COURT OF APPEAL FOR BRITISH COLUMBIA

Citation: *Hanson-Tasker v. Ewart,*
2023 BCCA 463

Date: 20231208
Dockets: CA48233

Between:

Kyrcee Hanson-Tasker

Appellant
(Plaintiff)

And

Dr. D. Brian Ewart and Dr. Sheila Ewart

Respondents
(Defendants)

Before: The Honourable Justice Mackenzie
The Honourable Mr. Justice Fitch
The Honourable Mr. Justice Abrioux

On appeal from: An order of the Supreme Court of British Columbia, dated March 16, 2022 (*Hanson-Tasker v. Ewart*, 2022 BCSC 432, Vancouver Docket S162160).

Counsel for the Appellant:	C. E. Hunter, K.C. C.V. van Wiltenburg B.E. Webster-Evans
Counsel for the Respondents:	G.P. Brown, K.C. J.A. Morris
Place and Date of Hearing:	Vancouver, British Columbia May 25, 2023
Place and Date of Judgment:	Vancouver, British Columbia December 8, 2023

Written Reasons by:
The Honourable Mr. Justice Fitch

Concurred in by:
The Honourable Justice Mackenzie
The Honourable Mr. Justice Abrioux

Summary:

The appellant challenges the dismissal of her negligence claim against the respondent physicians. The trial judge found the respondents breached the standard of care by failing to monitor and test the appellant's bilirubin levels in her first days of life, but that the breach did not cause or contribute to the brain damage she sustained as a result of a later arising, unpredictable and acute, oxidative hemolytic event of independent origin. The appellant alleges that the trial judge: (1) erred in law by failing to consider drawing an adverse inference of causation against the respondents in light of the evidentiary gap attributable to the respondents' negligence; and (2) erred by misapprehending evidence that was material to the causation analysis. Held: Appeal dismissed. The trial judge implicitly declined to draw the adverse causal inference against the respondents in light of the evidence before him. Such an exercise of discretion is consistent with the application of the governing legal framework. The trial judge also made no palpable and overriding error in his understanding of the causation evidence. The judge was entitled to accept the respondents' biphasic theory of causation, considering the untenability of the progressive theory adduced by the appellant.

Table of Contents

Reasons for Judgment of the Honourable Mr. Justice Fitch:

I. Introduction

[1] This is an appeal from the dismissal of a medical negligence claim brought by the appellant, Kyrcee Hanson-Tasker ("Kyrcee"), against the respondents, Dr. Brian Ewart and Dr. Sheila Ewart, the family physicians charged with her care in the days after her birth. The negligence of the respondents was alleged to have caused Kyrcee brain damage resulting from untreated and dangerously high levels of bilirubin in her blood.

[2] In detailed reasons for judgment, indexed as 2022 BCSC 432 ("RFJ"), the trial judge found the respondents to have breached the standard of care, but that their negligence did not cause or contribute to Kyrcee's injuries. In the result, the claim was dismissed.

[3] The appellant's grounds of appeal take aim at the judge's causation analysis. First, relying primarily on *Snell v. Farrell*, 1990 CanLII 70 (SCC), [1990] 2 S.C.R. 311 and *Benhaim v. St-Germain*, 2016 SCC 48, the appellant submits the judge erred by failing to consider whether it was appropriate to draw an adverse inference of causation in circumstances where causal uncertainty was attributable to the respondents' negligence. Second, the appellant argues that the trial judge misapprehended evidence material to the issue of causation. The appellant submits the appeal should be allowed and judgment entered against the respondent physicians or, in the alternative, a new trial ordered.

II. Background

[4] As the case is factually complex, some additional background is necessary to put the grounds of appeal in context.

[5] Kyrcee was born on July 29, 1996, slightly pre-term. She was discharged from the hospital two days later. She suffered life-altering injuries attributable to neurotoxic levels of bilirubin discovered upon her re-admission to the hospital on August 6, 1996.

[6] In simple terms, bilirubin is a yellowish chemical by-product of the natural breakdown of red blood cells. The liver processes bilirubin from the bloodstream and releases it into the intestinal tract to be excreted. Newborns typically have increased red blood cell turnover and still-maturing livers that struggle to process bilirubin quickly enough, causing an increase in serum bilirubin concentration. In some cases, this imbalance between an infant's bilirubin production and the ability to eliminate it from the bloodstream results in hyperbilirubinemia, which manifests as a yellowing of the skin and eyes, commonly known as jaundice.

[7] Physiologic jaundice is a normal occurrence in many infants due to the increased breakdown of red blood cells in the first week after birth. It develops in approximately 50% of term babies and is somewhat more common in pre-term neonates. Jaundice serves as only a very rough indicator of bilirubin levels and typically becomes apparent at bilirubin levels of 85–120 µmol per litre. As bilirubin levels rise, an infant's jaundice will deepen in colour and spread from the head down the body. Physiologic jaundice usually resolves on its own as the infant's liver matures and becomes more effective in processing bilirubin. In pre-term infants such as Kyrcee, it typically peaks and plateaus within the first five to seven days of life and poses no harm.

[8] In rare instances, a significant imbalance in an infant's bilirubin production and elimination can cause the buildup of bilirubin in the bloodstream to cross the blood-brain barrier and cause damage to the infant's brain, resulting in acute bilirubin encephalopathy. The symptoms of acute bilirubin encephalopathy include lethargy, refusal to feed, high-pitched crying, severe back arching, seizures and apnea. This acute condition can progress to chronic bilirubin encephalopathy, characterized by irreversible brain damage—or kernicterus—as a result of the bilirubin neurotoxicity.

[9] Kyrcee's bilirubin level was measured on July 31 before she was discharged from the hospital. It was not at a level requiring phototherapy—the first-line treatment for reducing hyperbilirubinemia in infants.

[10] She was examined by Dr. Brian Ewart on August 1, 1996—three days after her birth and the day after her discharge from hospital. While Dr. Ewart noted Kyrcee to be “icteric” (jaundiced), no future appointment was scheduled to monitor her condition, and no follow-up bilirubin test was ordered.

[11] The trial judge found that, in light of Kyrcee’s elevated risk profile for hyperbilirubinemia and evident jaundice about 38 hours after her birth, the respondents failed to meet the standard of care expected of them by, among other things, failing to monitor her condition in the days following her birth and discharge from the hospital to assess the need for a follow-up bilirubin test.

[12] With this finding made, the issue at trial became whether the respondents’ negligence caused Kyrcee’s injuries.

[13] Kyrcee’s bilirubin level was not measured again until her re-admission to the hospital on August 6, when she was in distress and displaying classic symptoms of acute bilirubin encephalopathy. Sadly, Kyrcee’s high bilirubin levels led to the development of kernicterus and, despite prompt medical intervention, the injuries she suffered could neither be avoided nor reversed.

[14] Because she was not tested after July 31, Kyrcee’s bilirubin levels between August 1 and August 5 are unknown. In addition, the etiology of the hyperbilirubinemia Kyrcee experienced in the days following her birth could not be definitively determined.

[15] On causation, the parties, through their respective expert witnesses, presented two competing theories concerning the likely trajectory of Kyrcee’s rising bilirubin levels between July 29 and August 6 and why she developed acute bilirubin encephalopathy.

[16] Kyrcee’s primary expert witness on causation, Dr. Kaplan, posited a pathologically abnormal, progressive, and essentially linear increase in her bilirubin levels from birth to her re-admission to the hospital eight days later. He opined that, had appropriate action been taken following Kyrcee’s discharge from the hospital,

the severe hyperbilirubinemia and resultant bilirubin neurotoxicity would have been avoided.

[17] The respondents' expert witnesses on causation, Dr. Boulton and Dr. Van Aerde, opined that it was far more likely, if not certain, that there was a biphasic trajectory of bilirubin in Kyrcee's system, not a linear one. In their view, while there was an initial, normal and physiological rise in bilirubin that plateaued in the days following Kyrcee's discharge from the hospital, this was followed by a massive and unpredictable oxidative hemolytic event that likely began about 24 hours before her re-admission to the hospital on August 6. This event caused the rapid destruction of Kyrcee's red blood cells resulting in acute anemia, severely elevated bilirubin levels and, ultimately, brain damage.

[18] Drs. Boulton and Van Aerde agreed that, even if a follow-up bilirubin test had been ordered when Dr. Brian Ewart examined Kyrcee the day after her discharge on August 1, it would not have changed the outcome. Her severe hyperbilirubinemia was due to an acute oxidative hemolytic event of independent origin that arose several days after Kyrcee's appointment with Dr. Brian Ewart. In other words, they opined that Kyrcee's injuries were inevitable and would have been sustained without the defendants' negligence.

[19] The trial judge accepted the evidence of the respondents' expert witnesses that Kyrcee's bilirubin levels were the product of unrelated biphasic processes—one consistent with a predictable, physiological condition common to newborns, and the other attributable to an unrelated and wholly unpredictable oxidative event that caused Kyrcee's bilirubin levels to spike on August 5.

[20] The judge found as a fact that Kyrcee failed to demonstrate, on a balance of probabilities, that a follow-up bilirubin test ordered by the respondents before August 5 would have prevented her injuries. He accepted the evidence of Dr. Boulton that the sudden, oxidative hemolytic event that occurred shortly before her admission to hospital was an independent event—one so extreme that it would have overwhelmed the ability of any liver to process it.

[21] In drawing this inference, the judge relied primarily on two considerations.

[22] First, if Dr. Kaplan's progressive, essentially linear projection of Kyrcee's bilirubin levels was correct, Kyrcee would likely have been displaying the prodrome (or early symptoms) associated with toxic hyperbilirubinemia well before those symptoms became apparent to her mother on the morning of August 6. Apart from increasing jaundice, there was no evidence that Kyrcee had displayed any of the symptoms typically associated with toxic hyperbilirubinemia until the morning of August 6. The judge referred to this as "a glaring hole in the theory of the plaintiff's case."

[23] Second, the judge accepted the evidence of the respondents' experts that the sharp drop in Kyrcee's hemoglobin between 11:15 a.m. and 8:54 p.m. on August 6 was objective evidence of an acute hemolytic event that could not have been occurring over the more prolonged period of time posited by Dr. Kaplan's linear progression theory.

[24] With this overview in mind, I will proceed to address the evidence relevant to causation before summarizing the findings of fact made by the trial judge.

III. Evidence Relating to Causation

1. Kyrcee's Birth, Discharge and Re-admission to Hospital

[25] Kyrcee was born after 36 weeks and 3 days gestation. As 37 weeks is considered full-term, Kyrcee was born four days pre-term. From birth, she was exclusively breast-fed by her mother, Nadine Dray ("Nadine"). Both of these factors elevated Kyrcee's risk for hyperbilirubinemia.

[26] Dr. Pegg, a pediatrician, attended to Kyrcee in hospital after her birth. She had no recollection of her interactions with Kyrcee, which occurred more than 25 years before she gave evidence at trial. Dr. Pegg made a clinical note on July 31, when Kyrcee was about 37 hours old, that she had "slight jaundice". Dr. Pegg testified that her observation of "slight jaundice" meant there was a yellow hue around Kyrcee's face.

[27] Dr. Pegg ordered a serum bilirubin test. A blood sample was taken at 9:20 a.m. on July 31. Kyrcee's bilirubin level was reported to be 165 µmol/l, under the threshold for the initiation of phototherapy. Kyrcee was discharged from the hospital on July 31 at 2:15 p.m., about 42 hours after her birth. Dr. Pegg arranged for follow-up the next day with Kyrcee's family physician, Dr. Brian Ewart.

[28] Shari Pereira ("Shari"), Nadine's sister and Kyrcee's aunt, visited them on July 31 and noted that Kyrcee was "a little bit yellow." The trial judge found Shari to be a credible witness and accepted her evidence regarding the daily, progressive deepening of Kyrcee's jaundice from July 31 to either August 4 or 5, the last day Shari visited before Kyrcee was re-admitted to the hospital.

[29] On August 1, Kyrcee was examined by Dr. Brian Ewart. He had no recollection of the consultation, but reported in his clinical notes that Kyrcee was "icteric". Dr. Brian Ewart gave conflicting evidence about the extent to which Kyrcee was jaundiced on examination. During his examination for discovery (read in at trial) he was asked whether the jaundice extended over Kyrcee's trunk and extremities. He replied, "[i]t would have been all over, yes." At trial, he testified that he does not know why he said this in examination for discovery. He said he was well aware of the significance of the progression of jaundice to a baby's extremities, and if Kyrcee's trunk and extremities were yellow, he would have ordered a follow-up bilirubin test and likely initiated phototherapy. He testified that he would have discussed jaundice with Nadine, advised her to monitor it and to go to the hospital if it worsened. He did not, however, order ongoing monitoring or further bilirubin testing.

[30] On Friday, August 2—the day before a long weekend—Edna McLellan, a public health nurse, conducted a home visit with Nadine and Kyrcee. She testified that Kyrcee's jaundice was limited to her head, eyes and the very upper part of her chest. It was not put to Ms. McLellan that Kyrcee's jaundice was all over her body or that it extended to her feet. She testified that she would have reviewed with Nadine the "red flags" to watch for, including the progression of Kyrcee's jaundice.

Ms. McLellan testified that Kyrcee looked fine and that she had no concerns about her at the time of the visit. Nevertheless, she left a message for Dr. Sheila Ewart advising her that Kyrcee was jaundiced.

[31] Dr. Sheila Ewart testified she had no recollection of receiving this phone call from Ms. McLellan. She said it was her usual practice to make a clinical note if the public health nurse had a concern, in which case she would have arranged for the baby to be brought into the clinic. The judge concluded that Ms. McLellan likely had concerns about Kyrcee's jaundice getting worse over the long weekend, and expressed her concerns in the message left with Dr. Sheila Ewart.

[32] Shari saw Kyrcee again on August 2, after Ms. McLellan's visit. She described Kyrcee's face and hands as being "more yellow" than they had been the previous day. Shari gave no evidence that the jaundice extended to Kyrcee's feet on this or any subsequent date, although it is unclear from the evidence whether Shari would have been in a position to make this observation.

[33] Nadine testified that on August 2, 3, 4 and 5, Kyrcee fed well and was alert. In Nadine's mind, Kyrcee was perfectly healthy apart from her jaundice. A sampling of the evidence she gave on this issue at trial is reproduced below:

> Q. During that long weekend did you continue to breastfeed your daughter?
>
> A. I did.
>
> Q. And during that long weekend up until the Monday night, did she continue to wake you up at night in order to be fed?
>
> A. Yes.
>
> Q. What were your observations of her when she was awake? How did she seem to you?
>
> A. She seemed alert, happy, healthy. Seemed like a regular baby.
>
> Q. I want to now take you to what happened on Tuesday. Prior to waking up on Tuesday morning, do you recall when the last time was that you fed her?
>
> A. Around midnight.
>
> Q. Okay. From your observations or recollections of that breastfeeding, how did that last feeding go?

A. In my opinion it went fine.

…

Q. And at the time of the public health nurse's visit on August 2nd you had no concerns at all about Kyrcee; correct?

A. I thought I was doing perfectly fine.

Q. I want to make sure the evidence is clear on that. My question was you had no concerns at all about Kyrcee at that time; you agree with that?

A. I agree with that.

Q. The next day is Saturday, August 3rd; okay?

A. Yes.

Q. Kyrcee was eating, sleeping and alert that day?

A. She was.

…

Q. You had no concerns at all about Kyrcee on August 3rd; correct?

A. No concerns.

…

Q. The next day is Sunday, August 4th; okay?

A. Okay.

Q. Kyrcee was exactly the same on Sunday, August 4th as she was on Saturday August 3rd?

A. Yes.

Q. Again, she was feeding every three to four hours?

A. Yes.

Q. She was alert?

A. Yes.

Q. She wasn't sleeping any more on Sunday than she had on any previous days?

A. No.

Q. Next day is Monday, August 5th; okay?

A. Okay.

Q. Kyrcee was exactly the same on Monday, August 5th as she was on Saturday, August 3rd and Sunday, August 4th?

A. Yes.

Q. She was feeding every three to four hours?

A. Yes.

Q. She was alert?

A. Yes.

…

Q. She was as alert as she had been on previous days?

A. Yes.

…

Q. The next day is Tuesday, August 6th; okay?

A. Yeah.

Q. And that's the day when things changed with Kyrcee?

A. That's the day things changed with Kyrcee.

Q. In terms of the timeline, you fed Kyrcee late at night either on August 5th or early in the morning of August 6th around midnight?

A. Yes.

Q. And when you fed Kyrcee at that time, you had no difficulty feeding her, did you?

A. No.

Q. It was a normal feed?

A. It was a normal feed from what I remember.

…

Q. And so when Kyrcee woke up on the morning of August 6th, she was sleepy, she was limp, and she wasn't feeding; correct?

A. Correct.

Q. And that was the first time that you observed any of those issues with Kyrcee?

A. First time she ever showed signs of those.

[34] Shari visited Nadine and Kyrcee on August 3. She was alarmed at the progression of the jaundice and described Kyrcee as looking like a "f---ing banana."

[35] Shari briefly visited again on either August 4 or 5 and recalled that Kyrcee's skin colour had worsened to a brighter orange/yellow shade. She acknowledged in cross-examination, however, that apart from the progressive jaundice, she had no recollection of observing anything unusual about Kyrcee's behaviour between July 31 and her last visit.

[36] On August 5, a family friend, Carol Perry, visited Nadine and Kyrcee. She testified that Kyrcee's skin colour was "a very dark yellow", but that Kyrcee seemed to be alert, moving, happy and healthy.

[37] Nadine fed Kyrcee at approximately midnight on the night of August 5. She seemed to be well and acting normally when put to bed. The next morning, Kyrcee was limp and disinterested in feeding. Nadine immediately took Kyrcee back to Dr. Brian Ewart's office, who arranged for her to see Dr. Pegg at the Kitimat hospital.

[38] Dr. Pegg again took charge of Kyrcee's treatment. Nadine told Dr. Pegg that Kyrcee had been breast-feeding well from discharge until re-admission to the hospital on August 6. She also told Dr. Pegg that the jaundice seemed to have improved after Kyrcee's discharge from the hospital, but that it had intensified over the past 72 hours and most markedly over the past 24 hours. Nadine also reported that Kyrcee was arching her back, had a high-pitched weak cry, and would not feed on the morning of August 6.

[39] Testing revealed a toxic level of bilirubin in Kyrcee's blood—766 µmol/l. Dr. Pegg provided intravenous fluids for Kyrcee and began phototherapy. Later that day, Kyrcee was transported to B.C. Children's Hospital in Vancouver for exchange transfusion and further investigation and management. Between 11:15 a.m. and 8:45 p.m., Kyrcee's hemoglobin dropped dramatically from 86 g/l to 53 g/l.

[40] The sad reality in this case is that Kyrcee now lives with athetoid cerebral palsy, hearing impairment and other developmental disorders.

2. The Expert Evidence

[41] Dr. Fazal, a pediatrician, was called by the appellant and qualified to give opinion evidence on the standard of care expected of a reasonably prudent medical practitioner providing care to a newborn in 1996, the likely trajectory of Kyrcee's bilirubin level, and the cause of her injuries.

[42] Consistent with the evidence of other experts, Dr. Fazal testified that the typical prodrome for acute bilirubin encephalopathy includes lethargy reflecting neurological impairment, loss of interest in feeding, a high-pitched cry and, as bilirubin levels increase, back arching. Dr. Fazal thought that some of these symptoms would become manifest in the severe to critical range of 340–420 µmol/l. He opined that bilirubin encephalopathy, or kernicterus, is likely at serum bilirubin levels greater than 510 µmol/l. He agreed that at least some of the prodrome of kernicterus would likely be present before an infant's serum bilirubin level reached 500 µmol/l. While there are no established numbers at which the prodrome of kernicterus will become evident in a particular case, he agreed that seizures would be likely if the bilirubin level was in the range of 650 µmol/l. At this level, he would also expect some infants to stop breathing.

[43] Dr. Kaplan, a neonatologist, was called by the appellant. He was qualified to provide opinion evidence on the standard of care, the likely progression of Kyrcee's bilirubin level, the likely etiology of her hyperbilirubinemia, and the cause of her injuries. His evidence was accurately summarized by the trial judge:

> [126] Dr. Kaplan opined that Kyrcee's bilirubin level likely followed an approximately linear path after her birth (when her bilirubin level would have been approximately 25 mol/l) to the level of 165 mol/l – as tested approximately 37 hours after birth – and increased constantly and progressively over the ensuring days until August 6, 1996, when the level was measured at 766 mol/l. Dr. Kaplan estimated that, assuming such a linear progression, Kyrcee's bilirubin level would have been approximately 304 mol/l at the time of Dr. Brian Ewart's examination on August 1 and that Kyrcee's skin would have looked "decidedly yellow". Dr. Kaplan opined that, at the time of Ms. McLellan's home visit on August 2, Kyrcee's bilirubin level would have been approximately 393 mol/l and her skin colour would have been an orange-yellow, but she still would have been within the treatment range.
>
> [127] Dr. Kaplan stated that, had Kyrcee's levels of bilirubin been monitored and re-tested before they reached a neurotoxic level, she would have been treated in a routine way - - with phototherapy or, if that failed, an exchange transfusion -- and that she would not have suffered the injuries she did.
>
> [128] Dr. Kaplan noted in his response report that during hemolysis, red blood cells break down releasing heme, which is subsequently metabolized to bilirubin. During the early stages of a hemolytic episode, the hemoglobin value frequently remains stable and decreases only in the later stages, when there is redistribution of fluid and plasma with resultant hemodilution. He

> opined that, therefore, Kyrcee's low hemoglobin level at the time of re-admission to hospital on August 6 is most likely indicative of a more prolonged hemolytic process, rather than an acute event.
>
> [129] Dr. Kaplan noted studies which have shown that reticulocyte counts and blood smears are not reliable indicators of hemolysis in neonates because of the overlap between hemolytic and non-hemolytic states.
>
> [130] On cross-examination, Dr. Kaplan disagreed that the development of extremely high hyperbilirubinemia and subsequent kernicterus in Kyrcee was due to an episode of late onset, massive acute hemolysis. He opined that the theory that Kyrcee suffered a biphasic, exponential bilirubin trajectory would be plausible if Kyrcee had been found to have a G6PD deficiency. G6PD is an enzyme that plays an important role in protecting red blood cells from oxidants. A newborn with a G6PD deficiency is at greater risk of hemolysis. Dr. Kaplan opined that late onset hemolysis and hyperbilirubinemia in Kyrcee's case is unlikely, because neither a G6PD deficiency nor any other kind of oxidant known to cause hemolysis (such as pyruvate kinase deficiency or hereditary spherocytosis) was confirmed to have been present in her case. … It was his opinion that, in Kyrcee's case, it is likely that the breakdown of the red blood cells was an ongoing process and that her hemoglobin did not suddenly drop shortly before August 6.
>
> [131] Dr. Kaplan agreed that not only can oxidants form in the body, they are also present in the environment. Examples of products containing oxidants that can be present in a home include mothballs, some medications, and possibly cleaning fluids, powders, and shampoo. He agreed that exposure to an environmental oxidant can trigger an acute hemolysis in a newborn with a G6PD deficiency, causing a massive increase in serum bilirubin which, in many cases, is unpredictable.

[44] Dr. Kaplan prepared a graph plotting what he considered to be the likely progression of Kyrcee's bilirubin levels from the date of her birth to August 6.

[45] Accepting the information in Dr. Kaplan's graph, on August 2—the day of Nurse McLellan's house call—Kyrcee's bilirubin level would have been approximately 393 µmol/l. Dr. Fazal cautioned that serum bilirubin levels are not always a good predictor of the risk of bilirubin encephalopathy, and there is no established number at which the typical prodrome of kernicterus will appear. As he put it, "[e]ach baby would be different." He agreed, however, that it is likely symptoms will become more manifest as bilirubin levels increase. He opined that at least some of the prodrome of kernicterus would begin to appear in the severe to critical range of 340–420 µmol/l. It is noteworthy that none of the symptoms identified by Dr. Fazal were noted by Nadine, Shari or Nurse McLellan that day.

[46] Accepting Dr. Kaplan's progressive theory, Kyrcee's bilirubin level would have exceeded 500 µmol/l on August 3. Dr. Fazal testified that the behavioural prodrome of kernicterus would likely be present at this level. Other than worsening jaundice, neither Nadine nor Shari made any unusual behavioural observations about Kyrcee that day.

[47] On August 5, Kyrcee's bilirubin level would have been between 600–650 µmol/l. Dr. Fazal testified that seizures would be likely at this point and that "I think you would start to see a child who stops breathing; apnea." Nadine testified that Kyrcee behaved normally on August 5. In addition, Ms. Perry spent an hour with Nadine and Kyrcee that day. Although Kyrcee was noted to be jaundiced, she was alert and moving. She was not limp or fussing. Ms. Perry described it as "a happy visit."

[48] According to Dr. Kaplan, Kyrcee's bilirubin levels would have been about 680 µmol/l by the evening of August 5. And yet, when Kyrcee was fed and put to bed at midnight, Nadine testified there was nothing unusual about her behaviour.

[49] In cross-examination, Dr. Kaplan confirmed that his opinion—that Kyrcee would not have suffered the injuries she did had she been monitored and retested on August 1 or 2—was based on his theory that her bilirubin levels steadily progressed in a generally linear fashion from birth.

[50] Dr. Boulton, a neonatologist, was called by the respondents and qualified to give opinion evidence on the assessment, management and causes of neonatal jaundice, hyperbilirubinemia and kernicterus.

[51] All of the respondents' experts were asked to accept as an assumed fact that Kyrcee had mild jaundice when she was examined by Dr. Brian Ewart on August 1.

[52] In her primary report, Dr. Boulton expressed the view that Kyrcee's hemolytic condition that caused the severe hyperbilirubinemia was not present at birth. Had the hemolytic condition been present, her bilirubin level would have risen quickly and been far higher than 165 µmol/l on July 31.

[53] In her responsive report, Dr. Boulton disagreed with Dr. Kaplan's opinion that Kyrcee's bilirubin trajectory was progressive from birth and likely linear. Instead, she opined that Kyrcee's severe hyperbilirubinemia was "clearly related" to an unpredictable and acute hemolytic event:

> In suggesting that the trajectory of the bilirubin was linear, Dr. Kaplan seems to make the assumption that the elevation of bilirubin was due to ongoing hemolysis from birth. As stated in my original report, babies who have ongoing hemolysis from birth have far higher levels of bilirubin than was measured in Kyrcee at 38 hours of age. In Kyrcee's case, it is likely that there was a biphasic trajectory of bilirubin rather than a linear one. There was an initial rise in bilirubin that may well have plateaued over the initial days following discharge followed by a hemolytic event that caused acute destruction of her red blood cells resulting in severe anemia and severely elevated bilirubin levels.

[54] Dr. Boulton also disagreed with Dr. Kaplan's opinion that, had Kyrcee's serum bilirubin been taken on August 1 or 2, the hyperbilirubinemia would have been recognized and successfully treated before it reached neurotoxic levels. She expressed the view that Kyrcee's acute hemolytic event and the associated spike in her bilirubin levels likely occurred much later than August 2, and closer to her re-admission to the hospital on August 6.

[55] Dr. Boulton further disagreed with Dr. Kaplan's opinion that Kyrcee's low hemoglobin on August 6 was likely a sign of a more prolonged hemolytic process. In her view, the drop in Kyrcee's hemoglobin during the first hours after her re-admission to hospital suggests that she was then in the throes of a very acute hemolytic event that could not have been going on for more than about 24 hours.

[56] Finally, Dr. Boulton disagreed with Dr. Kaplan's opinion that, had Kyrcee's bilirubin level been tested on August 1, the unfortunate outcome in this case would have been avoided. She expressed the view that a follow-up bilirubin test on August 1 would not have changed the outcome, since the severe hyperbilirubinemia Kyrcee experienced was due to an independent and later-arising, acute hemolytic event.

[57] Like Dr. Boulton, Dr. Van Aerde, also a neonatologist called by the respondents, did not agree with Dr. Kaplan's progressive, essentially linear trajectory theory because "the trajectory of neonatal bilirubin metabolism is not linear in…near-term neonates; it increases in the first 3 days, the rate of increase slows down between days 3 and 5, and stops increasing altogether by 5 days of age."

[58] In addition, in Dr. Van Aerde's opinion, Kyrcee's rapidly decreasing hemoglobin on August 6 indicated a "massive acute hemolysis which could not have been going on for 8 days at that rate with a normal starting hemoglobin level of about 140 g/L for the average newborn; the acute hemolysis must have started toward the end of the first week of this baby's life" (emphasis in original). Dr. Van Aerde testified that the decrease in Kyrcee's hemoglobin was "the strongest evidence we have" that an acute, oxidative hemolytic event began no earlier than the morning of August 5.

[59] Apart from the rapid drop in Kyrcee's hemoglobin on August 6, Dr. Van Aerde identified other objective signs supporting the sudden onset of an acute and massive hemolysis. Most significantly, a blood smear taken from Kyrcee on August 6 showed fragmented cells indicative of the type of massive hemolysis seen when red blood cells are broken up fast.

[60] In his view, and assuming that Kyrcee had a normal hemoglobin level at birth, the acute hemolytic event would likely have started at about lunchtime on August 5, after which clinical symptoms, including a progressive refusal to eat, would have emerged. In cross-examination, Dr. Van Aerde conceded that there was "a small chance" that the acute, oxidative hemolytic event Kyrcee experienced started as early as August 4.

[61] In Dr. Van Aerde's opinion, acute oxidative hemolysis was the cause of Kyrcee's injuries. Her serum bilirubin levels probably followed the typical curvilinear path for jaundiced newborns but, instead of reaching a plateau on day five, spiked up at a very fast rate, likely on August 5. Had a bilirubin test been taken on August 1, the outcome of this unpredictable event would not have been altered.

[62] Dr. Manhas, a neonatologist, was called by the defendant nurses who are not parties to this appeal. The evidence he gave relevant to causation was summarized by the judge:

> [177] Dr. Manhas agreed with Drs. Boulton and van Aerde that the rapid decrease in Kyrcee's hemoglobin on August 6, 1996 from 86 g/l to 53 g/l is suggestive of sudden oxidative or hemolytic stress that caused her red blood cells to burst and release hemoglobin, which was broken down into heme and then bilirubin, resulting in a rare, unexplained oxidative hemolysis and severe hyperbilirubinemia. Dr. Manhas commented that such an occurrence not associated with breastfeeding or prematurity and could not have been predicted.
>
> [178] Based upon Nadine's reports, the clinical records that Kyrcee was generally looking and feeding well and that Kyrcee's jaundice worsened over the three days prior to August 6 (and most notably within the past 24 hours prior to her hospital admission), Dr. Manhas opined that this sudden hemolytic event occurred in a short time frame prior to August 6. Otherwise, Kyrcee would have shown symptoms of lethargy or lack of activity and interest in feeding at a much earlier time. However, on cross-examination, he agreed that if Kyrcee's jaundice was worsening over the course of the 72 hours preceding August 6, it could have been indicative of a continuing hemolytic event.

[63] Dr. Manhas disagreed with Dr. Kaplan's opinion that Kyrcee's severe anemia (low hemoglobin) on August 6 was indicative of a more prolonged hemolytic process preceding her re-admission. In Dr. Manhas' view, Dr. Kaplan's suggestion that Kyrcee's anemia occurred earlier but only became evident in the later stages when there was a "redistribution of fluid and plasma with resultant hemodilution" is scientifically incorrect because Dr. Kaplan was mixing up anemia from blood loss—which Kyrcee did not experience—with anemia from hemolysis.

[64] The appellant emphasizes evidence given by Dr. Manhas in cross-examination in support of her position that he would not have endorsed the respondents' biphasic theory so readily if the factual assumption he was asked to make—that Kyrcee was mildly jaundiced on August 1 and 2—was not correct. This factual assumption was not established by the evidence at trial. I reproduce below the evidence upon which the appellant places particular reliance:

> Q. So if you assume that -- if I ask you to assume…for the purpose of this that the baby was not -- did not have mild jaundice on August 1st and did not have mild jaundice on August 2nd, in fact, the jaundice

was much more significant than mild on August 1st and 2nd, then the first facts and assumption in that opinion is wrong; correct? Your opinion would have to change?

A. If the baby looked severely jaundiced and had other signs and symptoms, yes, I would change that opinion.

Q. Okay. But just for that paragraph alone, if the second line said both agreed that the -- she was significantly jaundiced, significantly icteric. If that's what --

A. Yeah.

Q. -- the proposition I'm putting to you was, that the assumption was both agreed she was significantly icteric, then you would agree with me that the worsening that occurred at the 72-hour mark was just a continuation of the worsening that was already under way?

A. More likely than not that would be correct, yes.

[Emphasis added.]

3. Reasons for Judgment (2022 BCSC 432)

[65] The judge accepted Shari's evidence regarding the daily, progressive, deepening of Kyrcee's jaundice from July 31. He found her evidence to be "consistent with the typical trajectory of jaundice in preterm babies."

[66] The judge also accepted Nadine's evidence that, up to and including Kyrcee's feed around midnight on the evening of August 5, she was active, alert and seemed fine.

[67] The judge noted that Dr. Kaplan's opinion regarding the trajectory of Kyrcee's bilirubin was in stark contrast to the opinions offered by Drs. Boulton, Van Aerde and Manhas. More importantly, he found Dr. Kaplan's opinion to be at odds with Nadine's uncontroverted evidence regarding Kyrcee's behavioural presentation prior to August 6.

[68] The judge found that Drs. Brian and Sheila Ewart failed to meet the standard of care of a reasonably prudent family physician in 1996. A reasonably prudent family physician would have recognized Kyrcee's increased risk factors and the need for further follow-up. The judge concluded that Nurse McLellen called Dr. Sheila Ewart because she was concerned about Kyrcee's jaundice and wanted

to ensure that her family doctor was apprised of the situation. He found that a reasonably prudent family physician in Dr. Brian Ewart's circumstances would have communicated directly with Nurse McLellan on August 2 and arranged for Kyrcee's assessment over the long weekend, including a follow-up bilirubin test. He came to a similar conclusion with respect to Dr. Sheila Ewart. He held that a reasonably prudent family physician in Dr. Sheila Ewart's situation would have taken steps to ascertain the extent of the jaundice noted by Nurse McLellan on August 2, and obtained information about whether it had worsened. Instead, "Dr. Sheila Ewart did nothing, other than possibly passed [*sic*] the information to … Dr. Brian Ewart, who in turn did nothing."

[69] In his causation analysis, the judge did not expressly refer to *Snell*, but recognized, in language taken directly from *Snell* (at p. 330), that he was required to take a robust and pragmatic approach in determining the issue, and that a causal inference was available in the absence of scientific proof of causation. He accepted the proposition that factual causation is a practical question that is often best answered by ordinary common sense.

[70] Relying on *Sacks v. Ross*, 2017 ONCA 773 at para. 117, the judge concluded that, in an action for delayed medical diagnosis and treatment, a plaintiff must establish that the delay caused or contributed to the unfavourable outcome. The question in such a case is whether, on a balance of probabilities, the plaintiff has proven that the unfavourable outcome would have been avoided with prompt diagnosis and treatment. The judge described (at para. 267 of the RFJ) the causal reasoning process, again with reference to *Sacks*:

> (2) The Causal Reasoning Process
>
> [47] Regardless of whether the defendant's breach of the standard of care is an act or an omission, the trier of fact's cognitive process in determining causation has three basic steps. The first is to determine what likely happened in actuality. The second is to consider what would likely have happened had the defendant not breached the standard of care…
>
> [48] There are two possible outcomes to the trier of fact's imaginative reconstruction of reality at the second step. On the one hand, if the trier of fact draws the inference from the evidence that the plaintiff would likely have been injured in any event, regardless of what the defendant did or failed to do

> in breach of the standard of care, then the defendant did not cause the injury. On the other hand, if the trier of fact infers from the evidence that the plaintiff would not likely have been injured without the defendant's act or failure to act, then the "but for" test for causation is satisfied…
>
> [Emphasis added.]

[71] The judge then applied this analytical framework to the circumstances of the case before him and the facts as he found them to be. Because it is fundamental to the resolution of this appeal, his reasoning and conclusion on causation is reproduced in some detail below:

> [269] It is common ground that Kyrcee's injuries were caused by a hemolytic event. The core dispute is as to when that event occurred: as a result of a progressive and continuous increase in Kyrcee's bilirubin level since birth which, if treated, would likely have prevented her injuries, or suddenly and unpredictably on approximately August 5, 1996?
>
> …
>
> [271] The plaintiff relies on the opinion of Dr. Kaplan that the trajectory of Kyrcee's bilirubin level did not follow the expected nomogram. Rather, he opined that her hemolysis was an ongoing occurrence, as the trajectory of Kyrcee's physiological bilirubin level rose steadily and linearly from birth until August 6, 1996.
>
> [272] The defendants rely on the opinions of Drs. Boulton, van Aerde, and Manhas that the trajectory of Kyrcee's bilirubin level indeed followed a normal and expected trajectory below treatment level until approximately August 5, when she suffered a sudden, acute, and unforeseeable oxidative hemolysis, entirely unrelated to her physiological bilirubin. In other words, the defendants say that there were two unrelated pathological "biphasic" processes at play.
>
> [273] All of the medical experts agreed that the symptoms of acute bilirubin encephalopathy are arching of the back, a high-pitched cry, lethargy, and loss of interest in feeding. The uncontroverted evidence is that none of those symptoms were apparent prior to midnight on August 5. Nadine's evidence was steadfast that, although she was jaundiced, Kyrcee was an alert, active, and seemingly healthy baby up to and including when she finished feeding her at approximately midnight on August 5. Ms. Perry's observations that afternoon are corroborative of that assessment.
>
> [274] Dr. Kaplan did not address this critical evidence. If Dr. Kaplan's theory is correct, Kyrcee's bilirubin level would have been far higher than 165 µmol/l in hospital and over 500 µmol/l on August 3, at which point it is more probable than not that the symptoms of hyperbilirubinemia would have been obvious.
>
> [275] Drs. Bolton, van Aerde, and Manhas opined that, because there were no symptoms of oxidative hemolysis present prior to midnight on August 5, Kyrcee's hemolysis could not have started more than 48 hours prior to her admission to hospital on August 6. Importantly, each of Drs. Boulton, van Aerde, and Manhas opined that the significant drop in Kyrcee's hemoglobin

> between her admission to Kitimat Hospital on August 6 and her later admission to BC Children's hospital is objective evidence of an acute hemolytic event that could not have been occurring for the prolonged period of time, as was suggested by Dr. Kaplan.
>
> …
>
> [277] Neither counsel for the plaintiff nor for Nadine attempted to explain what can only be described as a glaring hole in the theory of the plaintiff's case.
>
> [278] I accept, as Dr. Fazal opined, that a follow-up bilirubin test prior to August 5 may have resulted in the initiation of phototherapy. However, the plaintiff has failed to demonstrate, on the balance of probabilities, that initiation of treatment for her jaundice prior to August 5 would have prevented her injuries. I accept the opinion of Dr. Boulton that, although the ongoing bilirubin in Kyrcee's blood would have impacted the ability of her immature liver to process it, the level of hemolysis that occurred after midnight on August 5 was so extreme that it would have overwhelmed the ability of any liver to process it.
>
> [279] I accept the opinions of Drs. Boulton and Manhas and find it is more likely than not that the severe anemia suffered by Kyrcee was the result of sudden oxidative or hemolytic stress that caused her red blood cells to burst, release the hemoglobin which was broken down into heme, and then bilirubin. This sudden surge was simply too much for her body to clear on its own.
>
> [280] Accordingly, the plaintiff has failed to establish Dr. Brian Ewart and Dr. Sheila Ewart's breach of the standard of care caused her injuries. The expert evidence I accept is that the plaintiff did not exhibit prodrome or symptoms indicative of elevated levels of bilirubin warranting treatment until, at the earliest, midnight on August 5, 1996. Thus, the plaintiff has not proven, on a balance of probabilities, had Drs. Ewart ordered a follow-up bilirubin test prior to August 5, 1996, the plaintiff's injuries would not [*sic*] have been avoided.
>
> [281] The plaintiff's theory of the case fails in this regard. In my view, the most that can be said is that there is a possibility that Kyrcee's hemolysis was not sudden and acute and that Dr. Kaplan's theory of her bilirubin trajectory in fact occurred. However, given Nadine's evidence that none of the symptoms of hemolysis existed prior to midnight on August 5, there is no basis for a finding that an earlier follow-up bilirubin test would have would have prevented her injuries…
>
> [Emphasis added.]

[72] I wish to emphasize that the parties agreed on the oral hearing of this appeal that the judge's use of the word "not" in para. 280 of his reasons was simply a grammatical error. It was accepted that the judge meant to say this: "Thus, the plaintiff has not proven, on a balance of probabilities, had Drs. Ewart ordered a

follow-up bilirubin test prior to August 5, 1996, the plaintiff's injuries would have been avoided."

IV. Grounds of Appeal

[73] The appellant argues on appeal that:

1. The trial judge erred in law by failing to consider the availability of an adverse inference of causation in circumstances where causal uncertainty is attributable to the defendant's negligence. In support of this ground of appeal, the appellant relies on *Snell* and *Benhaim*; and

2. The trial judge erred by misapprehending evidence material to the causation analysis. It is alleged that he did so by:

   (a) forgetting, ignoring or misconceiving key objective evidence, including his own factual findings concerning Kyrcee's increasingly severe jaundice;

   (b) confusing two key medical terms; kernicterus (the injury) and hyperbilirubinemia (the condition requiring treatment); and

   (c) misconceiving the expert evidence on Kyrcee's bilirubin trajectory and the onset of detectable hemolysis.

V. Analysis

[74] Absent an extricable error in law, causation is a factual inquiry reviewed for palpable and overriding error: *Clements v. Clements*, 2012 SCC 32 at para. 8; *Ediger v. Johnston*, 2013 SCC 18 at para. 29.

[75] On the first ground of appeal, the appellant says the judge's causation analysis reflects extricable error in principle. Specifically, the appellant submits that the evidentiary gap most relevant to proof of causation—Kyrcee's bilirubin levels between July 31 and August 6—was attributable to the defendants' negligence. In these circumstances, the appellant submits that the judge was required to consider whether an adverse inference should be drawn with respect to causation.

[76] To succeed on the second ground of appeal, the standard of review is deferential. The appellant is obliged to demonstrate palpable and overriding error in the judge's factual findings. In other words, the error must be plainly seen and so material as to be determinative of the outcome of the case: *Salomon v. Matte-Thompson*, 2019 SCC 14 at para. 33.

1. Failure to Draw an Adverse Inference on Causation

[77] The appellant asserts that causal uncertainty arose in this case due to the negligence of the defendants in failing, among other things, to monitor Kyrcee and order repeat bilirubin testing in the days after her discharge from the hospital. The appellant does not suggest that the judge was required to draw an adverse inference in these circumstances. Indeed, that position is not open to her: *Benhaim* at para. 50. Rather, she submits the judge was required to consider whether to draw such an inference. The appellant submits that we should conclude, based largely on the absence of any express reference in the reasons for judgment to *Snell* or *Benhaim*, that the judge failed to appreciate the availability of an adverse inference in a case involving negligently-created causal uncertainty and so erred in the application of the governing legal framework.

[78] The appellant further submits that the trial judge may have been led into this error by the respondents' closing submission that because the defendants led evidence respecting causation, it would be inappropriate for the court to infer causation from an established breach of the standard of care.

[79] For the reasons that follow, I am unable to accede to this ground of appeal.

[80] It will be helpful, at the outset, to restate, in a non-exhaustive way, some of the foundational principles respecting proof of causation in negligence actions that are of particular relevance to this appeal:

- The "but for" test is the generally applicable test for proof of causation. The plaintiff must show on a balance of probabilities that "but for" the defendant's negligent act, the injury would not have occurred: *Clements* at para. 8;

- In all cases, the plaintiff assumes the burden of proving causation on a balance of probabilities: *Ediger* at para. 36. Causation need not, however, be proven with scientific precision: *Snell* at 328. This is because the law requires proof of causation only on a balance of probabilities. Courts should take a "robust and pragmatic" approach to the facts and may draw inferences of causation based on common sense": *Benhaim* at para. 54;

- In medical malpractice cases, the defendant is often in a better position than the plaintiff to determine the cause of the injury: *Snell* at 322. In weighing the evidence, the trier of fact may, therefore, consider the relative ability of each party to present evidence on a fact in issue. To borrow the words of Lord Mansfield in *Blatch v. Archer* (1774), 1 Cowp. 63, 98 E.R. 969 at p. 970, "evidence is to be weighed according to the proof which it was in the power of one side to have produced, and in the power of the other to have contradicted";

- In some cases, it may be that very little affirmative evidence on the part of the plaintiff will justify the drawing of a common-sense inference of causation in the absence of "sufficient evidence to the contrary": *Snell* at 328–29; *Benhaim* at para. 54. As the Court put it in *Ediger* at para. 36, "[t]he trier of fact may, upon weighing the evidence, draw an inference against a defendant who does not introduce sufficient evidence contrary to that which supports the plaintiff's theory of causation";

- Put differently, in cases of negligently-created causal uncertainty where a plaintiff adduces some evidence of causation, it is open to a trial judge to draw a causal inference unfavourable to the defendant that serves to discharge the plaintiff's burden of proof: *Benhaim* at para. 42. The inference operates as something of a counterweight, offsetting the imbalance and consequent unfairness that may arise, particularly when a defendant seeks shelter in the evidentiary vacuum created by their own negligence and relies on the burden of proof shouldered by the plaintiff to defeat the claim. The

underlying policy goal seeks to balance two considerations: (1) ensuring that defendants are held liable for injuries only where there is a substantial connection between the injuries and their fault; and (2) preventing defendants from benefiting from the uncertainty created by their own negligence: *Benhaim* at para. 66;

- The available inference is permissive, not mandatory. It is simply a component of the fact-finding process, and one that is not unique to this context: *Benhaim* at paras. 54–55. Whether to draw a causal inference unfavourable to the defendant is a matter best left to the discretion of the trial judge: *Benhaim* at para. 52;

- Further, whether to draw the inference must be based on an evaluation of all of the evidence, including the weaknesses in the plaintiff's expert evidence relating to causation: *Benhaim* at paras. 44, 52. As noted in Allan M. Linden et al., *Canadian Tort Law*, 12th ed. (Toronto: LexisNexis Canada Inc., 2022), at s. 4.03:

  > An inference of causation can always be rebutted by other, more probative evidence; therefore, the inference approach to causation can have the effect of flushing out causal evidence in circumstances where it otherwise might be tactically withheld.

[81] Appellate courts are obliged to read reasons for judgment in context: *R. v. Sheppard,* 2002 SCC 26 at paras. 24–26; *Hill v. Hamilton-Wentworth Regional Police Services Board*, 2007 SCC 41 at para. 101; *Bjornson v. Shaw*, 2010 BCCA 510 at para. 18. There are several contextual features in this case that leave me unpersuaded by the appellant's argument that the judge failed to appreciate the general principles set out above, or turn his mind to whether an inference of causation should be drawn in this case.

[82] First, there is nothing manifest in the reasons for judgment confirming the appellant's submission that the judge failed to appreciate that, in a case of negligently-created causal uncertainty, the law permits the drawing of a causal inference unfavourable to a defendant: see *R. v. R.E.M.*, 2008 SCC 51 at para. 19.

[83] The judge clearly understood there was an evidentiary gap attributable to the failure of the respondents to order follow-up bilirubin testing after Kyrcee's discharge from the hospital. Although *Snell* was not expressly referenced in the reasons for judgment, the judge understood he was required to take a "robust and pragmatic" approach to determining causation—language taken directly from *Snell*. He also appreciated that scientific proof of causation was not required, referencing *Clements* on this point. Finally, it is apparent that the judge understood that whether Kyrcee's injuries were caused by the respondents' negligence turned on the factual inferences he was prepared to draw from the evidence as a whole (see RFJ at para. 267, reproduced herein at para. 71). In my view, there is no manifest error in principle reflected in what the judge said on the issue of causation.

[84] Second counsel made extensive reference at trial to the circumstances in which causal inferences may be drawn. In her opening submissions, counsel for the appellant at trial (not counsel on appeal) drew the attention of the judge to the issue, citing *Ghiassi (Litigation guardian of) v. Singh*, 2018 ONCA 764 at para. 25 for the proposition that "in the absence of evidence to the contrary by the defendant, an inference of causation may be drawn although positive or scientific proof of causation has not been adduced."

[85] In *Ghiassi*, the defendant nurse noticed but failed to report that the infant plaintiff was becoming increasingly jaundiced. When a bilirubin test was eventually taken, the infant was found to have been suffering from severe hyperbilirubinemia. Treatment proved unsuccessful and the infant developed kernicterus. The plaintiff led expert evidence that the injuries could have been avoided had phototherapy been initiated at an earlier time. Significantly, this evidence was unchallenged by the defendant, who adduced no expert evidence on causation. The judge accepted the plaintiff's evidence on causation and found the defendant liable in negligence. In dismissing the appeal, the Court noted that "if there is a gap in the evidence about what would have happened had phototherapy been introduced [earlier]" and "that gap is the product of [the defendant's] negligence… [she] should not be permitted to

rely on the lack of evidence that her own negligence produced…": *Ghiassi* at para. 29.

[86] The point is this: the appellant emphasized the availability of a causal inference in a case of negligently-created causal uncertainty from the outset of the trial. In addition, both parties addressed the availability of such an inference and the circumstances in which it should be drawn in extensive written and oral closing submissions. The respondents sought to distinguish *Ghiassi* on the basis that the defendant nurse failed to adduce any evidence on causation. By contrast, the respondents adduced a substantial body of evidence with respect to whether Kyrcee's injuries were caused by their breach of the standard of care. In her reply submissions at trial, the appellant again relied on *Benhaim* for the proposition that an adverse inference remains available even where a defendant adduces evidence on causation. In her factum, the appellant acknowledged that the submissions made at trial reveal that both parties were *ad idem* on the law in this area.

[87] Against this background, it is difficult for the appellant to successfully argue that the judge was unaware of the availability of an adverse inference on the question of causation.

[88] In essence, we are being asked to infer error in the application of a framework of principles about which counsel were largely in agreement, and do so on the basis that the judge made no express mention of the availability of an adverse causal inference in his reasons for judgment.

[89] I am not prepared to make such an inferential leap in this case. In my view, it is much more likely, if not clear, that the judge found it unnecessary to refer to the availability of an adverse causal inference given his factual finding that the respondents' "evidence to the contrary" effectively severed the link between their wrongful act and Kyrcee's injuries. Having made the factual findings he did—that there was a "glaring hole" in the appellant's theory of the case on causation and that it was more likely Kyrcee's injuries were caused by an unpredictable, sudden onset, oxidative hemolytic event that did not occur until August 5—there was no room left

for the judge to draw a causal inference unfavourable to the defendants. Indeed, on the facts he found from the evidence as a whole, I do not see how he could properly have drawn such an inference in this case.

[90] In summary, the judge was made aware of the availability of an adverse causal inference discussed in *Snell* and *Benhaim*. The case at bar is quite unlike *Ghiassi* given the substantial body of expert evidence called by both parties on the issue of causation. The judge reviewed this evidence at length, noting the extent to which it revealed weaknesses in the appellant's expert evidence relating to causation. He made factual findings that Kyrcee's injuries were not caused by the respondents' negligence, but were more likely attributable to a later-arising independent event. He concluded that a follow-up bilirubin test shortly after Kyrcee's discharge from the hospital would not have prevented her injuries. Although he made no express reference to the availability of the adverse causal inference, I consider it implicit in the reasons that the judge, in the exercise of his discretion and having regard to the factual findings he made, understandably declined to draw one: *Benhaim* at para. 52. I see no reviewable error in any of this. The judge's factual findings were well grounded in the evidence. His analysis is, in my view, consistent with a principled application of the governing framework set out earlier in these reasons.

[91] Finally, I am unable to accept the appellant's position that the judge's analysis was likely tainted by submissions made on behalf of the respondents at trial that no causal inference can be inferred from a breach of the standard of care where the defendants have led some evidence on causation. While the respondents' submissions may well have pitched the claim too high in light of the guidance provided in *Benhaim*, I am unpersuaded that this submission had any material bearing on the judge's reasoning process or the outcome of this case.

[92] For the foregoing reasons, I would not give effect to this ground of appeal.

2. Alleged Misapprehension of the Evidence

[93] The appellant submits that the judge forgot, ignored or misconceived crucial evidence of causation in two ways: (1) by failing to consider Kyrcee's worsening jaundice over the long weekend when determining her likely bilirubin trajectory; and (2) by confusing the onset of the condition requiring treatment (hyperbilirubinemia) with the onset of the resulting injury (kernicterus). The appellant submits that these errors led the judge to mistakenly conclude that Kyrcee's brain injury was unpreventable. The appellant submits that these errors are both palpable and overriding.

[94] On the first of the alleged errors, the essence of the appellant's position is that the judge preferred the respondents' biphasic theory despite making factual findings inconsistent with that theory. More specifically, the judge accepted Shari's account that there was a "daily, progressive deepening of Kyrcee's yellow skin colour from July 31, 1996", and that it had spread to her legs by August 5. He therefore rejected a significant component of the assumed facts upon which the respondents' experts relied in advancing their biphasic theory—that Kyrcee's jaundice was "mild" on August 1 and 2, and that there was no observable change in her skin colour from August 3 to 5.

[95] The appellant argues that evidence respecting the progression of Kyrcee's jaundice was critical in this case—it was the only probative and contemporaneous evidence of her bilirubin trajectory following her discharge from the hospital. The appellant submits that this evidence accorded with Dr. Kaplan's gradual but progressive theory of bilirubin accumulation and undermined the biphasic theory advanced by the defence, which was premised on the assumption that Kyrcee's jaundice was mild on August 1 and 2 and did not progress over the next three days.

[96] The appellant submits that, while the judge found Kyrcee's jaundice to have become more pronounced over the long weekend, he failed to engage with the implications of this finding in his causation analysis. The judge's failure to do so is

said to demonstrate that he forgot, ignored, or misconceived the evidence in a way that went to the core of his reasoning on causation.

[97] I am unable to accept the appellant's submissions on this issue. Jaundice is only one symptom of hyperbilirubinemia. Physiological jaundice can be expected to increase initially and then plateau within five to seven days. It does not usually require treatment. Standing alone, jaundice is not, as the appellant suggested in her factum, "a symptom indicative of elevated levels of bilirubin warranting treatment."

[98] The judge found it unsurprising on the medical evidence before him that Kyrcee's jaundice deepened and progressed in the days following her discharge. He concluded that Shari's observations were "consistent with the typical trajectory of jaundice in pre-term babies", and not evidence of toxic hyperbilirubinemia. I see no basis upon which we could properly interfere with this finding.

[99] Further, I am not persuaded that the judge ignored the evidence of Kyrcee's worsening jaundice in his causation analysis. The judge placed significant weight on the absence of any behavioural symptoms that would likely have become apparent well before August 6 on Dr. Kaplan's progressive, linear theory. He was entitled to approach the case with the obvious disconnect between Dr. Kaplan's theory and Kyrcee's behaviour in mind. He was also entitled to have regard to the totality of the expected symptoms associated with severe hyperbilirubinemia, rather than assign the fact of progressing jaundice dispositive weight. That choice was squarely within his discretion as the trier of fact and I see no basis to interfere with it.

[100] Moreover, the judge did refer to Kyrcee's jaundice in his causation analysis but concluded that "the plaintiff did not exhibit prodrome or symptoms indicative of elevated levels of bilirubin warranting treatment until, at the earliest, midnight on August 5, 1996" (see RFJ at para. 280, reproduced herein at para. 71; emphasis added). In this portion of his causation analysis, the judge was addressing the prodrome of kernicterus. Read in context, the judge was commenting on the fact that Kyrcee was not displaying symptoms typically associated with acute bilirubin encephalopathy warranting treatment until midnight on the evening of August 5.

There is no inconsistency between the judge's finding that Kyrcee's jaundice progressively worsened, and his finding that the bilirubin in her system did not reach a level at which the symptoms of acute bilirubin encephalopathy requiring treatment would become apparent until midnight on the evening of August 5.

[101] Finally, I do not accept the appellant's contention that the respondents' biphasic theory rests entirely upon the assumption that Kyrcee's jaundice did not progress from August 3 to 5. In my respectful view, this submission mischaracterizes the judge's reasons for preferring the biphasic theory. As the reasons for judgment make clear, the judge accepted the biphasic theory, at least in part, because additional and acute symptoms—including lethargy, arching of the back, a high-pitched cry and loss of interest in feeding—would have manifested themselves before the evening of August 5 if Dr. Kaplan's theory was correct.

[102] In short, I am not persuaded that the judge ignored or otherwise forgot about Kyrcee's worsening jaundice in his causation analysis. He simply concluded that jaundice was typical for a pre-term baby and that the plaintiff did not establish that treatment of Kyrcee's bilirubin levels prior to the midnight of August 5 would have prevented her injuries.

[103] On the second error alleged, the appellant asserts that the trial judge's failure to appreciate the distinction between the condition requiring treatment (hyperbilirubinemia) and the resulting injury (kernicterus) led him to overlook key evidence of increasing jaundice and erroneously conclude that Kyrcee's brain injury could not have been prevented until it started to occur. The appellant points out that hyperbilirubinemia can be effectively treated with phototherapy well in advance of a baby developing kernicterus. Put differently, the appellant submits the judge's misunderstanding of the evidence led him to conclude that causation had not been established because of the absence of signs that the resulting injury had already occurred.

[104] In support of her position, the appellant argues that at paras. 273–274 of the RFJ (reproduced herein at para. 71), the judge conflated hyperbilirubinemia with

acute bilirubin encephalopathy and, as a result of the error, concluded that Dr. Kaplan's theory was inconsistent with the evidence as a whole. For convenience, the judge summarized in these two paragraphs the symptoms of acute bilirubin encephalopathy and noted the uncontroverted evidence that none of the symptoms typically associated with the onset of this condition were apparent before midnight on the evening of August 5. He concluded that Dr. Kaplan's opinion did not account for this critical evidence and that if his progressive theory was correct, Kyrcee's bilirubin level would have been over 500 µmol/l on August 3, at which point "the symptoms of hyperbilirubinemia would have been obvious." The appellant argues that the symptoms of hyperbilirubinemia were obvious on August 3—Kyrcee's jaundice was progressing.

[105] In my respectful view, the appellant is inappropriately parsing language used by the judge who was attempting to distill in his reasons a complex body of evidence and a lexicon of medical terms—terms that were not always used consistently by the medical experts who gave evidence. In my view, and when read in context, it is apparent that the judge meant to say in paras. 273–274 of the reasons that at 500 µmol/l the symptoms of toxic hyperbilirubinemia or acute bilirubin encephalopathy would have been obvious. There was ample evidence upon which the judge could have relied in coming to this conclusion which was, of course, central to his causation analysis.

[106] I say the same about the appellant's contention that the judge rejected her theory of causation because she failed to explain how "physiologic hyperbilirubinemia" could have occurred in the absence of any of the "prodrome to kernicterus" (RFJ at para. 276). Read in the context of the symptoms being discussed in para. 276 of the RFJ, it is apparent to me that the judge meant to say that the appellant failed to explain how "toxic hyperbilirubinemia" could have occurred in the absence of the typical prodrome to kernicterus. In my view, and read in context, the passage does not have the effect of undermining the judge's causation analysis. The point the judge was making is this: no evidence was adduced to explain how Kyrcee's bilirubin level could have been as high as that

suggested by Dr. Kaplan in the absence of any symptoms of toxic hyperbilirubinemia.

[107] The appellant submits that the conflation of hyperbilirubinemia and kernicterus is also evidenced in paras. 281–282 of the RFJ. In those paragraphs, the judge said, among other things, that as "none of the symptoms of hemolysis existed prior to midnight on August 5" (emphasis added), there is no basis for finding that an earlier bilirubin test would have prevented her injuries. The appellant submits that symptoms of hemolysis were present as there was substantial evidence she was considerably jaundiced. The appellant relies on this paragraph to argue that the judge erroneously viewed treatment as being ineffective until the prodrome of kernicterus were present. Put differently, the judge is said to have misapprehended the uncontroverted evidence that hyperbilirubinemia is treatable before the prodrome for kernicterus emerge.

[108] Again, I think it clear that in para. 281 of the RFJ (reproduced herein at the para. 71), the judge was referring to the absence of symptoms of acute hemolysis. The judge's use of the word "hemolysis" reflects nothing more than the tendency of the expert witnesses to use that word throughout the trial as a shorthand synonym for "acute hemolysis." In my view, there is nothing in any of this that undermines the integrity of the judge's causation analysis.

[109] In any event, there was no evidence in this case that the injuries the appellant suffered resulting from the sudden onset of an acute hemolytic event could have been avoided with earlier medical management. The evidence the judge accepted led him to the conclusion that the plaintiff failed to demonstrate that initiation of medical treatment, such as phototherapy, prior to August 5 would have prevented her injuries. Again, that factual finding was supported in the evidence and I see no basis upon which we could properly interfere with it.

[110] Finally, the appellant says that proof of the claim did not turn on whether the judge accepted Dr. Kaplan's progressive theory. She submits—for the first time on appeal—that causation was established on either the progressive or the biphasic

theories advanced by the appellant and respondents. As I understand it, this argument has three key building blocks. The first is that the judge found the respondents to have breached the standard of care by failing to closely monitor Kyrcee's bilirubin levels and arrange for daily tests over the course of the long weekend. The second is that, even on the evidence of the respondents' causation experts, the acute hemolytic event Kyrcee was experiencing would therefore have been detectable on August 4 or 5, before the emergence of symptoms associated with acute bilirubin encephalopathy. The third, which follows from the first two, is that the sudden and acute hemolytic event posited by the respondents' experts would have been detected at an earlier time if the standard of care had been observed. As a result, Kyrcee's injuries would have been avoided. On this basis, the appellant asserts that it is open to this Court to conclude that, "but for" the respondents' negligence, it is likely she would not have sustained her injuries.

[111] In addition, the appellant alleges that the judge failed to consider the possibility of an effective treatment intervention on August 5. In this regard, she highlights the findings of the judge that neither a follow-up bilirubin test nor the initiation of treatment for her jaundice prior to August 5 would have prevented her injuries (see RFJ at paras. 278, 280, reproduced herein at para.71).

[112] The appellant's approach to this issue rests largely on the testimony of Dr. Feinstadt, who gave evidence for the appellant at trial on the standard of care. Dr. Feinstadt testified that a reasonably prudent family physician in 1996 would have: (1) arranged for a second bilirubin test on receiving the message from Nurse McLellan on August 2 that Kyrcee was still jaundiced; and (2) ordered daily bilirubin tests over the course of the long weekend. The appellant notes that the judge accepted the evidence of Dr. Feinstadt on the standard of care and concluded that a reasonably prudent family physician would have known that close monitoring of her condition was warranted (see RFJ at paras. 87, 243). From here, the appellant submits that daily testing would have detected the onset of an acute hemolytic event on or before August 5, and allowed time for successful therapeutic intervention. In

essence, the judge is said to have ignored the significance of the findings he made concerning the breach of the standard of care in addressing causation.

[113] As I see it, there are two major problems with this submission. The first is that the breach of the standard of care found by the judge was in the failure of the respondents to order a follow-up bilirubin test on August 2 or as soon thereafter as was practicable. Reading the reasons as a whole, it is not at all clear to me that a finding was made that the respondents breached the standard of care by failing to order daily bilirubin tests commencing on August 2. The following passages from the reasons for judgment are instructive on this point:

> Dr. Brian Ewart
>
> [245] I find that a reasonably prudent family physician in the circumstances Dr. Brian Ewart found himself in would have communicated directly with Ms. McLellan after his office received her telephone call on August 2, 1996, discussed her assessment with her and, knowing the risks of hyperbilirubinemia, made arrangements for Kyrcee's assessment and possible treatment over the long weekend, including a follow-up bilirubin test. Dr. Brian Ewart did nothing.
>
> …
>
> Dr. Sheila Ewart
>
> [250] I accept the opinion of Dr. Feinstadt that, in such circumstances and given the known risks to pre-term, exclusively breastfed, jaundiced babies, a reasonably prudent family doctor would have ordered a follow-up bilirubin test as soon as practicable thereafter, particularly since there was an upcoming long weekend.
>
> [Emphasis added.]

[114] Second, the appellant unsuccessfully advanced a specific theory of causation at trial. In her closing written submissions, the appellant urged rejection of the respondents' biphasic theory, characterizing it as speculative and lacking any evidentiary basis. She now seeks to advance an alternative position based on the respondents' biphasic theory, and does so in the absence of evidence that her injuries could have been prevented if the beginning of an acute, hemolytic event was detected at some point in the two days before her re-admission to hospital. The appellant carried the burden of proof on causation. It was incumbent on her to show, as a matter of fact, that she would not have suffered the loss "but for" the negligence

of the respondents. There is no evidence supporting the proposition that a treatment intervention, had one been undertaken prior to August 5, would likely have changed the outcome of the acute hemolytic event. I cannot give effect to the appellant's submission on this point in the absence of this evidence,

VI. Conclusion

[115] For the foregoing reasons, I would dismiss the appeal.

"The Honourable Mr. Justice Fitch"

I agree:

"The Honourable Justice Mackenzie"

I agree:

"The Honourable Mr. Justice Abrioux"