternal expulsion forces.

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[30] ACOG NBPP Task Force, p. 45
[31] ACOG NBPP Task Force, p. 45
[32] ACOG NBPP Task Force, p. 47 and Appendix C: ACOG Patient Safety Checklist, *Documenting Shoulder Dystocia*
[33] ACOG NBPP Task Force, p. 55
[34] ACOG NBPP Task Force, p. 43

Moreover, shoulder dystocia may not be as rare as the defence would suggest and proper protocols when faced with the emergency appears to lead to better outcomes.

One might argue that in the presence of shoulder dystocia with a severe NBPP there should be an inference of negligence such that the defence would be required to prove a non-negligent reason for the outcome.[35] The explanation would have to be more than mere speculation without evidentiary support.[36] I would submit that the medical literature is nothing more than speculation and ought not to displace the defence onus.

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[35] see for example, *Supreme Court of Canada in Fontaine v. British Columbia*, [1998] 1 S.C.R. 424

[36] see for example, *Hassen v. Anvari*, [2003] O.J. No. 3543; 2003, CanLII 1005 (OCA)

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