COURT FILE NO.: 29440/98

ONTARIO

SUPERIOR COURT OF JUSTICE

B E T W E E N:	)	
	)	
ANNE LOUISE MILNE, ALBERT ROLLAND GIBSON, JESSY MILNE by his Litigation Guardian, Albert Rolland Gibson, ALBIE JR. JOHN GIBSON, by his Litigation Guardian, Albert Rolland Gibson, JOAN MARCILLA GIBSON, GEORGE JOHN GIBSON and PATRICIA CAROLL BARNES	)	Richard Halpern, Aleks Mladenovic & Desmond H. Dixon, for the plaintiffs
Plaintiffs	)	
- and -	)	
ST. JOSEPH'S HEALTH CENTRE, DR. JOSEPH BARRY MACMILLAN, NURSE JANE DOE, DR. ROBERT BARRY GAMBRILL, DR. ROBERT GAGNON, DR. FELICE DEBBIE	)	Deborah Berlach for the defendants, St. Joseph's Health Centre and Nurse K. Planques
LACKMAN, DR. RENATO NATALE, DR. F. POTESTIO, DR. BRYAN STANLEY RICHARDSON, NURSE K. PLANQUES, NURSE JANE DOE, NURSE JANE DOE, DR. JOHN DOE, and DR. JOHN DOE	)	David I. W. Hamer, Christine L. Lonsdale and Anna Matas for the defendant, Dr. Felice Lackman
Defendants	)	
	)	**HEARD:** May 11, 12, 13, 15, 19, 20, 21, 25, 26, 27, 28, 29, June 01, 02, 03, 04, 08, 10, 16 & 22, 2009

Morissette J.:

BACKGROUND:

[1] Jessy Gibson should have been born a healthy newborn. As a result of the negligence in the management of both his mother's care and his own as a fetus on August 18th, 1997, Jessy suffered catastrophic brain damage from oxygen deprivation. As a result of this negligence, Jessy suffers from hypoxic ischemic encephalopathy which has caused a neuromuscular disorder called cerebral palsy.

[2] He is dependent on others for his care 24 hours a day. He is unable to communicate, cannot feed himself and is unable to ambulate. Jessy will never work or live independently. He has a condition known as spastic diplegia, which means that his limbs and particularly his lower limbs are spastic and cannot be well controlled. Jessy also has visual impairments as a result of his brain injury.

[3] The junior obstetrical resident at the time of Ms. Milne's admission at triage, Dr. Felice Lackman, has already admitted liability for breaching the standard of care by not communicating with Dr. Gagnon, the obstetrician on call, after conducting her assessment of Ms. Milne. As a result, a Perringer agreement was agreed to between the plaintiffs and the defendant Dr. Lackman, which resulted in the action against Dr. Lackman being dismissed. So too was the cross-claim by the hospital against the doctor, because the claim by the hospital against the doctor was not an independent cause of action, but rather a claim for indemnity arising from any joint and several liability.

[4] During the trial, the parties came to an agreement as to the quantum of damages.

[5] The issue is whether the triage nurse, Ms. Planques, has breached the relevant standard of care and if so, did it cause or contribute to the injury to Jessy. The essence of the plaintiffs' submission is that Nurse Planques had an independent obligation to ensure

that Dr. Gagnon was told of Ms. Milne's condition and to ensure that the fetus was continuously monitored.

[6] In other words, this Court is being asked to determine the several liability of the nurse. In doing so, this Court must consider how liability is to be apportioned between the doctor and the nurse with respect to their relative degree of responsibility.

THE PARTIES:

[7] Anne Milne and Albert Gibson are the parents of Jessy Gibson. Ms. Milne was born on October 29, 1975 and was 21 years old when Jessy was born. Jessy is their second child. Albie Jr. John Gibson (AJ) is Jessy's older brother. AJ was born on October 6, 1993. Ms. Milne's pregnancy with AJ was entirely normal.

[8] Nurse Planques is an obstetrical nurse at the hospital. She obtained her diploma in nursing from Fanshawe College in 1986. She began working full-time as a registered nurse in 1987. In 1989, Nurse Planques joined the staff at the hospital as a full-time registered nurse in obstetrics. She was working in the area of the hospital called the Family Birthing Centre. Nurse Planques worked full-time and continuously from 1989 to August 18, 1997 as an obstetric nurse. By 1991, her duties included providing care to mothers in labour, assuming the position of team leader from time-to-time and working in triage.

[9] Dr. Lackman was a second-year obstetrical resident on August 18, 1997. She graduated from medical school in 1996 and began her residency on July 1, 1996. The obstetrical residency program was a five-year program. In her first year of residency, Dr. Lackman had a three-month rotation in obstetrics/gynecology. The balance of her first year of training was spent in fields other than obstetrics and gynecology. Dr. Lackman's second year of residency began on July 1, 1997. During the month of July 1997, Dr. Lackman was training in gynecology, although she did have some involvement in labour

and delivery. Dr. Lackman began the obstetrics rotation of her second year of residency on August 1, 1997, less than three weeks before Jessy was born.

[10] The hospital is a tertiary care institution and a teaching hospital. It is the major tertiary care centre for all of southwestern Ontario. The hospital routinely sees high risk patients in obstetrics. At a teaching hospital, obstetrical care is provided by a team. The members of the obstetrical team include medical students, interns, junior residents, senior residents, obstetricians and nurses. In obstetrics the nurses outnumber the doctors in providing obstetrical care.

CHRONOLOGY OF AUGUST 18TH, 1997:

[11] The following are a summary of the chronology of events for Augusts 18th, 1997.

[12] At around 10:30 on August 18, 1997, the plaintiff Anne Milne, who was waiting in a motor vehicle for her husband, Albert Gibson and his parents outside the Ministry of Transportation, started to experience severe lower abdominal pain. Ms. Milne was approximately 33 weeks pregnant at the time.

[13] When Mr. Gibson and his parents returned to the car, Ms. Milne made them aware of the pain. The group returned to Mr. Gibson's parent's house, which was 10 to 15 minutes away.

[14] Ms. Milne went into the residence and laid down in her mother-in-law's room. After some time, Ms. Milne went to the washroom where she began to vomit. She and her husband decided that it was time to go to the hospital.

[15] Prior to leaving for the hospital, Mr. Gibson left a voice message with Ms. Milnes's family physician, Dr. Gambrill, advising him of Ms. Milne's pain and that they were going to the hospital.

[16] Up until this point, Ms. Milne's pregnancy had been relatively uneventful save for a hospital visit in June 1997 for hematuria (blood in her urine), which was ultimately diagnosed as a bladder infection.

[17] The plaintiffs arrived at the hospital and were redirected from the emergency department to the triage area in the labour & delivery unit. Ms. Milne was admitted to the hospital at 13:19.

[18] Nurse Planques assessed Ms. Milne by obtaining a history, conducted a brief examination and applied the electronic fetal monitor (EFM) at 13:28 to conduct a non-stress test (NST). Nurse Planques noted the following as a result of her history and examination:

i) Ms. Milne was 33 weeks and two days pregnant with an estimated date of confinement of October 4, 1997;

ii) The reason for Ms. Milne's visit to triage was abdominal pain, which started at 10:30. The pain was described as constant, varying in intensity and severe in the patient's back since she came to hospital; Ms. Milne had vomited due to pain at 12:30.

iii) The baby had a Symphysis Fundal Height of 32, which was consistent with the age of gestation;

iv) Ms. Milne was not experiencing uterine activity.

v) Ms. Milne's vital signs were within normal limits; and

vi) The fetal heart rate was 140 bpm (beats per minute), which was within normal limits.

[19] At approximately 13:40, Nurse Planques contacted Dr. Gambrill, Ms. Milne's family physician. Although Ms. Planques has no independent recollection of what information was conveyed to Dr. Gambrill during this call, her usual practice would have been to provide him with a history as to why Ms. Milne had presented in triage and to

provide both the maternal and fetal vital signs. She would also have indicated that an NST was in progress.

[20] Although there is no record of what was conveyed to Dr. Gambrill, we know that the communication was sufficient to raise a concern in Dr. Gambrill's mind that he requested an obstetrical consult. As noted by the plaintiffs' expert Dr. Davies, Dr. Gambrill likely made this request because the information conveyed to him by Nurse Planques allowed him to recognize that Ms. Milne's presenting problem was outside his scope of practice as a family doctor and required an obstetrical consult.

[21] Nurse Planques likely put Dr. Gambrill through to the switchboard which would have transferred his call to the obstetrician on-call, Dr. Gagnon. Nurse Planques gave evidence that the routine practice at the hospital in 1997 was that a family doctor who requested an obstetrical consult would speak to the obstetrician on-call, who would, in turn, speak to the resident and request the resident go and see the patient. As result of this request, the second-year obstetrical resident – the defendant Dr. Felice Lackman – attended in triage to assess Ms. Milne.

[22] Dr. Gambrill`s evidence was that he spoke to either a resident or an obstetrician in obstetrics.[1]

[23] Dr. Gagnon's evidence was that, if called to attend on a triage patient, he was available to attend the patient "within 10 minutes."[2]

[24] While Nurse Planques waited for the obstetrical consult to arrive, the NST continued to run until it was disconnected at approximately 13:55 to allow Ms. Milne to go to the bathroom. At the time the NST was disconnected there was a 27-minute

[1] Exhibit 4.
[2] Exhibit 5.

tracing, which was non-reactive.[3] The fetal heart rate had a baseline of 130-135 bpm, there was minimal variability and no accelerations or decelerations.

[25] Dr. Lackman arrived to assess Ms. Milne at 14:00, just as Ms. Milne was exiting the bathroom. Shortly after Dr. Lackman arrived, Ms. Milne vomited and she was assisted with that by Nurse Planques. The EFM was not reconnected until 14:53.

[26] The vast majority of the experts who testified said that in a teaching hospital, the attendance by a resident to assess this patient, even where Ms. Milne's presentation suggested a placental abruption, would not surprise a nurse. Indeed, Nurse Planques gave evidence that it was the usual practice at the hospital to have residents assess such patients.

[27] Dr. Lackman gave evidence that although she has no independent recollection of the day in question, her usual practice would have been that upon arriving to assess Ms. Milne she would have taken a thorough history, performed a physical assessment, which included palpating Ms. Milne's abdomen, listening for bowel sounds for one minute in each quadrant, and an internal examination. Dr. Lackman estimated the time for taking the history at about ten minutes and the physical assessment would have taken her eight to ten minutes. She then would have proceeded with a real time scan (RTS), which is an ultrasound test in order to obtain a biophysical profile score (BPP). The BPP measures four variables and allots a score of two to each if normal, or zero to each if abnormal. The four variables measured are fetal breathing movements, fetal movements, fetal tone and amniotic fluid volume. The test is then assigned a score out of a possible eight points. A RTS is usually conducted for 30 minutes. The RTS performed by Dr. Lackman lasted 20 minutes, because she stated in her late note: "conducted 20 minutes

[3] Reactivity of the fetal heart rate requires the presence of accelerations, or increases in the baseline of 15 bpm lasting 15 seconds. At least two of these accelerations must be seen in a 20 minute period in order for the observer to be reassured about fetal well-being. The absence of accelerations in a 20 minute tracing means that the tracing is "non-reactive" and potentially non-reassuring.

due to pt. intolerance". Dr. Lackman assigned a BPP profile score of 2 out of 8. That score is non-reassuring.

[28] Dr. Lackman completed her assessment of Ms. Milne and left her room to observe an elective Caesarean section ("C-section"). She left Nurse Planques with interim orders for continuous monitoring and blood work. The nurse carried out these orders as requested.

[29] At 14:53 or shortly after, Nurse Planques also spoke with Dr. Lackman about Ms. Milne's request for pain medication. Dr. Lackman declined to prescribe any pain medication.

[30] When the EFM was reattached at 14:53, the nurse was able to obtain a continuous tracing until 15:06 when Ms. Milne again got up to go to the bathroom. The monitor was disconnected to allow for that to happen.

[31] After Ms. Milne returned from the bathroom, a short tracing was obtained at 15:10, which was consistent with the tracing from 14:53-15:06, continuing to be non-reactive. Thereafter, there was difficulty obtaining a continuous tracing.

[32] At 15:18 the fetal heart rate fell below 110 and nurse Planques paged or called Dr. Lackman, who was still observing the C-section. Dr. Lackman told Nurse Planques to page Dr. Gagnon.

[33] Dr. Gagnon's evidence is that he received a call between 15:20 and 15:25, which is consistent with the time that Nurse Planques would have paged him in response to Dr. Lackman's request.

[34] By 15:28, a physician had still not attended to re-assess Ms. Milne. Nurse Planques took further action by corralling Dr. Natale, an obstetrician at St. Joseph's who just happened to be passing in the hallway, and asking him to assess Ms. Milne.

[35] Dr. Natale assessed Ms. Milne's abdomen and noted the fetal heart rate was in the 120 range but it was seemingly erratic. He put on the real time scan and noticed that the fetal heart rate was 80 to 100 bpm and there seemed to be a large blood clot behind the placenta. He then looked at the fetal heart rate again, which was now 60 to 70 bpm.

[36] Dr. Natale formed the impression that Ms. Milne had a severe abruption and decided to proceed with an emergency C-section. Ms. Milne arrived in the C-section room at 15:35. The procedure began at 15:40 and Jessy was delivered only three minutes later at 15:43, eight minutes from the time of the decision to proceed to C-section.

[37] Dr. Natale noted that the placenta delivered spontaneously and, in fact, followed Jessy immediately. Dr. Natale removed "1500 cc's of fresh blood, which was behind the placenta. It was contained within the membranes. The blood was still red, the impression being that it had been likely an abruption in evolution and, in fact, likely had just happened to the degree that it had happened."[4]

[38] After birth, Jessie's Apgar score was 0 out of 10. This test is designed to assess fetal status at birth which measures five criteria: heart rate, respiratory rate, tone, reflex responses and colour. Jessy had no heart rate at all and the resuscitation team began cardiac compressions to restore his heart rate. It was not until seven minutes of age that Jessy's heart rate was restored.

TIMING OF THE REAL TIME SCAN:

[39] The timing of the RTS has become an issue in this trial. Interestingly, the defence takes the position that the timing of the RTS is a critical issue on both the standard of care and causation. On the other hand, the plaintiffs' position, as stated by Mr. Halpern: "it's neither here nor there". Even though the plaintiffs contend that the RTS was commenced at 14:12 and completed at 14:32, they say that even if the RTS was

completed at 14:50 as the defence suggests, it does not protect the defence from an adverse finding on the standard of care by Nurse Planques or causation. I will say more about that later.

The Evidence and Findings of Fact of the timing of the RTS:

[40] The following is my review of the evidence about this issue of timing and my findings of fact.

[41] Neither Nurse Planques nor Dr. Lackman have maintained adequately detailed notes and there are few contemporaneous notes to be found in the chart. There is, however, a contemporaneous record to be found on the fetal heart tracing.[5]

[42] As conceded by Nurse Planques, the fetal heart rate tracing operates as a "time stamp" in the sense that it is easy and convenient for the nurse to write directly on the tracing paper, as it is fed through the machine, events that occur at the time indicated on the tracing paper. For example, Nurse Planques noted on the tracing at 13:55 hours, panel 66831, that Ms. Milne was up to the bathroom. This explains why the ink tracing stops at precisely that moment. It should also be noted that the tracing paper continues to run through the machine as the time continues to be stamped on the paper every 10 minutes. All experts agree that the reason why the machine was left running was because it was important to re-attach the EFM to Ms. Milne just as soon as she returned from the bathroom in order to obtain more information about fetal well-being. At least at that point, there was no reason to turn off the EFM.

[43] There are other contemporaneous notes on the fetal heart tracing. At 14:03 hours, panel 66833, the patient vomited. At 15:07 hours, panel 66840, Ms. Milne was up to the bathroom again. At 15:32 hours, panel 66847, the ink tracing was stopped to allow

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[4] Exhibit 1, Vol. 1, p.21.
[5] Exhibit 2.

Dr. Natale to do a RTS. A minute later, at 15:33 hours, the patient was being wheeled into Case Room #5 for an emergent C-section.

[44] The evidence of Dr. Lackman is that she likely came to assess Ms. Milne at 14:00. At some point after 14:00 and likely after Ms. Milne finished vomiting, Dr. Lackman testified that she likely took a ten-minute history and then performed an eight to ten minute physical examination before beginning the RTS. Dr. Lackman's evidence, if accepted, would suggest the RTS began between 14:20 and 14:30. The plaintiffs have sought to call Dr. Lackman's evidence into question in two primary ways:

> 1) First, the plaintiffs have had the experts who they retained opine that the length of time taken seems too long or is inconsistent with their own experience; and
>
> 2) Second, the plaintiffs have sought to establish that a note made by Nurse Planques on the electronic fetal monitor tracing – "U/s Dr. Lackman" (U/s ultra sound) – was contemporaneously made at 14:12 and therefore indicates that Dr. Lackman started the RTS at that point.

[45] The plaintiffs bear the burden of establishing that the preponderance of the evidence supports their theory that Dr. Lackman completed her assessment of Ms. Milne by approximately 14:32.

[46] For the following reasons, the preponderance of the evidence supports the conclusion that Dr. Lackman did begin the RTS between 14:25 and 14:30.

[47] First, the only evidence on timing from any fact witnesses is that of Dr. Lackman. She did not waiver in her evidence as to the time she would have taken to complete a history.

[48] Dr. Davies, an obstetrician with considerable experience teaching residents, testified that the entire process, in the hands of a resident, could not have taken more than about ten minutes. In all of his considerable experience teaching residents, Dr. Davies

testified that he had never seen a resident take four minutes to listen to bowel sounds, as Dr. Lackman testified. In his opinion, it would only take 30 seconds. Dr. Braithwaite, an obstetrician, gave similar evidence.

[49] The experience of the plaintiffs' experts is not determinative. While they may have observed other residents over the years, they never observed Dr. Lackman as a resident take a history or conduct a physical examination. In addition, while Dr. Davies stated that he thought that it took too long, he also confirmed that taking ten minutes to complete a history and eight to ten minutes to complete a physical exam, including the vaginal examination, was not a breach of the standard of care.

[50] In any event, the evidence of the plaintiffs' experts is countered by that of the defendants' experts, who both testified that it could take a resident ten minutes to take a history and eight to ten minutes to perform a physical assessment.

[51] In addition, there is confirmatory evidence in the record which supports the conclusion that Dr. Lackman concluded her assessment of Ms. Milne at or around 14:45 to 14:50. The uncontroverted evidence is that at some time after the completion of Dr. Lackman's assessment of Ms. Milne she gave Nurse Planques two orders: continuous monitoring of the fetal heart rate and blood work. The evidence clearly shows that the EFM was restarted at 14:53 and Ms. Milne's blood work from triage was entered into the computer at 14:58, which supports an inference that the blood was drawn close to that time.

[52] Furthermore, Dr. Lackman clearly indicated her recollection that she left Ms. Milne to attend a C-section. She gave evidence that the junior resident would enter the operating room for a C-section with or soon after the patient does. The records from the C-section that Dr. Lackman attended – identified as the records of Patient "X"[6] provide

[6] Exhibit 1, Vol. 1, Tab 3.

objective evidence that Patient "X" entered the operating room at 14:50. This also corroborates the evidence that the RTS was likely completed between 14:45 and 14:50.

[53] The handwritten note – "U/s Dr. Lackman" – on which the plaintiffs rely does not lead to the conclusion on a balance of probabilities that notation was contemporaneous as to the RTS beginning at 14:12. Importantly, the plaintiffs' own expert – Nurse Doren – demonstrated the ambiguous nature of this note. Nurse Doren, when directed to anticipate the evidence of Dr. Lackman that the ultrasound was completed at or about 14:50, conceded that the note on the strip was consistent with that time. Likewise, Nurse Trépanier also gave evidence that the "u/s Dr. Lackman" note is ambiguous. Specifically, she noted that given that the tracing had been turned off sometime between 14:10 and 14:20, one could not tell one way or the other when the note was made.

[54] The plaintiffs argue that the note must suggest that the RTS began at 14:12 because this is consistent with Nurse Planques' practice on the rest of the strip of making notes contemporaneously. However and more importantly, that argument ignores that the "U/s Dr. Lackman" note is the only note on the strip that is made while the strip is stopped as opposed to while the strip is running. Nurse Planques' evidence is that the EFM was not reapplied while Dr. Lackman was performing her history and physical and that, in accordance with Nurse Planques' usual practice, she would have been in and out of the triage room while Dr. Lackman was attending to the patient. Accordingly, it is reasonable to conclude that Nurse Planques may not have been in the room when the ultrasound began and accordingly, her note was not contemporaneous.

[55] All of the experts who commented on the timing of the RTS opined that it occurred at 14:12. Even Nurse Trépanier, a defence expert witness, indicated so in her initial report, notwithstanding the fact that she subsequently claimed to be unsure of the timing. Nurse Trépanier's change of heart was expressed for the first time only when she

gave evidence at trial. Although her evidence about her change of mind did appear to be contrived, that change of mind does show the ambiguity about the note on the strip.

[56] Ms. Milne testified that the monitor was stopped for her to go to the bathroom and she was in the bathroom for only a couple of minutes. When she returned to the room Dr. Lackman appeared. Ms. Milne recalls vomiting after returning to bed and estimated that the RTS started about five minutes later. The plaintiff, Albert Gibson, recalls Dr. Lackman (who he described as an ultrasound technician) coming into the room with the ultrasound machine just before Ms. Milne returned from the bathroom. He recalled Dr. Lackman asking some questions and then starting the RTS within about five minutes.

[57] Both Ms. Milne and Mr. Gibson struck me as very honest witnesses. However, some aspects of their evidence is not reliable because it conflicts with the information contained in the medical records – an objective document.

[58] Mr. Gibson admitted that his estimates about timing were guesses and that they were not based on having looked at a clock.

[59] Equally, Dr. Lackman's estimates of time are not reliable as evidenced by her late note on that day (one hour after Jessy's birth), as having left Ms. Milne's room at 15:15, which she conceded that was not the time the note was in fact made.

[60] The plaintiffs argued that Nurse Planques' indication that she did not recall if she turned off the EFM in order for the RTS to start was inconsistent with her evidence on examination for discovery. The excerpt that plaintiffs' counsel used to try to impeach Nurse Planques was:

> 120. Q. Were you there at any point when Dr. Lackman was in the room with the patient?
>
> A. Yes, I would have been.

> 121. Q. Okay. And you're looking at the fetal heart rate tracing to aid you in answering that question. What does that tell you that assists you?
>
> A. I would have removed the monitor for the real time scan.

[61] The passage identified by plaintiffs' counsel merely indicates the reason that the monitor was removed. It says nothing as to when the monitor was turned off. At most, and as indicated by the author of the note, the note suggests that the RTS occurred sometime between 14:10 and 14:53.

[62] Given the ambiguity of the note on a stopped EFM strip, the evidence of Dr. Lackman's timing for conducting her assessment and balancing that evidence with the evidence of the parties' experts, I find that the RTS commenced between 14:25 and 14:30, and therefore completed between 14:45 to 14:50.

THE DUTY OF CARE

[63] There is no question but that Nurse Planques owed a duty of care to both mother and fetus, indeed Nurse Planques did not argue otherwise.

[64] There is a long line of cases evidencing judicial recognition of a medical caregiver's duty of care to a fetus, a duty which crystallizes upon the live birth of the infant.[7] In the leading Canadian text on medical malpractice issues, *Legal Liability of Doctors and Hospitals in Canada*, Madam Justice Picard of the Court of Appeal of Alberta has written:

> One significant aspect of obstetrics that distinguishes it from other areas of medical practice is that the physician owes a duty of care to *two* patients simultaneously: the expectant mother and her fetus.

[7] See, e.g. *Crawford (Litigation Guardian of) v. Penney*, [2003] O.J. No. 89, (S.C.J.), *aff'd*, [2004] O.J. No. 3669 (C.A.); *Tsur-Shofer v. Grynspan*, [2004] O.J. No. 2361 (S.C.J.); *Ediger (Guardian ad litem of) v. Johnston*, [2009] B.C.J. No. 564(S.C.); *Fullerton (Guardian ad litem of) v. Delair*, [2005] B.C.J. No. 276 (S.C.); *Meyer v. Gordon*, [1981] B.C.J. No. 524 (S.C.).

> Usually, these two duties are not in conflict because the proposed treatment or procedure will be in the best interests of both patients.[8]

[65] In this case, there was no conflict in the duty owed to Ms. Milne and her fetus since the treatment necessary to preserve both maternal and fetal health was urgent C-section delivery.

[66] In *Crawford (Litigation Guardian of) v. Penney*, *supra*, Justice Power conducted a detailed analysis of the duty of care owed to both mother and fetus and found that "there can be no doubt about the existence of a duty of care owed by the Defendants…to the [mother and child] Plaintiffs."[9]

[67] Most recently, in the case of *Ediger (Guardian ad litem of) v. Johnston*, *supra*, Justice Holmes of the British Columbia Supreme Court explicitly stated that a duty of care is owed by medical professionals to a fetus. In so holding, Justice Holmes cited the "long history of judicial recognition of a physician's duty of care to a fetus." [10] Justice Holmes' reasons apply equally to members of the hospital nursing staff.

[68] Without question, Nurse Planques and Dr. Lackman owed a duty of care to Ms. Milne and Jessy.

THE STANDARD OF CARE

[69] In a medical malpractice case, the plaintiffs bear the onus of proving that the conduct of the defendants fell below a reasonable standard of care. Whether the health care practitioner whose care is at issue is a physician or a nurse, the leading formulation of the standard of care is as follows:

> …Every medical practitioner must bring to his task a reasonable degree of skill and knowledge and must exercise a reasonable degree

[8] Picard, *Legal Liability of Doctors and Hospitals in Canada*, *4th Ed*., (Toronto: Carswell, 2007) at p. 334.
[9] *Crawford*, *supra*, note 7, para. 208.
[10] *Ediger*, *supra*, note 7, para. 206.

> of care. He is bound to exercise that degree of care and skill which could reasonably be expected of a normal, prudent practitioner of the same experience and standing, and if he holds himself out as a specialist, a higher degree of skill is required of him than one who does not profess to be so qualified by special training and ability.[11]

[70] The principles for assessing the standard of care of a nurse are the same as those for physicians. Justice Picard writes:

> The principles that were discussed earlier in the book with respect to the negligence of doctors apply equally to nurses. In particular, the principles relating to the assessment of the standard of care are the same. Thus, the standard of care expected of nurses is that of the reasonable nurse in similar circumstances.[12]

[71] In the present case, Nurse Planques must be judged according to what a reasonable obstetrical nurse with a similar degree of experience would do in the circumstances. Nurse Planques testified that obstetrical triage nurses are exceptionally experienced and that one cannot become a triage nurse without the requisite level of knowledge and experiences. Similar evidence was given by the plaintiffs' experts, Ms. Rokosh and Ms. Doren.

[72] Ms Rokosh is an obstetrical nurse with 20 years' experience in Alberta. She left clinical practice in 2008 to devote herself to her consulting business, which focuses, in large part, on the preparation of medico-legal opinions.

[73] Ms. Doren, an obstetrical nurse currently practising at Halton Healthcare Services, which is a community hospital, also worked at the Humber River Regional Hospital between 1995 and 1997. She was initially retained by counsel for Dr. Lackman to provide an opinion on the standard of care of Nurse Planques.

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[11] *Crits v. Sylvester* (1956), 1 D.L.R. (2d) 502 (Ont. C.A.), aff'd (1956), 5 D.L.R. (2d) 601 (S.C.C.).
[12] Picard, *supra*, note 8, at p. 489.

[74] Because of the "team approach" to modern health care delivery, evidence of both nurses and physicians is admissible in determining whether the nursing standard of care was met.[13]

[75] Nurses are professionals who also possess special skills and knowledge. They have a duty to use those skills in making appropriate assessments of patients and to communicate accurately those assessments to physicians.

[76] In addition to being influenced by the level of experience of the nurse, the applicable standard of care is also influenced by the foreseeable risk of the clinical circumstances. As the degree of risk of the situation increases, so too must the vigilance and the standard of care of the nurse rise. In particular, the standard of care will rise where the medical practitioner has or ought to have knowledge of the particular risk.[14]

[77] The standard of care is to be assessed at the time and in the circumstances of the alleged negligence, not with the benefit of hindsight.

[78] In the present case, Nurse Planques gave evidence that she has no independent recollection of the events of August 18, 1997, but did testify that she would have known or strongly suspected that Ms. Milne was suffering from an abruption very shortly after doing her initial assessment. Moreover, she testified that she clearly understood the catastrophic risks of an abruption to mother and fetus as well as the potential for sudden deterioration of both patients. This was consistent with the evidence of all of the experts in this case.

[79] The majority of the experts for both the plaintiffs and the defendants were largely supportive of the care provided by Nurse Planques initially upon admission. The evidence was fairly consistent that:

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[13] *Ibid*, at p. 491. See also *Fullerton v. Delair*, *supra*, note 7.
[14] *Ibid*, at pp. 237-240.

a) Nurse Planques performed an appropriate nursing assessment when Ms. Milne initially presented in triage;

b) Nurse Planques appropriately placed Ms. Milne on an EFM;

c) Nurse Planques appropriately took Ms. Milne's vital signs; and

d) Nurse Planques appropriately notified Dr. Gambrill of Ms. Milne's presentation in triage and conveyed sufficient information to enable him to determine that he required an obstetrical consult.

[80] Before Dr. Lackman attended at 14:00, Nurse Planques had a clinical picture and a NST that was non-reactive. Nurse Planques testified that she would have considered the abdominal pain that Ms. Milne presented with as due to four potential causes:

a) bladder infection;

b) kidney infection;

c) pregnancy induced hypertension; or

d) placental abruption.

[81] Nurse Planques testified that she would have ruled out all of these potential causes but for placental abruption "until proven otherwise."

[82] Nurse Planques also testified that she was aware that a history of smoking increased the risk of placental abruption, although it is not clear if Nurse Planques knew of Ms. Milne's smoking habit upon admission.

[83] Nurse Planques testified that she was aware of the signs and symptoms of a concealed abruption and that these kinds of abruptions (compared to a marginal abruption that is accompanied by vaginal bleeding) are particularly problematic because one cannot tell the extent of the hemorrhage. Nurse Planques was aware of the tendency of these abruptions to evolve. Further, she acknowledged that these abruptions cannot get better, but most certainly can be expected to get worse.

[84] Nurse Planques testified that she was aware of the role of the obstetrics team in continuously monitoring fetal status so that intervention could take place before fetal compromise and as a result, obstetrical triage played an important part in preventing compromise to the fetus by early recognition of potential emergencies.

[85] With respect to the care of Ms. Milne, Nurse Planques was aware of the need to watch for signs of maternal problems through repeated measures of vital signs. She recognized that she failed to observe Ms. Milne's vital signs after the initial measure on admission.

[86] With her own nursing diagnosis of placental abruption, Nurse Planques attached the EFM to Ms. Milne to assess fetal well-being. Nurse Planques testified that she recognized the fetal heart rate patterns that suggested impaired oxygenation. In particular, she was aware that minimal variability is one of the main indicators of impaired fetal oxygenation. She also was aware that a non-reactive tracing is suggestive of impaired oxygenation.

[87] The initial EFM ran for 27 minutes, from 13:28 to 13:55. Clearly from all of the experts that testified in this trial, the fetal heart tracing during this first interval was non-reactive, with minimal variability and a baseline of 135 bpm. The baseline was within normal range. Minimal variability is an indicator of impaired fetal oxygenation. The clinical presentation and the non-reactive EFM suggested to Nurse Planques a diagnosis of concealed abruption which, if correct, was a very worrisome scenario. Nurse Planques acknowledged in cross-examination that her anxiety about maternal and fetal well-being would have been heightened.

[88] As a result of the failure of Nurse Planques to document her interactions with Ms. Milne contemporaneously or with any detail, one is not able to determine from the records the amount of time spent by Nurse Planques with Ms. Milne. Ms. Milne testified

that Nurse Planques was with her about 90% of the time she was in triage. There was no evidence offered by Nurse Planques or the hospital to suggest otherwise. Nurse Planques testified that she had no reason to refute the evidence of Ms. Milne regarding the time she spent with her.

[89] Unfortunately, there is no record of a conversation between Nurse Planques and Dr. Lackman at 14:00. Nurse Planques acknowledged in cross-examination the importance of communication among members of the obstetrical team and the need to keep records of those communications. Despite this, there is no record of any communication between Nurse Planques and Dr. Lackman. Nurse Planques is unable to say whether she told Dr. Lackman about her concern about a concealed placental abruption. Nurse Planques is unable to say whether she informed the junior resident about the non-reassuring fetal heart rate tracing between 13:28 and 13:55. Nurse Planques failed to record whether she communicated her level of anxiety for maternal and fetal well-being to the junior resident in view of both the clinical evidence and the tracing evidence.

[90] In particular, there is no record of a discussion about the potential for an emergent delivery in light of what both Dr. Lackman and Nurse Planques perceived to be a likely placental abruption. There was no confirmation as between these two obstetrical team members as to who would contact Dr. Gagnon, the attending obstetrician, in order to ensure that Ms. Milne and her fetus received prompt care from a qualified obstetrician. Most importantly, there was no record of a confirmation that Dr. Gagnon would be contacted immediately to attend Ms. Milne.

[91] Nurse Planques testified that she assumed Dr. Lackman would contact Dr. Gagnon upon completing her assessment, because this was the usual practice at the hospital. Residents were the ones to call their supervisors upon completion of their assessment. Dr. Lackman’s plan, as documented in her late note, was to order blood

work, await the results and then discuss the patient with Dr. Gagnon. In the meantime, Dr. Lackman would be attending another patient's planned C-section. Dr. Lackman testified that she gave Nurse Planques no reason to believe she would be contacting Dr. Gagnon.

[92] At 14:53, Nurse Planques re-attached the EFM. There are no nursing notes to explain the reason for the 58 minute gap in monitoring despite the fact that all of the experts agreed that continuous fetal heart rate monitoring was required in this clinical setting.

[93] When the tracing resumed at 14:53, it showed a fetal heart rate that continued to have what virtually every expert witness said was minimal variability and no reactivity. In addition, the baseline had dropped from its previous level of 135 bpm to a level of 120 bpm. In contrast, the defence expert, Nurse Brown, suggested that variability was average. Nurse Brown is an obstetrical nurse who has practised at Women`s College Hospital and Sunnybrook Health Sciences Centre, both tertiary care centres.

[94] The other defence nursing expert, Nurse Trépanier, also testified that there was average variability in parts of the strip after 14:53, but this testimony was in direct conflict with her own report in which she interpreted the tracing after 14:53 as minimally variable. Nurse Trépanier insisted that her opinion had changed as a result of "intra-observer variability" in interpreting the tracing. Nurse Trépanier has spent only three years in a clinical practice, leaving practice in 1984 for an academic career teaching.

[95] Notwithstanding the two defence experts' interpretation of the EFM, Nurse Planques testified that the tracing at 14:53 was non-reactive with minimal variability and that she could not have been reassured by the tracing at that time. The tracing ran from 14:53 until approximately 15:06 when Ms. Milne was documented on the EFM paper to have gone again to use the bathroom.

[96] In her late note, Nurse Planques documented that at 14:53, Ms. Milne was complaining of "++ pain" and asking for pain medication.[15] Nurse Planques documented her conversation with Dr. Lackman, requesting pain medication for Ms. Milne, which the resident refused. There is no documentation of a conversation between Nurse Planques and Dr. Lackman at 14:53 of the fact that Dr. Gagnon had not yet arrived to see Ms. Milne. In fact, there is no record of any discussion at that point between Nurse Planques and Dr. Lackman about whether Dr. Gagnon had been called.

[97] After 15:06, there was a further absence of continuous fetal heart rate monitoring. At 15:10, there appears to be a 40 second portion of tracing, then no tracing at all until 15:18. Nurse Planques recorded in her late note that the fetal heart rate at 15:10 was 118 bpm.

[98] When the tracing resumed again at 15:18, Nurse Planques recorded a baseline heart rate of 109 bpm. She then recorded in her late note that she contacted Dr. Lackman in Cesarean section room 4 and that Dr. Lackman "requested that I contact Dr. Gagnon." Dr. Gagnon's evidence, given by way of an Agreed Statement of Evidence was that he received a call from Nurse Planques sometime between 15:20-15:25.

[99] In her late note, Nurse Planques recorded that at 15:25, "FHR 110 bpm". She subsequently recorded at 15:28, "Dr. Natale seen in hall and I asked him to see patient, he requested I call Dr. Gagnon, which I did."

[100] When Dr. Natale came into the room, he did a quick assessment and a RTS. He appreciated, almost immediately that Ms. Milne had suffered a massive placental abruption and ordered an emergent C-section delivery. Nurse Planques' late note bears the entry, "Dr. Natale saw patient, RTS done, clot seen on scan, patient moved to CR#5 for emergent C-section."

[15] Exhibit 1, Vol. 1, p. 14.

[101] The medical records reveal that Nurse Planques initiated an intravenous line for Ms. Milne at 15:30.[16] Nurse Planques testified that this was done in anticipation of the emergent C-section after Dr. Natale became involved.

[102] Ms. Milne was taken to C-section room 5 at 15:35. When Ms. Milne arrived in the C-section room, Nurse Planques attempted to auscultate the fetal heart but was unable to do so.

[103] Jessy was delivered shortly after that, at 15:43.

Analysis on the standard of care of Nurse Planques:

[104] Nurse Planques admitted that she knew Dr. Lackman was a junior resident. She testified that she knew that a junior resident had limited experience in obstetrics. She knew that residents have varying levels of skill and knowledge. Nurse Planques admitted that the hospital takes care to ensure that only experienced obstetrical nurses are allowed to work in triage and this is for patient safety reasons. She acknowledged her obligation to communicate with other members of the obstetrical team in order to advocate for patient care. She testified that she would want the resident to appreciate any concerns that she, as a nurse, had about patient safety.

[105] Nevertheless, Nurse Planques never contacted the on-call obstetrician before 15:20. Based on all the evidence about the need for communication among the obstetrical team, the standard of care would clearly require that both the nurse and the resident initiate that communication for patient safety reasons.

[106] Nurse Planques testified that she knew Dr. Lackman was doing a BPP or a RTS. Nurse Plaques admitted she would have been very concerned with the outcome of the BPP. She knew, given the fact that she already suspected an abruption and had a fetal

[16] *Ibid*, p. 7 (bottom left, under the heading "Progress Notes").

heart tracing with which she was "unhappy," that the BPP score would have provided very important supplemental information about fetal well-being.

[107] Nurse Planques confessed that she would have been sufficiently anxious that she would want to have known the BPP result "as quickly as possible". Had the BPP been completed at between 14:45 and 14:50, as my finding is, that is the time that Nurse Planques should have been aware of the results. Nurse Planques also admitted that she had an obligation to find out on her own what the results of the BPP were. She also conceded that with the BPP results she would have been "even more anxious about fetal well-being" and would have been "very worried about abruption and asphyxia".

[108] Even if the resident failed to share her impressions with Nurse Planques, more particularly not having provided Nurse Planques with the results of the BPP, even defence expert Nurse Brown conceded that an experienced obstetrical triage nurse dealing with a suspected abruption would have an interest in knowing the results of the BPP that was done.

[109] This is not a case where Nurse Planques testified that she suspected a serious condition like placental abruption and then had her anxiety calmed because the physician told her it was in fact something far more benign. On the contrary, on Nurse Planques' own evidence, she was operating on the assumption that it was placental abruption "until proven otherwise."

[110] During cross-examination, Nurse Planques agreed with the following propositions suggested by Mr. Halpern:

i) as an experienced triage nurse, she would have wanted the junior resident to appreciate any concerns that she, as a nurse, had about patient safety;

ii) Nurse Planques would have wanted to know the results of the BPP as soon as possible and knew that a score of two out of eight given the

> clinical presentation and the non-reactive EFM, would have heightened her anxiety about abruption and possible emergent C-section delivery.

[111] Nurse Planques did none of these things and as a result her conduct establishes that she failed to deliver the level of nursing care that she acknowledges the patient was entitled to. Nurse Planques, through her own testimony, has established a breach of the standard of care.

[112] That standard did not require her to supervise the junior resident. That standard required that she absolutely recognize that Ms. Milne had a placental abruption from the history taken on admission and the first 27 minutes of tracing. This she did. The standard further provided that she share her concerns with the obstetrical team in a manner that ensured maternal and fetal well-being. This she did not or there is no record of that being done.

[113] The defence's position regarding Nurse Planques' standard of care is that there was a "protocol" at the hospital back in 1997, about communication between nurse and obstetricians. No one testified on behalf of the hospital regarding any "protocol."

[114] In my view, Nurse Planques was perfectly entitled to rely upon and assume that the junior resident had met the standard of care by having communicated with Dr. Gagnon immediately after completing her assessment.[17] Nurse Doran, in cross-examination, agreed that a nurse would have every expectation that the resident would contact the obstetrician on call.

[115] However, there comes a time when Nurse Planques was required pursuant to the nursing standard of care to act and, in particular, to have herself called Dr. Gagnon when he did not arrive in a timely way. I find that it was not appropriate for her to continue to assume Dr. Gagnon had been called, especially when it became clear five to

ten minutes after Dr. Lackman left Ms. Milne's room that Dr. Gagnon was not yet in attendance.

[116] In addition, Nurse Planques ought to have anticipated that an emergency C-section delivery would be very likely and steps ought to have been taken to prepare the C-section room.

[117] With my finding that Dr. Lackman's assessment was completed between 14:45 and 14:50, ultimately Nurse Planques ought to have become suspicious when Dr. Gagnon had not yet arrived by 14:55 to 15:00 and, therefore, she ought to have activated the chain of command by no later then 15:00. In so doing, it is very likely that Dr. Gagnon would have arrived by no later then 15:10.

[118] Based on the evidence that I have accepted as fact, Nurse Planques failed to advocate for the safety of her patients (mother and fetus), in that she failed to adequately communicate with any of the obstetrical team about her awareness and concerns for:

a) the fact that she suspected a concealed abruption;
b) the potential for maternal compromise;
c) the potential for fetal compromise;
d) the likelihood of impaired fetal oxygenation;
e) the potential for a sudden change in fetal status;
f) the potential for an emergency delivery;

[119] The most significant breaches of the standard of care of Nurse Planques were her failure to continuously monitor the fetal heart rate and her failure to ensure timely intervention by Dr. Gagnon. This is not to minimize the other aspects of nursing care where Nurse Planques was deficient in her lack of documentation of conversations.

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[17] *Granger (Litigation Guardian of) v. Ottawa General Hospital*, [1996] O.J. No. 2129.

However, the most important breaches which most clearly led to the delay in appropriate intervention was the failure to ensure timely care by a qualified obstetrician, namely Dr. Gagnon, in the face of a suspected abruption and non-reassuring results from antenatal surveillance including the NST and RTS.

[120] As a result of these failures and on a balance of probabilities, Nurse Planques has breached her standard of care.

[121] I will discuss the issue of several liability later.

DR. LACKMAN'S BREACH OF STANDARD OF CARE:

[122] Both Dr. Braithwaite and Dr. Davies testified that Dr. Lackman breached the standard of care of a second-year resident by failing to call the on-call obstetrician, Dr. Gagnon, immediately after completing the RTS. Accordingly, had Dr. Gagnon been called when she completed her assessment between 14:45 and 14:50, Dr. Gagnon would have attended by no later than between 14:55 and 15:00. Jessy would likely have been born by no later than 15:20 to 15:25.

[123] I say this because we know that Dr. Natale's assessment including a RTS and an emergent C-section to deliver Jessy was conducted in 13 minutes. Given this rapid response by an experienced obstetrician, I see no reason why Dr. Gagnon, another experienced obstetrician assessing the situation 30 minutes earlier, would not have reached the same conclusions as Dr. Natale. Dr. Gagnon likely would have conducted a RTS and would have seen what Dr. Natale did (that is a large blood clot behind the placenta). Surely, the clinical signs, the non-reassuring EFM, the BPP of two out of eight and the drop in fetal heart rate over the monitoring period, would have suggested to Dr. Gagnon that this fetus had to be delivered immediately before bradycardia set in. All of the experts agreed on that point.

CAUSATION:

[124] In order to understand the issue of causation in this action, I must review some elements of the scientific evidence presented in this case.

Placental abruption:

[125] The placenta is attached to the uterus. The fetus is connected to the placenta via the umbilical cord. The umbilical cord contains two arteries and one vein. The baby receives oxygen from the mother through the placenta. Fetal circulation carries oxygen enriched blood to the fetus through the umbilical vein. The umbilical arteries return waste products from the fetus to the placenta for eventual elimination by the mother.

[126] The ability of the fetus to receive adequate oxygen depends on the delivery process between mother and fetus that normally occurs through a placenta attached to the uterus. A placental abruption is the premature separation of the placenta from the uterus.[18] When the placenta separates it can impair the exchange of gases between mother and fetus.

[127] There are two types of placental abruption: marginal and concealed. A marginal abruption generally involves some vaginal bleeding and little or no pain. It is called marginal because only a small portion of the placenta separates from the uterus. With a marginal abruption, there is no accumulation of blood because it is discharged through the vagina.

[128] With a concealed abruption, there is no bleeding through the vagina and the mother presents to hospital with a sudden severe onset of abdominal pain. This condition

[18] Premature in the sense that shortly after birth the placenta does completely separate from the uterus and is expelled.

is called “concealed” because the bleeding, or hemorrhage, occurs between the placenta and uterus and gets trapped there, resulting in severe abdominal pain for the mother.

[129] The management of the care of a patient who presents with marginal abruption is quite different from the management of the care of a patient presenting with a concealed abruption. The marginal abruption can often be managed conservatively, provided there is a reassuring evidence on EFM of fetal well-being.

[130] With concealed abruption, however, management of the patient is quite different. First, the diagnosis of placental abruption is a clinical diagnosis. That is, the diagnosis is made on the basis of the medical history and examination of the patient, without the need for other diagnostic tests. According to Dr. Braithwaite, mothers with concealed abruptions present with severe abdominal pain together with a tense and tender abdomen. They may vomit from the severity of the pain. Even in the presence of abruption maternal vital signs[19] are often normal.

[131] Both mother and fetus are in peril in the setting of a concealed abruption. As most of the experts have testified, the fact that the abruption is concealed means that one is unable to tell the extent of the separation and therefore the risk to mother and fetus.

[132] Abruptions evolve. The evolution of a placental abruption was described by Dr. Braithwaite and illustrated on Exhibit #9. The abruption may start with a small separation of the placenta from the uterine wall with a small hemorrhage. The placenta, according to Dr. Braithwaite, has a certain degree of redundancy built in, which means that a small degree of separation may not impact fetal oxygenation at all. The risk to mother and fetus is of the separation spreading further. As the abruption evolves, a larger area of the placenta tears away from the uterus resulting in further blood collecting

[19] Heart rate, blood pressure, temperature.

between the placenta and the uterus. At this stage, the oxygen supply to the fetus becomes more and more compromised.

[133] The objective in the clinical setting of a placental abruption is to intervene by delivering the baby before a complete abruption occurs. In other words, before the baby goes over the precipice. With a complete abruption there is a massive separation of the placenta from the uterus which causes the oxygen supply to the fetus to be cut off entirely. Dr. Perlman described a complete abruption as a catastrophic event causing a sudden and profound asphyxic environment in which the fetus has little opportunity to avoid permanent neurological injury. The complete separation of the uterus from the placenta results in a sudden profound asphyxia which causes the fetal heart rate and blood pressure to fall. A fall in blood pressure (hypotension) causes ischemia[20] to the organs, including the brain.

[134] In circumstances where the clinical picture is suggestive of a concealed placental abruption and the fetal heart rate is in the normal baseline range then, according to Dr. Braithwaite, the fetus is likely not "in extremis" and if delivered promptly you can expect an "intact" baby, or a baby without any neurological injury. Dr. Braithwaite testified that the obstetrical team would want to deliver the baby before there is any evidence that the fetus is "grossly distressed."

[135] Dr. Braithwaite testified that Ms. Milne's clinical presentation when she came to triage was classic for placental abruption. Dr. Davies was of the same view.

Prolonged partial asphyxia

[136] Placental abruption impairs the delivery of oxygen to the fetus. While the placenta has built-in redundancy, allowing the fetus to be unaffected by a marginal

[20] A drop in fetal blood pressure can result in organs, including the brain, being inadequately perfused with blood and can lead to a condition called "ischemia."

separation of the placenta from the uterus, as the degree of separation evolves, the impairment of oxygenation begins to impact on the fetus more significantly. The fetus is equipped with a number of compensatory strategies that allow it to withstand this impaired gas exchange for hours, if not days. The ultimate impact of impaired gas exchange on the fetus will depend on the duration and magnitude of the insult.

[137] The word "insult" describes the process by which fetal oxygenation becomes impaired. It is to be distinguished from "injury," which is used to describe the consequences where the insult has been of sufficient duration and severity to begin to cause harm to the fetus and may not be reversed.

[138] The placenta, in part, operates as a means of gas exchange between mother and fetus. Oxygen (O_2) is delivered to the fetus and carbon dioxide (CO_2) and other metabolites are expelled. When gas exchange is impaired, there is less oxygen in the fetal blood (known as hypoxemia) and a build up of CO_2 in the fetal blood. Asphyxia is simply the impaired blood gas exchange which leads to progressive decrease in O_2 and increase in CO_2 leading to a condition in the fetal blood called "metabolic acidosis." Increasing levels of metabolic acidosis may expose the fetus to an increased risk of neurological injury, among other things.

[139] Dr. Armstrong said that a gradual and incomplete impairment of gas exchange is called a "Prolonged Partial Asphyxia" (prolonged partial). The prolonged partial can continue for hours and even days without causing permanent neurological harm to the fetus. Fetal response to this impaired gas exchange can be revealed through changes of the fetal heart rate pattern as shown on the EFM tracing. Therefore, clinical indications that a fetus might be decompensating due to prolonged partial will be revealed on the tracing. As Dr. Perlman indicated, while it may not be possible to detect the degree of hemorrhage in a concealed abruption, fetal response to the abruption will be revealed through the level of fetal disturbance on the heart rate monitor.

[140] As indicated in the section dealing with metabolic acidosis, the body has ways of burning fuel without oxygen. This is part of the compensatory measures available to the fetus to avoid harm from increasing metabolic acidosis.

[141] With prolonged partial, acidosis gradually increases. The fetus has three main compensatory responses to increasing acidosis which help the fetus avoid any permanent injury.

[142] Dr. Armstrong said that the initial response to a build-up of acids is called the "Buffer Stage". The fetal blood contains natural alkaline (base) chemicals which are said to buffer or neutralize the acids when they build up. The buffering capacity of the blood can hold off acidification of the blood for some time. With prolonged partial, the stages are as follows:

(i) An event causes some impairment of gas exchange.

(ii) There is a build-up of acid so that there is more acid than base in the fetal blood.

(iii) A chemical reaction occurs which neutralizes the build up of acid (called the buffer).

[143] During the Buffer Stage, there is no risk of neurological injury to the fetus.

[144] When the body detects that more than just the natural chemical buffers are needed to neutralize the building acidity, the fetus initiates a cardiovascular compensatory measure.

[145] The cardiovascular stage of fetal compensation for increasing acidosis involves a change in blood flow. There are two significant aspects to the cardiovascular response. First, blood is directed from non-essential organs to essential organs. The essential organs that receive preferred blood flow are the heart, brain and adrenal glands.

The second aspect of the cardiovascular response is to cause blood vessels to be dilated and fetal blood pressure to increase, allowing a greater volume of blood to be circulated. In this way, the essential organs continue to be adequately perfused with blood.

[146] The increased blood supply to the heart and the dilated blood vessels allow the heart to pump enough blood to the brain to avoid neurological injury. As long as this process continues, the increased perfusion of blood to the brain compensates for the reduction of oxygen in the blood.

[147] The adrenal glands, which sit on the kidneys, are essential organs and are protected because they are important in stressful situations. The adrenal glands release important hormones that help the fetus cope with the stress of impaired oxygenation. Some hormones released from the adrenal glands increase the pumping action of the heart, constrict some blood vessels to non-essential organs and dilate blood vessels to essential organs.

[148] During the cardiovascular stage of fetal compensation, the brain is protected from injury. The increased blood flow delivers oxygen and allows the brain to get rid of acids.

[149] Following the cardiovascular stage, another compensatory measure provides added protection to the brain. This is the brain blood flow stage. Increasing acidosis triggers a response within the brain to direct more blood to the areas of the brain with high metabolic needs and less blood to the areas of the brain with lower metabolic demands. Those areas of the brain with higher metabolic demand are the deep structures, including the basal ganglia and thalami. The areas of the brain with less need for oxygen are the outer areas of the brain, referred to as the cerebral hemispheres or the cerebral cortex.

[150] In the brain blood flow stage, the cerebral cortex receives less blood, while preferential amount of blood goes to the deep structures. When this occurs, the cerebral cortex, even though receiving less blood, can remain uninjured because its metabolic demands drop so that its energy demands are very low. In order to continue to avoid neurological injury, maintaining blood pressure is essential. The clinical evidence of maintained blood pressure is the fetal heart rate. A baseline heart rate within the normal range of 120 to 160 bpm indicates that blood pressure is being maintained. When the heart rate drops below the normal range, it means that blood pressure also drops, resulting in less perfusion to the brain and, if allowed to continue, the risk of permanent neurological injury exists.

[151] According to Dr. Armstrong, for the heart rate to drop, there must be ischemia to the heart. The heart is first affected by ischemia, followed by a drop in heart rate and blood pressure which then leads to brain ischemia. Dr. Armstrong testified that when the heart is pumping properly there will be no ischemia to the brain. Dr. Marrin testified that where there is a normal baseline, the essential organs (the heart, brain and adrenal glands) are receiving the same volume of blood. Put another way, according to Dr. Armstrong, hypoxia (or hypoxemia) is unlikely to cause neurological injury as long as heart rate and blood pressure are maintained. Dr. Marrin echoed this opinion.

[152] When the fetal heart rate drops, there is a drop in blood pressure and a corresponding decrease in blood perfusion to the brain. In the case of prolonged partial, the area of the brain that will be first to suffer damage is the cerebral cortex, because blood flow to that area of the brain is already reduced. The chance of any brain damage to the cerebral cortex before there is a fall in heart rate and blood pressure is very unlikely, according to Dr. Perlman, and if there were it would be very subtle. For more profound injury to the brain to occur, there must be ischemia which occurs with the drop in heart rate and blood pressure.

[153] Both hypoxia (less oxygen) and ischemia (poor perfusion of blood to the organs) is needed to cause damage to the brain. The process starts with hypoxia, for which the fetus will compensate as indicated above. When the point is reached where the acidity in the blood becomes unbearable, and circulating acids cannot be removed, ischemia will develop and begin the process of increasing neurological harm.

[154] In the absence of a profound total asphyxia, discussed below, the areas of the brain most vulnerable to injury from prolonged partial asphyxia are the cerebral cortex and the white matter. Sudden profound asphyxia can occur with or without prolonged partial asphyxia. When they both occur, there is said to be a "mixed" pattern, which will have slightly different consequences in terms of the areas of the brain that are damages.

Sudden profound asphyxia (acute total):

[155] Sudden profound asphyxia or total profound asphyxia is a complete or near-complete interruption in the supply of oxygen. This total lack of oxygen can be tolerated, as one would assume, for only a relatively brief period of time before there is permanent neurological damage and, if unabated, death.

[156] As explained by both Dr. Perlman and Dr. Armstrong, with total profound asphyxia the heart rate drops dramatically within a minute or two of the onset of the profound interruption in blood supply and oxygen. In the presence of a total profound asphyxia the fetal heart rate will drop to a level of 60-70 beats within three to four minutes of onset. This is called a severe bradycardia. A total profound asphyxia can happen with a severe placental abruption.

[157] Dr. Armstrong testified that a sudden profound asphyxia results from a very low fetal blood pressure that leads to little or no oxygen supply. This is caused by an extreme intrauterine event like a massive placental abruption. The fetal brain receives no

oxygen and ischemia results, causing neurological injury. Dr. Armstrong further testified that a severe sudden profound asphyxia will damage both the cerebral hemispheres (or cerebral cortex) and the deeper structures (the basal ganglia and thalami). He testified that a severe bradycardia can be expected to injure the brain globally (which includes the cerebral hemispheres and deeper structures).

[158] Dr. Perlman testified that when a fetus has been exposed to a prior period of prolonged partial asphyxia, the capacity to withstand a subsequent profound total asphyxia is compromised. With a prior period of hypoxia due to prolonged partial asphyxia, the fetus will have already become somewhat acidotic, with a low pH. This means that it will take less time during a profound total asphyxia to reach a critical level of acidosis where the fetus is over the precipice.

[159] According to Dr. Armstrong, the injury to Jessy's brain began with the bradycardia identified by Dr. Natale at 15:33, where the fetal heart rate was 80-100 bpm, followed a minute later by a fetal heart rate of 60-70 bpm, and subsequently at 15:35 when the fetal heart rate was not auscultatable.[21] Dr. Armstrong testified that this was followed by cardiac arrest, with no heart beat at all.

[160] With complete separation of the placenta from the uterus, no oxygen was getting to the fetus. This would cause a bradycardia within three or four minutes and would be followed by a rapid rise in the base deficit (BD) (To be explained later).

Mixed pattern:

[161] Where the fetus is exposed to a period of prolonged partial asphyxia followed by a period of profound total asphyxia, it is said to be a "mixed asphyxial insult". Outcomes for fetuses exposed to mixed insults are worse than those exposed to either a prolonged partial asphyxia or a profound total asphyxia.

[162] In the presence of an evolving placental abruption, the fetus is initially exposed to a partially impaired gas exchange that results in a gradually increasing acidosis. While this can be tolerated for lengthy periods of time, the gradual increase in acidosis makes the fetus particularly vulnerable to a subsequent total profound asphyxia. This is because when a total profound asphyxia begins, the fetus has already been exposed to a period of hypoxia. As a result, it takes less time for acidosis levels to increase to the point of neurologic injury. In other words, a fetus may have a BD of between 12 and 16 due to prolonged partial asphyxia and, if rescued, will very likely be entirely normal. If, however, when total profound asphyxia begins, the BD is already say 16, then it will not take much time during the total profound asphyxia to cause the BD to rise above 20, increasing the likelihood of permanent brain damage.

[163] The longer the total profound asphyxia continues, when superimposed on a fetus already vulnerable from a prolonged partial asphyxia, the more profound the neurological injury.

[164] Dr. Armstrong testified that with a mixed pattern of asphyxia, the fetus may suffer injury to both the cerebral hemispheres and the deeper brain structures. He also testified that a severe sudden profound asphyxia on its own can damage both the deeper structures and the cerebral hemispheres.

Brain anatomy and imaging:

[165] The only expert witness called and qualified to testify about brain imaging was Dr. Derek Armstrong, pediatric neuroradiologist at the Hospital for Sick Children in Toronto. Dr. Armstrong is currently on staff at the Hospital for Sick Children where he practices as a diagnostic radiologist for children, including newborns following any period of gestation. He is also an Associate Professor of Neuroradiology at the

[21] Could not be heard with an ultrasound or Doppler device.

University of Toronto. Neuroradiology involves imaging of the central nervous systems, the brain and its coverings through, among other things, MRI, CT scan and ultrasound.

[166] Dr. Armstrong testified that neuroradiology can be used to pinpoint the timing of an injury to the brain suffered in the uterus. Imaging alone can pinpoint the timing within days and when coupled with the available clinical information, the timing of any neurological injury can be pinpointed far more precisely. According to Dr. Armstrong, he has the expertise to time neurological injury to within minutes of the occurrence of the injury when diagnostic imaging interpretation is married to clinical events.

[167] Dr. Armstrong described the parts of fetal and newborn brain anatomy relevant to the matters in issue. These parts of brain anatomy were illustrated in Exhibit #14, Early Normal Fetal Brain Anatomy. The important structures include the cerebral hemispheres, the ventricles and the deeper structures of the brain.

[168] The cerebral hemispheres, which are part of what has been described as the cerebral cortex, are the outer layers of the brain. The cerebral cortex includes both grey matter and white matter. The metabolic demand of the cerebral cortex is relatively low compared to the deeper structures. As a result, the cerebral cortex is able to withstand a certain degree and duration of hypoxia without permanent neurological injury.

[169] The ventricles are spaces within the brain that are filled with cerebral-spinal fluid. When there is injury to other structures of the brain, the ventricles can enlarge, entrapping more fluid because the fluid is not allowed to drain properly. If the natural ability to drain fluid continues to be impaired, the ventricles enlarge which in turn compresses the rest of the brain resulting in neurologic injury. Where inadequate drainage of fluid from the ventricles exists, a shunt can be surgically inserted into the brain to allow fluid to drain and relieve pressure.

[170] The deeper structures of the brain include the basal ganglia and thalami. These structures have a higher metabolic demand and therefore need more blood perfusion and oxygen in order to avoid neurological injury. As a result, one of the natural compensatory strategies a fetus has to cope with decreased oxygenation is the brain blood flow reflex that causes a preferential share of blood to be directed from the cerebral cortex to the deeper structures, which provides resistance to brain injury.

[171] According to Dr. Armstrong, when the brain blood flow reflex redirects blood from the cerebral cortex to the deeper structures of the brain in response to hypoxia, it does not necessarily mean that there is any injury to the brain. As the cerebral cortex has lower metabolic demands, it can withstand this reflex for some time. Dr. Armstrong testified that the process can go on for several hours without injury to the cerebral cortex. The cerebral cortex continues to be perfused with blood. It is only when the condition progresses to the point that there is a drop in fetal heart rate and blood pressure that ischemia sets in that the brain can be injured. In other words, it is the hypotension that results from a bradycardia which leads to brain injury.

[172] Jessy suffered from Hypoxic Ischemic Encephalopathy (HIE). Hypoxia refers to reduced oxygenation. Ischemia refers to a reduction in the amount of blood supplied to an organ, such as the heart or brain. Encephalopathy refers to disease of the brain. It is important to note that a fetus can be exposed to hypoxia without becoming ischemic. Dr. Armstrong testified that ischemia does not occur until the fetal heart rate drops, with a corresponding drop in blood pressure which subsequently leads to ischemia. Dr. Marrin, a neonatologist called by the defence also testified that ischemia will occur only when there is a drop in the heart rate and hypotension. Both of these experts testified that the clinical evidence of ischemia is a bradycardia where the fetal heart rate drops below the normal range of 120 to 160 bpm.

[173] A CT scan done on August 21, 1997, at three days of age, was illustrated on Exhibit #17. This scan provided information about the timing of Jessy's injury. The images demonstrated brain swelling involving the hemispheres. The images also demonstrated damage to the basal ganglia and thalami. Dr. Armstrong testified that these images establish that the brain injury suffered by Jessy was an acute injury. The images also demonstrated bleeding within the ventricles, a condition known as "intraventricular hemorrhage". The ventricles were enlarged with both blood and cerebral-spinal fluid. The intraventricular hemorrhage, according to both Dr. Armstrong and Dr. Max Perlman, were a direct result of asphyxia. The injury to the entire brain is a result of the hypoxic ischemic encephalopathy (HIE) which was a consequence of hypotension.

[174] Specifically, Dr. Armstrong testified that Jessy suffered from a moderate hypotension at about 15:33 followed by a severe hypotension a few minutes later. Initially, the moderate hypotension would have damaged the cerebral hemispheres. A profound hypotension followed immediately and damaged the deeper structures of the brain. Injury to Jessy's brain would have continued after birth, in the seven minutes it took the resuscitation team to restore Jessy's heart rate.[22]

[175] Illustrations of imaging done on August 25, 1997 (seven days of age), Exhibit #20, show further enlargement of the ventricles and some retraction of the hemorrhage within the ventricles. The cerebral hemispheres remain abnormal, with a loss of grey matter. Acute cerebral edema (brain swelling), according to Dr. Armstrong, lasts five to seven days. He further opined that this imaging established that Jessy's brain injury occurred at about the time of his birth. Dr. Armstrong testified that the imaging proves that Jessy's brain was entirely normal or "pristine" before that.

[22] Exhibit 1, vol. 1, pp. 84-85 for the Resuscitation Record.

[176] By September 12, 1997 (25 days of age), illustrations of imaging on Exhibit #22 show severely dilated ventricles[23] due to a blockage of the pathway for draining the fluid, resulting in a condition known as “hydrocephalus”. The hydrocephalus, or build-up of fluid in the ventricles, caused pressure to build in Jessy’s brain, thereby compressing his brain and causing the death of adjacent brain cells.

[177] More current images of Jessy’s brain injury are set out in an illustration of imaging done on September 11, 2008, when Jessy was 11 years of age. These images show the shunt that had been inserted surgically to drain cerebral-spinal fluid. The ventricles of Jessy’s brain remain abnormally large, according to Dr. Armstrong, because of loss of brain tissue in the cerebral hemispheres, called “encephalomalacia”. The basal ganglia and thalami are also diminished in size and there is no differentiation between the grey and white matter.

[178] With damage to the grey matter, white matter, ventricles and deep structures of the brain, Jessy has been left with a severely compromised central nervous system that significantly impacts on his neurological functioning. Damage to the hemispheres involves thinking, ideas, sensory and motor issues and vision. These areas of the brain, when damaged, result in clumsy and spastic movements associated with a condition known as cerebral palsy.

Metabolic acidosis:

[179] Dr. Perlman, the plaintiffs’ expert, and Dr. Marrin called for the defence explained metabolic acidosis.

[180] The body needs oxygen to burn fuel, like a fire. The less oxygen, the less available fuel that is burned. If the supply of oxygen is only marginally reduced, the fire is still capable of burning most of the fuel. As the oxygen becomes increasingly reduced,

gradually less fuel gets used up and less energy is generated. It is all a matter of degree. When the body has a shortage of oxygen, its ability to burn fuel and create energy becomes gradually impaired. The fuel that the body burns is generally glucose. When the ability to burn glucose is impaired, there is a build-up of acids in the body resulting in metabolic acidosis. It is termed "metabolic" because the ability of the body to process or metabolize fuel is reduced.

[181] Acids build up in the body and can be measured using the pH. The opposite of acid is base or alkali. The pH is a measure of acidity. When the acidity goes up, the pH goes down. A neutral pH level is 7.4, though there is a range of normal. As the fetus becomes increasingly acidotic, the pH level of its blood and tissues drops. When the body is unable to neutralize the acid, a metabolic acidosis develops. The degree of that metabolic acidosis (also referred to as an oxygen debt) is measured by the "base deficit" (BD).

[182] The BD measures the oxygen debt of the fetus. As the BD rises, the oxygen debt rises. The BD is measured in units of millimoles per litre (mmol/L). The BD of a fetus before labour is generally considered to be 2 mmol/L. A BD approaching 30 mmol/L is considered fatal.

[183] The BD is measured from blood taken from the umbilical artery. The blood in that vessel best represents fetal status at the time of birth because blood flows from the fetus to the placenta through the umbilical artery. The blood is then measured for gases. The measurements include BD as well as partial pressure of O_2 (oxygen) and partial pressure of CO_2 (carbon dioxide).

[184] To understand the risk of neurological injury with a rising BD, Dr. Perlman referred to the 1997 article by Low, called "Threshold of Metabolic Acidosis Associated

[23] Called "ventriculomegaly," which means enlargement of the ventricles.

with Newborn Complications" ("Low's 1997 article").[24] Page one of the article, makes reference to the fact that "fetal asphyxia of a particular degree and duration may cause brain damage".

[185] Low's 1997 article sets out a number of ranges for BD from the umbilical artery blood.[25] The subcategories of damage to the central nervous system are:

a) None – meaning no neurological injury at all.
b) Minor – meaning only irritability and jitteriness
c) Moderate – meaning profound lethargy or abnormal tone.
d) Severe – meaning coma or abnormal tone with seizures.[26]

[186] Below is a table taken from Table II in Low's 1997 article that summarizes the probability of permanent neurological injury that is either moderate or severe based on the indicated ranges for BD values:

	Probability of moderate neurological injury[27]	Probability of severe neurological injury	Probability of no injury or only minor injury
BD 4 - 8 mmol/L	3.4%	0%	96.6%
BD 8 - 12 mmol/L	0%	0%	100%
BD 12 - 16 mmol/L	6.9%	1.7%	91.4%
BD >16 mmol/L	28.8%	11.9%	59%

[24] Exhibit 30.
[25] *Ibid*, Table II, p. 1393.
[26] Exhibit 30, p. 1392, left column.
[27] These values are taken from Exhibit 30, Table II. For example, the probability of a moderate neurological injury with a BD of between 12 and 16 mmol/L is 4 (the number of newborns who suffered this type of injury) divided by 58 (the total number of neonates with a BD of between 12 and 16 mmol/L), multiplied by 100 to express as a percentage. 4/58 x 100 = 6.9%.

[187] This means that newborns with a BD between 12 and 16 mmol/L had a 1.7% chance of a severe neurological injury. It also means that those same newborns had a 98.3% chance of not suffering a severe neurological injury. Of the newborns with a BD of between 12 and 16 mmol/L, 91.4% had no neurological injury or only a minor injury.

[188] This table illustrates the fact that even with a rising BD, most newborns do well. It also illustrates that the longer the fetus is left in an intrauterine environment without adequate oxygenation, the higher the BD value rises and the greater the likelihood of permanent neurological injury.

[189] As Dr. Perlman testified the BD alone is not determinative of whether a newborn will suffer a neurological injury. One must also consider the clinical situation before the baby was born and any signs of fetal compromise. Babies have been born with BD values of 26 but still suffered no permanent neurological injury.

[190] It is necessary to look for evidence of decompensation from the fetal heart rate tracing to correlate a BD with the clinical circumstances in order to determine whether an insult is becoming an injury.

[191] The BD is also of value in timing when the insult became an injury. While there are a number of approaches that can assist in timing, extrapolating backwards from the umbilical cord artery BD can be one useful tool.

[192] Since the 1960's, scientists have studied acidosis, particularly in studies involving rhesus monkeys and sheep. There are also a handful of human studies. A thorough search of the literature by Dr. Perlman revealed data which suggests that the rate of rise of the BD in a total profound asphyxia setting is between 1 and 1.4 mmol/L every minute. There is little human data due to the obvious ethical considerations. A

paper by Pasternak[28] involved the study of human infants, with a relatively small sample. While there were very few subjects in this study that had a direct bearing on this case, the subjects that did, demonstrated rates of changes consistent with the conclusions drawn from animal studies.

[193] Further support for the opinion proffered by Dr. Perlman on the rate of rise of the BD during profound total asphyxia can be found in the work of Dr. Low. The data from Dr. Low on rates of change of BD with more profound asphyxia is entirely supportive of the conclusions offered by Dr. Perlman in his evidence.

[194] A leading study that looked at the change of metabolic acidosis over time was done by Low as described in a 1977 article called "Intrapartum fetal asphyxia: Clinical characteristics, diagnosis, and significance in relation to pattern of development" (Low's 1977 article).[29] This article is significant in that it measured BD during labour and at birth.

[195] In Low's 1977 article[30] there were three categories of newborns with intrapartum[31] fetal asphyxia. In one category, the fetuses had been asphyxiated in the last two hours before birth. In a second category, the fetuses had been asphyxiated in the last one hour before birth. The third category, and the category that is relevant to this case, is concerned with the "terminal asphyxia group". As indicated by Low:

> There were 46 patients in the terminal fetal asphyxia category in whom the fetus demonstrated no evidence of metabolic acidosis during labour but who, during the last 15 to 30 minutes of labour, developed a significant metabolic acidosis observed at delivery.[32]

[28] Exhibit 40.
[29] Exhibit 35, Low JA, "Intrapartum fetal asphyxia: Clinical characteristics, diagnosis, and significance in relation to pattern of development."
[30] *Ibid*.
[31] Intrapartum, meaning during labour.
[32] Exhibit 35, pp. 858-859.

[196] The “terminal asphyxia” group represents fetuses with acidosis developed in the last 15 to 30 minutes of labour. By definition, that means that most of those fetuses would not have suffered a profound total asphyxia because if they had, they could not have survived 30 minutes in the uterus in those conditions. It can be expected, therefore, that the rate of change of BD for that group would not be as rapid as the rate of change with more profound asphyxia.

[197] In Low’s 1977 study, Table I sets out the rate of change of BD in the three categories of asphyxiated fetuses.[33] During Dr. Perlman`s testimony in-chief, the relevant data from the Low’s study of 1977 was extracted and put in Exhibit #43. In the terminal group the average BD at between 0 and 15 minutes was 41.3. The average time over which those values was measured was 7½ minutes. The BD at birth (from the umbilical artery) was 34.1 for that same group. That represents a change of BD over an average of 7½ minutes of 41.3-34.1=7.2. This translates to an average change of about 1mmol/L every minute.

[198] Dr. Perlman also testified about animal studies and his own clinical experience that supports the rate of change identified through Low’s 1977 study. It was his evidence that 1 mmol/L per minute would be a conservative estimate of the rate of change during a profound total asphyxia. The rate of change must be distinguished from the rate expected during a prolonged partial asphyxia, which would be a much slower and more gradual change.

[199] During cross-examination of Dr. Perlman, an article by Ross and Gala was put to him.[34] Dr. Perlman testified that in his opinion the Ross and Gala article is mistaken in the interpretation of Low’s 1977 article and the conclusions in that article. Ross took an average rate of rise of BD of 7 mmol/L over 15 minutes to calculate a rate

[33] *Ibid*, p. 858, Table I.
[34] Exhibit 42, Ross and Gala, Use of Umbilical Artery Base Excess: Algorithm for the timing of hypoxic injury.

of rise of 1 mmol/L every 2 minutes. However, as Dr. Perlman testified, Ross failed to take into consideration that 15 minutes was not the time during which the measurements were taken, but rather 0 to 15 minutes, leaving an average of 7½ minutes.

[200] Ross also used the model of reduced uterine blood flow[35] to resemble placental abruption for the purposes of opining on the rate of change of the BD. Again, those conclusions have no application to this case because the model involved a "stepwise" reduction of uterine blood flow, resembling a partial placental abruption but by no means resembling the total abruption and profound total asphyxia.

[201] The umbilical cord artery blood gases for Jessy[36] revealed a BD of 26.8 mmol/L. This would have been indicative of Jessy's state of metabolic acidosis at the time of his birth, at 15:43. A BD of 26.8 mmmol/L is as close a measure can get to ensure irreversible brain damage. The pH in the arterial cord gas was 6.74, reflecting a very severe acidosis, that if any lower, would be incompatible with life. A pH that low is very likely to be associated with permanent brain damage.

[202] The severe bradycardia of 60 to 70 bpm noted by Dr. Natale occurred at approximately 15:34, or nine minutes before Jessy was born. Dr. Perlman`s opinion is that based on a rate of rise of BD of 1 mmol/L per minute, that would mean Jessy had a BD at 15:34 of 17.8 mmol/L (26.8-9=17.8) or at 15:33 of 16.8 mmol/L.

[203] Dr. Davies testified that in his opinion, Jessy's BD at 15:18 (when the heart rate was first noted to dip below 110 bpm[37]) was likely between 12 and 16 mmmol/L.

Analysis of the timing of the injury:

[35] *Ibid*, p. 7.
[36] Exhibit 1, Vol. 1, p. 407.
[37] *Ibid*, p. 14, where Nurse Planques records a fetal heart rate of 109 bpm.

[204] By measuring the gases in the blood sample, conclusions can be made about the degree of acidosis in the fetus and potentially the timing of neurological injury. The two important measures from the arterial cord blood gas test are the pH and the BD.

[205] It is important to state that all experts agreed that different fetuses have different capacities to withstand an increasing acidosis and an increasing BD. It is necessary to look at all the available clinical information when determining whether the fetus has reached its own threshold of tolerance, after which it will start to decompensate and suffer neurological injury. As indicated by Dr. Perlman, the BD cannot be used in isolation.

[206] At the same time, when considering the impact of the BD and the rate of change in the context of this case, it is essential to recall the evidence of all the experts who testified on causation. There was testimony from Dr. Braithwaite, Dr. Davies, Dr. Armstrong and Dr. Perlman to the effect that most, if not all of Jessy's brain damage occurred after 15:33. Dr. Braithwaite and Dr. Davies testified that had Jessy been delivered before 15:33 he would not have been injured. The only defence expert who testified on causation, Dr. Marrin, stated that he agreed with Dr. Perlman that it was very unlikely that severe brain damage had occurred before 15:33. Dr. Marrin went on to say that it was "possible that Jessy already had a degree of brain injury" at 15:33. Dr. Marrin however testified that Jessy most definitely suffered severe brain damage after 15:33. That is consistent with all of the experts who said that serious brain injury occurred after the bradycardia at 15:33.

[207] Dr. Perlman testified that the rate of change of BD during a period of acute severe asphyxia would be conservatively 1 mmol/L every minute. It is acknowledged by the defence expert, Dr. Marrin, that "the rate of change of the base deficit is influenced by the nature of the process leading to deprivation of oxygen". Dr. Marrin testified that the more severe conditions affecting oxygenation would give rise to the most rapid rate of

change of BD. He also acknowledged that the most severe of those conditions would be an acute total asphyxia. Dr. Marrin described the placental abruption as “massive”. Therefore, when looking at rates of rise of BD from the available scientific literature, we can assume that the most rapid rate of rise from the studies is that which would apply to Jessy.

[208] The 1977 Low study demonstrated that the rate of change of BD for the terminal asphyxia group was 1 mmol/L per minute on average. As indicated by Dr. Perlman, that is neither the most rapid rate nor the slowest rate for this group. It is the “mean”. As Dr. Marrin testified, there would have been fetuses in the terminal asphyxia group that had a more rapid rate of rise. He also accepted that there were fetuses in the group that may have only been exposed to a partial asphyxia, making the average rate slower and less representative of acute total asphyxia. Thus, Dr. Perlman’s testimony that the use of 1 mmmol/L per minute of rise of the BD could be conservative is credible and a reasonable scientific conclusion.

[209] Dr. Marrin acknowledged in his testimony that Dr. Perlman may well have been correct that the rate of change of the BD. for Jessy after 15:33 was 1 mmol/L every minute. Dr. Marrin said that he took exception to using that precise calculation. He preferred to say that there was a “range” of rates that could apply that included 1 mmol/L per minute. Yet the clinical evidence of a severe bradycardia beginning at 15:33 is entirely compatible with the beginning of fetal decompensation caused by a total placental separation which would yield the most rapid rate of rise of BD. Some of the fetuses in the 1977 Low study would have had an even more rapid rate of rise, also acknowledged by Dr. Marrin in his evidence.

[210] The rate of rise of the base deficit proposed by Dr. Perlman, acknowledged by Dr. Marrin as being within the range and supported by the Low 1977 study is also entirely consistent with the evidence of Dr. Armstrong, Dr. Braithwaite and Dr. Davies.

Dr. Armstrong testified that Jessy's brain was uninjured before the bradycardia at 15:33 since ischemia to the brain would not have occurred until hypotension developed. Dr. Braithwaite testified that Jessy would have been "intact" or neurologically normal had he been delivered at 15:33. Dr. Davies testified that Jessy would have had a BD of between 12 and 16 at 15:18 and would have been neurologically normal if delivered before 15:33. All of this evidence is compatible with Dr. Perlman's testimony on the rate of change and the likely BD at 15:33 of about 16.8 mmol/L.

[211] With regard to timing, Dr. Armstrong testified that by using the neuroimaging, he is able to time the injury within days but he can also be more precise when considered with the clinical evidence. When pressed in cross-examination about how many times Dr. Armstrong has been called upon to give an opinion on precise timing of neurological injury, he responded "in the majority of cases". During cross-examination Dr. Armstrong agreed that the bradycardia could have begun at 15:18, which is the first documented time that Jessy's heart rate dropped below 110 bpm. Dr. Armstrong described this as reflecting a moderate hypotention. Also in his cross-examination Dr. Armstrong reinforced his evidence in-chief that the pattern of brain injury suffered by Jessy is consistent with his conclusions about the injury occurring after 15:33.

[212] The expert testimony in this case is that with every minute that Jessy was left in the uterus after 15:33 when it is most likely when the bradycardia started, his condition worsened. Delivering Jessy even minutes earlier would have spared him some brain damage.

[213] In the 1997 Low study, more than 98% of the babies with a BD of between 12 and 16 mmol/L were spared severe brain damage.[38] Even for babies with a BD higher

[38] Exhibit 30, Table II. There were 58 babies with a BD between 12 and 16 mmol/l and only 1 had severe neurological injury.

than 16 mmol/L, 88% did not suffer a severe brain injury.[39] Almost 60% of the babies with a BD over 16 mmol/L had no neurological injury or only a minor neurological injury. This data supports the evidence of all the expert witnesses.

[214] The fact that the experts agree that the severe bradycardia signaled the onset of severe brain injury after 15:33 fits with the timing evidence of Dr. Perlman and the medical literature. It also fits with the evidence of Dr. Armstrong and Dr. Marrin that the bradycardia signaled the onset of brain ischemia which had not been occurring before that time.

[215] The fact that the severe bradycardia did not commence until 15:33 is compelling clinical evidence that whatever Jessy's BD level was immediately before 15:33 it was not sufficiently high to result in any significant neurological injury to him.

[216] The test for causation at law is a balance of probabilities, not scientific certainty. On the balance of probabilities, considering the totality of the expert evidence offered in this matter, I conclude that Jessy was uninjured at 15:33 when his BD was about 16.8 mmol/L.[40]

Law of Causation:

[217] The traditional legal test for causation in a medical negligence case is the "but for" test and involves an inquiry as to whether the injury would have occurred "but for" the defendant's actions or lack thereof.[41] In this case, simply stated, the analysis under the "but for" test would involve determining whether the delay in delivering Jessy by C-section resulted in any part of his brain injury. Stated another way, one can ask

[39] *Ibid*, Table II. There were 59 babies with a BD greater than 16. Out of that group 52 avoided a severe brain injury, or 88.13%.
[40] The base deficit from the arterial umbilical cord sample was 26.8 mmol/L at 1543 hours. A rate of rise of 1 mmol/L per minute would place the base deficit at 16.8 at 1533 hours. Obviously the base deficit would be lower even a minute or two before then.
[41] Picard, *supra* at note 8, pp. 269-273.

whether earlier delivery of Jessy would have avoided all or some part of his brain injury. If so, then the "but for" test of causation has been met. In the present case, it is clear from all of the experts who testified about causation that all or a substantial part of the brain injury occurred after 15:33. In other words, had Jessy been delivered prior to 15:33, in all likelihood, he would have avoided any significant permanent neurological injury.

[218] A court can determine causation by applying a less scientifically demanding approach known as the "robust and pragmatic" approach.[42] This approach was stated most clearly in the Supreme Court of Canada's decision in *Snell v. Farrell*.[43] The thrust of the approach laid down by Justice Sopinka in *Snell* was that trial courts were not to apply the rules of causation too rigidly. Under the "robust and pragmatic" approach, the evidence does not have to prove that the defendant's negligence was certainly or definitely the cause of the injury, but only probably so. In other words, causation under this approach need not be proven with scientific precision.

[219] Where the "but for" test is "unworkable", courts have adopted the "material contribution" test for determining causation. However, the Supreme Court of Canada has been explicit that the "material contribution" test has not displaced the "but for" test as the primary test for causation in negligence actions.[44]

[220] The special circumstances that permit the use of the "material contribution" test involve two requirements:

> 1) It must be impossible for the plaintiff to prove that the defendant's negligence caused the plaintiff's injury using the "but for" test due to factors outside of the plaintiff's control; and

[42] *Ibid* at pp. 278-282.
[43] (1990), 72 D.L.R. (4th) 289 (S.C.C.).
[44] *Resurface Corp. v. Hanke*, [2007] S.C.J. No. 7 (S.C.C.), para. 22.

2) It must be clear that the defendant breached a duty of care owed to the plaintiffs.[45]

[221] As in *Latin v. Hospital for Sick Children*, this is not a case where it is alleged that multiple harms have caused the injuries. Rather, this case, like *Latin*, is a case of delayed diagnosis and treatment. Therefore, as Justice Lax concluded in *Latin*, the "but for" test applies.[46]

[222] Loss of chance is non-compensable in medical malpractice cases and is not applicable in this case.[47]

Analysis on Causation:

[223] Dr. Braithwaite testified that the objective of fetal surveillance is to allow the obstetrical team to intervene before the fetus is "*in extremis*", which refers to saving the baby before there are permanent neurological consequences. Dr. Braithwaite also testified that if the RTS was completed by 14:32, Jessy would have been delivered by 15:00 assuming a ten minute response time for Dr. Gagnon to attend. Had Jessy been delivered by 15:00, Dr. Braithwaite was of the opinion that he would have been normal. If the RTS was completed at 14:45, then the baby would have been delivered by 15:15. Had Jessy been delivered by 15:15, Dr. Braithwaite's opinion was that he would have been normal in the long term.

[224] Dr. Braithwaite also testified that the complete placental abruption could not have occurred more than 10 or 15 minutes before Jessy was born, or he would not have survived.

[225] Dr. Davies testified that had the RTS been completed at 14:32, Jessy would have been delivered sometime between 14:45 and 14:55. Dr. Davies also testified that

[45] *Ibid*, para. 25.
[46] *Latin v. Hospital for Sick Children*, [2007] O.J. No. 13 (S.C.J.), para. 131.
[47] *Cottrelle v. Gerrard*, [2003] O.J. No. 4194 (ON C.A.), para. 36.

Jessy's brain injury occurred within three to four minutes of the bradycardia noted by Dr. Natale at 15:33. Dr. Davies' opinion was that had Jessy been born before 15:37, in all probability he would have been uninjured and would have had a normal outcome.

[226] As indicated earlier, Dr. Perlman testified that all or substantially all of Jessy's brain injury occurred after 15:33. He based this on the clinical evidence, the imaging and the rate of change of BD as established by the scientific literature.

[227] Dr. Armstrong testified that in his opinion there was no brain injury prior to the bradycardia and the majority of Jessy's irreversible brain injury started after 15:33.

[228] Even the evidence of the defence expert, Dr. Marrin, establishes that the majority of Jessy's brain injury occurred after 15:33. Moreover, the injury was progressive, meaning that every minute counts and delivering Jessy even minutes earlier would have spared him brain damage. Dr. Marrin was only prepared to acknowledge a "possibility" of mild brain injury before 15:33, but a certainty of severe and irreversible brain damage after 15:33. Dr. Marrin also conceded that there would have been some additional brain injury after Jessy was born at 15:43 while attempts were made in the first seven minutes of life to restore Jessy's heart beat.

[229] From the totality of the evidence in this case, it can be said that in all probability Jessy would have been spared brain damage entirely or virtually entirely had he been delivered before 15:33. Further, delivery even minutes before 15:43 would have mitigated his brain damage.

[230] It took Dr. Natale 13 minutes to take a history, assess Jessy with the RTS and deliver Jessy by C-section.

[231] Dr. Gagnon should have been called by Nurse Planques no later than 15:00. The evidence of Dr. Gagnon[48] is that he would have been available within 10 minutes. In other words, Dr. Gagnon would have attended in no more than 10 minutes. Nurse Planques testified that Dr. Gagnon was five minutes (two blocks) away.

[232] Given my finding that the RTS ended at between 14:45 and 14:50, Dr. Gagnon should certainly have been called by Dr. Lackman initially. He would therefore have attended by no later than 14:55 to 15:00 hours. Allowing for an examination similar to that done by Dr. Natale, Jessy could have been delivered by 15:15. Had Nurse Planques arranged for the staff and preparation for the c-section pending Dr. Gagnon's arrival, on the evidence, it is probable that the C-section could have been done even sooner.

[233] Given my findings on Nurse Planques' breach of standard of care in not communicating with Dr. Gagnon herself by 15:00, and allowing the full ten minutes it could have taken Dr. Gagnon to arrive, it is very likely that Jessy would have been delivered by no later than 15:25 or at the very worst by 15:30.

[234] Had Jessy been delivered by no later than 15:30, he would have avoided any serious irreversible neurological damage. It is very likely that had Dr. Gagnon been called by no later than 15:00, he would have performed the emergent C-section by no later than 15:30.

[235] The plaintiffs have met their burden on a balance of probabilities on causation and therefore Nurse Planques' failure to contact Dr. Gagnon by 15:00 has also contributed to the severe brain damage that Jessy suffered.

SEVERAL LIABILITY OF THE DEFENDANTS:

[48] Exhibit 5.

[236] In *M.(J.) v. B.(W.),*[49] the Ontario Court of Appeal squarely considered the issue of whether a Superior Court has jurisdiction to apportion liability against defendants who have entered into a Pierringer-like agreement with the plaintiffs in an action. The court concluded that in the circumstances of the case before it, the Superior court did have the jurisdiction to determine whether, and to what extent, any fault or neglect of the settling defendants caused or contributed to the damages suffered by the plaintiffs even though the settling defendants were not be parties to the action at trial.

[237] This is required out of fairness to non-settling defendants and indeed is necessary to enable the Court to determine the issue of a non-settling defendants' several liability. Justice Cronk wrote:

> "The [plaintiffs'] allegations, if proven, will make [the non-settling Defendants] and the settling defendants concurrent tortfeasors. The liability of the non-settling defendants, however, will be limited to their several liability, and their joint liability with each other, in accordance with the contractual concessions made by the appellants in the Agreements. In these circumstances, it is difficult to conceive how the several liability of the non-settling defendants could properly and justly be determined by the trial judge without regard to the proportionate fault or neglect of the settling defendants.
>
> … [F]airness requires that [the non-settling defendant's] several share of fault or neglect not be determined in a vacuum, without consideration for the several liability of all other proven tortfeasors. Were it otherwise, [the non-settling defendant] could be exposed at trial to the potential risk of being required to pay damages to the [plaintiffs] for part of the settling defendants' several shares of liability…"

Apportion of fault to Dr. Gagnon:

[49] *M.(J.) v. B.(W.),* [2004] 71 OR (3d)171, O.J. No. 2312 (C.A.).

[238] In her closing submissions, counsel for Nurse Planques asked this court to undertake not only to apportion fault against Dr. Lackman but also against Dr. Gagnon, who is no longer a party to this action. By way of Minutes of Settlement dated August 15th, 2006, the action was dismissed against Dr. Gagnon with no admission of liability. I note that Nurse Planques' counsel was a signatory to the minutes.

[239] Ms. Berlach on behalf of Nurse Planques argues that the recently decided Ontario Court of Appeal decision in *Taylor v. Canada (Minister of Health)*[50] permits the apportionment of liability against a non-party like Dr. Gagnon.

[240] Under the *Negligence Act*, R.*S.O. 1990, c. N.1*, where there is more than one tortfeasor and one defendant is required to pay more than its proportionate share of a plaintiff's damages, contribution rights arise. Section 1 of the *Negligence Act* authorizes the court to apportion fault so that among themselves, all tortfeasors will indemnify each other in accordance to their respective degrees of fault. Section 1 provides that

> **1.** Where damages have been caused or contributed to by the fault or neglect of two or more persons, the court shall determine the degree in which each of such persons is at fault or negligent, and, where two or more persons are found at fault or negligent, they are jointly and severally liable to the person suffering loss or damage for such fault or negligence, but as between themselves, in the absence of any contract express or implied, each is liable to make contribution and indemnify each other in the degree in which they are respectively found to be at fault or negligent. R.S.O. 1990, c. N.1, s. 1.

[241] This leads to the question of whether there is jurisdiction to apportion fault against a person who is not party to the action, such as Dr. Gagnon.

[242] In *Martin v. Listowel Memorial Hospital* (2000), 51 O.R. (3d) 384 (C.A.), the Court of Appeal stated that the court does not have jurisdiction. In that case, the infant plaintiff suffered serious brain damage at birth due to the negligence of two doctors, a

nurse and the ambulance attendants. The plaintiff and his family members sued the doctors, the ambulance attendants and the hospital in negligence. They did not sue the nurse, who was added as a third party by the doctors. A pre-trial settlement was reached between the plaintiffs and the doctors, with the result that the doctors did not participate at trial. The terms of the settlement agreement were not disclosed to the other defendants or to the court. The court, in *obiter dicta* considered the scope of s. 1 of the *Negligence Act* at para. 48:

> [48] In our view, the effect of s. 1 of the Act is to define the legal effect of a finding of fault by concurrent wrongdoers. The effect is to change the common law, and impose on concurrent wrongdoer's joint and several liability to the plaintiff. It is the only section of the Act which imposes liability, as opposed to apportioning fault. The section is substantive, not procedural. Therefore, when applying the section to any specific action, it is understood that joint and several liability to the plaintiff can and will attach only to a party defendant, although others who may also have been at fault could potentially have been found jointly and severally liable had they been sued by the plaintiff. Because procedurally the section only affects defendants, under this section the court is to apportion degrees of fault only to defendants. The court must also apportion fault to the other parties, the plaintiff and third parties, not under s. 1 of the Act but rather pursuant to ss. 3 and 4 of the Act, and in accordance with the requirements of the pleadings.

[243] In explaining the policy considerations behind ordinarily applying s. 1 of the *Negligence Act* only to parties, the court stated at paras. 36 and 37:

> [36] The effect of a finding of a degree of fault on a non-party could have significant consequences for the other defendants under this section. If the fault is apportioned only among the parties, then if there is a non-party who may also have been at fault and contributed to the damage, a larger percentage of the whole loss may be attributed to each party, so that the entire loss is divided for indemnity purposes, and no gap is left. But if a portion of the fault were attributed to a non-party, or to a party at fault but with a legal defence such as a limitation

[50] [2009] O.J. No. 2490, 95 O.R. (3d) 561 (ON C.A.).

> defence, the defendants who are liable to the plaintiff would be left with no one from whom they could recover that portion of the claim.
>
> [37] Because it is in the interests of the parties to ensure that everyone potentially liable is joined in the action, in practice, it is therefore most unlikely that any solvent, known person with the potential to be found at fault, would not be joined in the action as a party in some capacity. Section 5 of the Act makes special provision for adding parties, again to ensure that all parties who should be contributing to compensate the plaintiff for the loss are joined in the action which fixes everyone's responsibility.

[244] The Ontario Court of Appeal considered this issue in *M.(J.) v. B.(W.),* wherein Cronk J.A. considered *Martin v. Listowel Memorial Hospital* at length. As in this case, the litigation involved a Pierringer agreement, and she distinguished the decision in *Martin* for a number of reasons. In particular, Cronk J.A. pointed out at para. 47:

> [47] It is significant that findings of negligence and an apportionment of fault were made against the doctors in *Martin*, although they took no part in the trial. It is unclear from the reported decision in *Martin* whether the doctors consented to such an apportionment, notwithstanding the settlement entered into by them with the plaintiffs. The trial judge indicated in *Martin* that, had the nurse been a named defendant, he would have assigned equal fault to each of the two doctors, the nurse and the hospital. That apportionment of degrees of fault was ultimately [page 185] accepted by this court, without any suggestion that the trial judge erred by apportioning liability to the doctors.

[245] Cronk J.A. noted that in *Martin*, the person against whom it was proposed that negligence was to be apportioned had no opportunity to respond directly to the plaintiffs' allegations of negligence against her, or to their claims for relief. This was not the case in *M.(J.) v. B.(W.).*

[246] In *Misko v. Doe* (2007), 87 O.R. (3d) 517, at para. 20, Rosenberg J.A. observed that the excerpts from *Martin* were, in any event, *obiter* and therefore, strictly speaking, not binding. Further, he noted (as did Cronk J.A. in *M.(J.) v. B.(W.)*, *supra*) that

the court in *Martin* accepted the trial judge's apportionment against two doctors who had been sued but had settled before trial and were, therefore, not parties at trial. At para. 22, he went on to explain:

> For similar reasons, *Martin* does not apply here. It would be counterintuitive to refuse to apply s. 1 of the *Negligence Act* when by applying it the defendant would be protected because it would be held liable only for the degree of damages for which it is at fault. As [page 526] in *M. (J.)*, see para. 64, no one objects, or could legitimately object, to the apportionment of liability at trial as against De Bruin. De Bruin has no objection since he is protected by the release. The plaintiff has no objection since he claims from Liberty Mutual only the portion of his damages attributable to the fault or negligence of the second driver. Liberty Mutual can have no objection since it will be responsible only for the damages attributable to the second driver.

[247] In *Taylor*, *supra*, the plaintiff sued Health Canada for injuries suffered as a result of the surgical implantation of a device in her jaw. She sued no other parties, and only sought damages from Health Canada attributable to its proportionate degree of fault. The Attorney General, as representative of Health Canada, brought a third party claim against the dental surgeon who performed the surgery and the hospital where the surgery took place. The claim was dismissed on the ground that the Crown's exposure was limited to damages for which it would have no right to contribution.

[248] Laskin J.A. for the court stated that <u>in an appropriate case</u>, the court had jurisdiction under s. 1 of the *Negligence Act* to apportion fault against a person who was not a party to the action. [my emphasis added] At paras. 27 and 28 he explained:

> [27] Both statutory interpretation and policy support the holdings in *M.(J.) v. B.(W.)* and *Misko*. I think it noteworthy - although the panel in *Martin v. Listowel* did not - that s. 1 of the *Negligence Act* speaks of apportioning fault between "persons", not between "parties." And s. 5 speaks of adding a "person" not already a party to the action. As a matter of statutory interpretation it seems to me the Act itself recognizes that a court has jurisdiction to apportion fault against a

> person not a party to the action. Put differently, nothing in the language of s. 1 precludes a court from doing so.
>
> [28] Interpreting s. 1 of the *Negligence Act* to permit a court to apportion fault against a non-party makes good sense. Interpreting s. 1 in this way promotes the streamlining of litigation, as in the present case, and, as in other cases, the settlement of parts of the litigation.

[249] The current position of the Ontario Court of Appeal is that s. 1 of the *Negligence Act* gives the court jurisdiction to apportion fault against non-parties in appropriate cases, such as against Dr. Lackman who did not participate in this trial because the action was dismissed against her pursuant to the Perrienger agreement.

[250] Dr. Gagnon's situation is quite different. Nurse Planques agreed that he would no longer be a party to this action and on the basis that no liability was admitted. How can this Court apportion liability against a non party who has had no opportunity to defend himself such as in the *Martin* case?

[251] In my view, *Taylor* stands for the proposition that a court must not determine fault in a vacuum. Even if the trial judge apportions fault to the doctor and/or hospital in that case, the fact remains that the plaintiff has limited her damages to the portion of damages that Health Canada will be found at fault for.

[252] In my view, the Court of Appeal decisions reflect the necessity of dealing with Pierringer agreements or when a Plaintiff wishes to limit his or her claims to a certain defendant as in the *Taylor* case. As indicated by Laskin J.A in para. 29:

> Permitting apportionment without insisting that they be parties will mean fewer parties at trial, a shorter trial and reduced costs.

[253] The provision further requires that the "persons" against whom the fault is apportioned are contributors to the damages.

[254] Even if I were permitted to entertain an apportionment against Dr. Gagnon, there is a paucity of evidence against him. There is no evidence that he was called at all before 15:20 to 15:25. Three minutes later, Dr. Natale was on deck getting the work done.

[255] For these reasons, no liability is or can be apportioned against Dr. Gagnon.

Apportionment of liability between Dr. Lackman and Nurse Planques:

[256] With respect to Dr. Lackman's responsibility, clearly she failed to meet the standard by failing to communicate with Dr. Gagnon who was her supervisor on-call immediately after completing her assessment of Ms. Milne. Given that her primary differential diagnosis was placental abruption, she ought to have acted with dispatch. She should not have waited for blood work. She should not have chosen to observe an elective C-section on patient X.

[257] Plaintiffs' counsel argues that Nurse Planques ought to be found liable for at least 50%, if not more. Defendant's counsel argues that, if found liable, then Nurse Planques ought to be found liable at the most for 10%.

[258] Nurse Planques' failure is not the same as Dr. Lackman's breach. Dr. Lackman's breach is striking because that she knew that Ms. Milne was likely suffering from a placental abruption and she left her to observe a C-section without communicating with Dr. Gagnon. Her breach is much more egregious than the lack of action by Nurse Planques as I have determined.

[259] For these reasons, I hold that Dr. Lackman bears greater responsibility. Nurse Planques also failed in her duty of care as indicated in these reasons. However, her failures carry much less responsibility.

[260] In my view, Dr. Lackman bears at least twice as much responsibility than Nurse Planques. Accordingly I apportion fault at 67% to Dr. Lackman and 33% to Nurse Planques.

Damages:

[261] As indicated at the outset, the parties have agreed on the quantum of damages. This court is prepared to hear submissions on the damage settlement given the requirement for an infant settlement. The hearing shall be scheduled by the trial coordinator at a time agreeable to all parties.

[262] The issue of costs can be entertained at that hearing if necessary.

"Justice J. N. Morissette"
Justice J. N. Morissette

Released: September 25, 2009