TIN THE DISTRICT COURT
AT WELLINGTON

I TE KŌTI-Ā-ROHE
KI TE WHANGANUI-A-TARA

[2021] NZACC 126 ACR 143/19

UNDER	THE ACCIDENT COMPENSATION ACT 2001
IN THE MATTER OF	AN APPEAL UNDER SECTION 149 OF THE ACT
BETWEEN	KEVIN BOTHA Appellant
AND	ACCIDENT COMPENSATION CORPORATION Respondent

Hearing:	15 June 2021
Heard at:	Wellington/Whanganui-A-Tara
Appearances:	Ms B Peck for the appellant Mr S Bisley and Mr L Kibblewhite for the respondent
Judgment:	4 August 2021

RESERVED JUDGMENT OF JUDGE P A CUNNINGHAM
[Treatment Injury s 26 Accident Compensation Act 2001]

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[1] This case is about a young boy who has cerebral palsy and delayed development. It is accepted that he suffered from a degree of hypoxia[1] during the birthing process. The case for the appellant is that this caused a mild Hypoxia Ischaemic Encephalopathy (HIE) which explains or partly explains Kevin's cerebral palsy. Therefore, Kevin suffered a treatment injury during the birth process and accordingly he is entitled to cover.

[2] It is the position for ACC that the more significant hypoxia occurred when Kevin was still in utero and that any hypoxia during the birthing process did not play

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1 Lack of oxygen.

a significant part in the development of cerebral palsy. Therefore, Kevin is not entitled to cover.

Background

[3] Kevin is the first child of Sijia Botha who was aged 31 when he was born on 26th January 2016. During her pregnancy Mrs Botha was cared for by a midwife. On the day Kevin was born her usual midwife was on leave. Mrs Botha and her husband Shane met the relief midwife at the Botany Downs birthing unit at 9am.

[4] By 11 40am Mrs Botha was in the second stage of labour but the baby was still high up in the birth canal. The midwife ruptured the forewaters. Staining from meconium[2] was visible in the waters. At 1210pm Mrs Botha entered a birthing pool as suggested by the midwife. The temperature of the water was too hot, so cold water was added. Progress of the second stage of labour was slow. At 1350, Mrs Botha got out of the pool. At 1400 the midwife performed an episiotomy and 5 minutes later Kevin was born.

[5] Kevin was not breathing when he was born. His vital signs were poor. A second midwife assisted by a third resuscitated Kevin as best they could given that this was a birthing centre and not a hospital. An ambulance was called to transport Kevin to Middlemore Hospital (MMH). Kevin and his mother arrived at MMH at 1530. There was some delay with the ambulance service because the first one to arrive did not have the necessary equipment required. On arrival, Kevin was diagnosed as having respiratory distress, meconium aspiration syndrome (MAS), hypoglycaemia and persistent hypertension of the newborn and was suspected of having sepsis. Initially he was in the neonatal intensive care unit. He recovered well and was discharged home with his mother 6 days later on 1 February 2016.

[6] At age 4 months Kevin was not meeting his milestones. He was seen by Paediatrician, Dr Tuck. In a letter dated 17th May 2016 to Mrs Botha's GP, Dr Tuck described that Kevin had a history of poor feeding and posturing. Given his condition at birth, Dr Tuck suspected that Kevin had suffered an hypoxic brain injury

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2 A newborn baby's poo which is dark and sticky.

perinatally. Dr Tuck made referrals to a dietician for assistance with feeding for Kevin and also to a speech therapist. Dr Tuck also diagnosed moderate hearing loss, feeding difficulties and failing to grow. Kevin has required ongoing additional assistance and therapy ever since. Much of this has been carried out by his parents.

[7] Dr Katie Tuck completed a treatment injury claim for Kevin on 5 July 2016. She described the treatment injury as management of the birth process. That Kevin suffered an injury at birth suggesting a significant hypoxic event during labour was not detected by attending staff.

ACC's decision

[8] After undertaking some investigations, ACC issued a decision on 26 May 2017. The claim was not approved because ACC said it did not meet the criteria for a treatment injury. Included with the letter was a treatment injury report. This referred to reports by the attending midwife, the St John's ambulance and an external clinical adviser midwife. Issues that had been raised included the small amount of amniotic fluid when the waters were ruptured, the fact there was meconium staining, the slow second stage of labour and the fact this birth occurred in a birthing unit and not a hospital as well as the ambulance transportation issue. ACC's reasoning was that there had not been a failure to treat in a timely manner and that the brain injury had not been caused by treatment.

The Review Decision.

[9] The hearing was on 2 May 2019 and the decision is dated 17 May 2019. The decision focussed on the various medical reports which had been obtained both in relation to the birth of Kevin and what had caused the hypoxic event which led to the development of Kevin's problems. One of the issues at the Review hearing which was also an issue raised on behalf of Kevin on appeal, was the evidence given by Mrs Botha that she had wanted to give birth in a hospital setting and the extent to which that was discussed with her midwife.

[10] The Reviewer referred to the treatment injury provisions in the Act and the decision of *Adlam v Accident Compensation Corporation*.[3] This was a treatment injury case where the Court of Appeal said that where it is said there has been a treatment failure, it is necessary to show that an alternative treatment would have prevented the injury suffered and that it should have been given having regard to the clinical indications at the time. Reference was also made to the issue of informed consent in the treatment injury context.

[11] By the time of the review two reports had been obtained on behalf of Kevin and his family. This included a report from Dr McEvoy an Australian Obstetrician and Gynaecologist in relation to the birthing process. And from Dr Battin who is a neonatologist.

[12] The Reviewer said that Dr McEvoy lacked specific knowledge of the New Zealand birthing model and for that reason placed little weight on his findings. Those included the small amount of amniotic fluid and the passage of meconium during the birthing process should have prompted an alternative course of treatment. For example, this contraindicated the use of a birthing pool. Dr McEvoy said that a medical opinion should have been obtained at this point.

[13] Dr Battin's opinion was that Kevin may have had a mild HIE. He was critical of the temperature of the birthing pool and the fact that it spiked a temperature in Mrs Botha. This in turn could have affected the baby.

[14] The Reviewer held that in relation to the birthing pool, Kevin did not suffer a treatment injury because of a lack of informed consent. Dr Battin had not identified a failure to treat. Further that Dr Battin's opinion was only a possibility in relation to the mild HIE. There was insufficient evidence for the Reviewer to find there had been a treatment injury.

[15] This is an appeal from the Review decision.

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3 *Adlam v Accident Compensation Corporation [2017] NZCA 457 [2018] 2 NZLR 102*.

Medical Reports

Midwife's Report (Amanda Donald)

[16] A report was provided to ACC by the midwife, Amanda Donald, who delivered Kevin. The midwife who had cared for Mrs Botha during her pregnancy was not working on the day of Kevin's birth, and Ms Donald was the backup. She met Mrs Botha at the Botany Birthing Unit that morning. A student midwife was looking after Mrs Botha while Ms Donald looked at records pertaining to Ms Botha's pregnancy.

[17] Ms Donald described her decision to rupture the membranes was because the presenting part of the baby was too high. She discussed this with Mr and Mrs Botha. There was light meconium staining in the amniotic fluid. She said the amount of fluid was consistent with forewaters. She stated that the Counties Manukau District Health Board ("CMDHB") guidelines, as they applied to Mrs Botha, meant it was appropriate to continue intermittent auscultation of the fetal heart rate ("FHR"). Her decision to put Mrs Botha in the pool was both to reduce pain and to allow the mother to relax and hence assist with the descent of the baby's head. She said that cool water was added to reduce the temperature of the water in the pool. She felt Mrs Botha was making progress with labour.

[18] But the baby was not advancing. The midwife performed an episiotomy after she got Mrs Botha out of the pool. Kevin was born at 1405. She called for the assistance of a second midwife. Her report notes the FHR after the rupture of the membranes as follows:

> [i] 155bpm[4] just after the membranes were ruptured so just after 1140
> [ii] 130bpm at 1204hrs,
> [iii] 135bpm at 1210
> [iv] 140bpm at 1230,
> [v] 130bpm at 1235
> [vi] 130bpm at 1240
> [vii] 135bpm at 1308
> [viii] 132bpm at 1312
> [ix] 132bmp at 1338
> [x] 135bpm at 1340
> [xi] 126bpm at 1350

[4] (beats per minute),

[19] The baby was born with a lot of old and thick meconium behind him and minimal liquor. He was flat and not breathing. Ms Donald handed Kevin to the midwife who had come to assist. The assisting midwife took the baby to a resuscitation room where a third midwife helped her to resuscitate Kevin. He was dried and stimulated and given 5 inflation breaths. His Apgar score[5] at 1 minute was 2/10. The baby's heartbeat had dropped to 55bpm. Chest compressions were commenced. A minute later the baby's heart rate was up to 130bpm which is normal, and he was breathing himself but showing some effort and grunting. CPAP[6] was commenced. Oxygen saturations were 71% (they should be over 95%). An ambulance was called to transport the baby and his mother to MMH. At 1410 the Apgar score was 7 and the baby was breathing, his heart rate was 177bpm. CPAP was continued.

[20] At 1416 the oxygen saturations had increased to 86% and the heart rate was 177bpm. An ambulance arrived at 1420 but it did not have the correct harness to transport the baby so another one was called. Vital signs were oxygen saturations 88% respiratory rate 70 and heart rate 166bpm. At 1425 the baby was stable but still supported by CPAP. Oxygen saturations 90%, respiratory rate 66 and heart rate 166bpm. At 1448 Kevin's vital signs were good, but oxygen saturations had dropped a little to 87%. The second ambulance arrived. Kevin and his mother were taken to MMH. In the ambulance the baby's observations continued and were recorded as respiration rate 60, heart rate 158bpm and oxygen saturation 91%. The ambulance arrived at MMH at 1501.

Second Report of Amanda Donald

[21] Ms Donald was asked three questions. The first was whether she agreed that continuous fetal heart monitoring was required. In short, her response was no. She considered intermittent auscultation was appropriate. Mrs Botha was low risk. Although there was light meconium there was no evidence of fetal distress. Intermittent auscultation was in line with the CMDHB guidelines.

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5 Named after Dr Virginia Apgar, it gives a score out of 2 for 5 characteristics, skin colour, heart rate, reflexes and responsiveness, muscle tone, and breathing rate.

6 Continuous Positive Airways Pressure.

[22] The second question was about the temperature of the pool. Ms Donald stated that 38 degrees is higher than her normal practice. She did not consider Mrs Botha's temperature of 37.5 degrees for less than two hours met the definition of maternal pyrexia.[7] This likely reduced after she left the pool. But the temperature was not taken again for an hour and a half due to high maternal needs and the fetal head being in view. The last was about cord gases. The response was that they were not taken as neither the facility to process in a timely way, nor the equipment were available at any of the CMDHB primary birthing units.

Report of Specialist Midwife (Lorna Davies)

[23] ACC sought advice from Lorna Davies who is a specialist in midwifery. She was asked if the midwife should have adopted a different treatment path at any time. Having reviewed the records Ms Davies opined that the antenatal care had been satisfactory as was the care provided during labour and immediately afterwards. Ms Davies was asked about the choice of a birthing centre as opposed to hospital care. She noted that there was no record of Mrs Botha's choice but that could be down to a lack of documentation. She was of the view that Mrs Botha was low risk and that a birthing centre was considered the appropriate choice for that reason.

Second Report of Lorna Davies.

[24] Ms Davies was asked the same questions as Ms Donald had been asked. In relation to continuous monitoring, Ms Davies agreed it was not indicated and that in this case intermittent auscultation was appropriate on the best available evidence and professional recommendation. Ms Davies agreed that the temperature in the pool was a valid concern, because of the risk of pyrexia which can result in fetal hyperthermia. In this case the midwife identified that the temperature was too warm and took steps to reduce it. She said that 37.5 degrees was mild pyrexia and that this can occur for unknown reasons including the impending birth of the baby. She said that 1.5 hours in the pool was not a long time. Overall Ms Davies felt that the midwife was acting in a way commensurate with reasonable practice. In terms of blood gases, she agreed it would have been useful for the neonatal team to have this information. But staff available were best utilised in resuscitating the baby, this was

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[7] Higher than normal temperature.

more important. Most primary birthing units in New Zealand do not have the equipment available to do this procedure.

Complex Claims Panel 02/02/2016

[25] This is an internal ACC convened panel of four doctors (including one obstetrician) and other ACC personnel. The consensus of the panel was that the selection of a birthing unit was appropriate as was the midwifery treatment. The most common cause of hypoxia is intra partum asphyxia or oxygen deprivation, but the panel opined that there was little evidence of this during labour. It is not uncommon for hypoxia to be idiopathic.[8] There was concern over the delay in a properly equipped ambulance arriving to transport Kevin to MMH and the panel requested that enquiries be made of St Johns about this. And to check if an MRI had been performed on Kevin.

Letter of Dr Sonja Farthing (Registrar for Dr Colette Muir, Paediatrician)

[26] This is dated 13th February 2018. This is a letter to Kevin's parents. It discussed the findings of a second MRI scan. Kevin has a generalised reduction in white brain matter. This was similar to the previous MRI scan. Which indicated Kevin does not have a progressive process. Dr Farthing had discussed the findings with the radiologist who wrote the report. He thought the findings most likely suggested an antenatal[9] insult to Kevin's brain at the end of the second trimester[10] or the start of the third. However, it was impossible to say this definitively and it could have occurred anytime up until the time of delivery.

[27] Dr Farthing said that the scan was not typical of hypoxic brain injury at the time of delivery and for that reason it would be reviewed at the Neuroradiology conference in two weeks' time. The letter contained an addition that this had happened. The opinion of the conference was that the changes seen on the scan could be consistent with placental insufficiency in late pregnancy.

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8 Of unknown cause.
9 Prior to birth.
10 Between the 4th and 6th month of pregnancy.

Letter of Dr Alhussain Aldabbagh (Paediatric Neurology Registrar)

[28] Kevin was seen by Dr Aldabbagh on 19 March 2018 and his letter dated 23 March 2018 is to Dr Colette Muir. The history, examination, and other information about Kevin preceded the Doctor's assessment of Kevin. He described Kevin as having global developmental delay and hypotonia[11] constituting a picture of hypotonic cerebral palsy. The MRI findings were consistent with perinatal insult. His profound bilateral sensorineural hearing loss was not entirely accounted for. His neuroimaging was not typical for CMV.[12] Dr Aldabbagh said that undoubtedly some of Kevin's disability is accounted for by perinatal events but he was not certain this accounted for all findings.

First Report of Dr Battin

[29] Dr Battin is a neonatologist. His view was that there had been a possible mild hypoxic ischaemic encephalopathy (HIE)[13] that was not appreciated at the time. His reasons for this view were outlined.

[i] Evidence of systemic renal injury with peak creatinine 99umop/1 on 27/01/2016 and 90umol/1 om 28/01/2016. This followed a period where there was metabolic stress demonstrated by lactacte > 4 on testing on 26/01/2016 at 1530. He was unsure of the level of stress as cord blood gases were not taken. It is reasonable to suggest that the perinatal events, poor Apgar scores and requirement for resuscitation were linked to the subsequent renal impairment. The timing of the peak creatinine value supports this.

[ii] The limited neurological examinations do not document features of a moderate or severe HIE. They are consistent with a mild HIE. The initial water temperature in the pool was above that recommended and there was associated maternal pyrexia. Dr Battin quoted from both the NZ College of Midwives Guildelines and the equivalent UK College of Midwives and the National Women's Guidelines. The NZ College of

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11 Weak muscle tone.
12 A viral illness.
13 HIE can cause developmental delay, cerebral palsy, epilepsy or cognitive impairment.

> Midwives state that because fetal temperature is regulated through the mother, the temperature should be as cool as the woman finds comfortable during the first stage of labour. It should be increased to 36 to 37 degrees for the baby's birth. The UK College states that fetal hyperthermia is linked with hypoxia and labouring women should avoid becoming pyrexic. Water temperature should be comfortable for the woman but should not exceed 37 degrees. An increase of temperature 1 degree above the baseline indicates that the use of a water pool should be discontinued. The National Women's Health guideline states that the temperature of the water pool on entry should be 35.5 to 36.5 Celsius or as cool as the patient feels comfortable.

[30] Dr Battin did not consider the injury (HIE) to be a necessary part of treatment or due to an underlying condition.

Post Review Hearing Reports – Dr Jenny Westgate

[31] ACC sought advice from Obstetrics and Gynaecology specialist Dr Jenny Westgate. Dr Westgate had the reports already in ACC's possession. She was asked what was the most likely cause of Kevin's injuries. After reviewing the information made available to her, Dr Westgate said she believed that the placental function had deteriorated in the last few weeks of pregnancy. There were two reasons for this. First a scan at 38 weeks and 6 days showed an estimated weight of the baby of 3225gms. Kevin weighed 2910gms at birth which was in the lowest 10% of birth weights for a full term baby. Kevin was able to recover quickly from the hypoxia he suffered at birth which indicates the hypoxic episode during the birth was brief.

[32] The second question asked of Dr Westgate related to the initial temperature in the birthing pool being 38 degrees Celsius and any likely effect on the fetal heartrate (FHR). After adding water to cool the water in the pool, the temperature was not checked until an hour later, by which time it was within the correct range. At 1240, Mrs Botha's temperature was 37.5. She left the pool at 1350. Dr Westgate explained that the fetus is usually one degree warmer than the mother. The only way the fetus can reduce temperature is to increase blood flow from the placenta. This results in the fetal heart rate increasing. There is no evidence of this. For these

reasons Dr Westgate thought that the temperature in the birthing pool did not result in an increase in Kevin's metabolic demand.

[33] Dr Westgate was asked if any alternative treatments should have been given to Mrs Botha. Dr Westgate was of the view that antenatally and during the first stage of labour there were no clinical findings of concern during labour. During labour when the meconium stained liquor was observed, this required a reassessment. Dr Westgate explained that the CMDHB guidelines for midwives state that the presence of meconium is an indication for continuous fetal heart monitoring. But there is a note which states that in some cases in the absence of other risk factors, light meconium with a documented normally grown baby, it may be appropriate to continue intermittent fetal heart monitoring. The midwife said the staining was light and there was no comment on the amount of liquor so the midwife was acting in accordance with the guideline. The Royal Australian and New Zealand College of Obstetrics and Gynaecology Guidelines lists the presence of meconium liquor as an indication for continuous fetal monitoring. The CMDHB guidelines on birthing pools state that they should not be used when there are no factors of wellbeing for mother and baby that would increase the risk for either. There is no specific mention of meconium. The NZ College of Midwives consensus statement is similar.

[34] Other DHBs provide different advice. For example, the Auckland DHB states that the use of a birthing pool in the presence of meconium should be discussed with the Charge Midwife or the medical team. And have appropriate telemetry available (underwater fetal heart monitoring). In terms of frequency of intermittent fetal heart auscultation, this should be every five minutes during the second stage of labour and this did not happen in Kevin's case.

[35] The next issue was the effects any alternative treatment would have had on Kevin's injuries. Dr Westgate repeated that under the ADHB and Royal College guidelines, there would have been continuous monitoring after the presence of meconium when the membranes were ruptured. Dr Westgate said she suspected that by 1350, there would have been FHR changes which indicated an earlier delivery was required. The FHR at that time was 126/bpm which is lower than it had been and it was suspected decelerations would have been occurring. In the next 15

minutes, there would have been a deterioration in the FHR. If detected, an earlier delivery should have occurred, by use of an episiotomy. If this had happened, Kevin would have been born in better condition.

[36] The next question was about what the low volume of liquor and meconium staining indicated and what steps should have been taken. Dr Westgate explained that low liquor volume is most likely to reflect a degree of fetal compensation for hypoxia. Chronic hypoxia can have significant implications for fetal growth and organ injury and can adversely affect both short and long term health after birth. But she was not persuaded there was low liquor in this case. It was difficult to say what proportion of Kevin's injuries were due to hypoxia in utero and hypoxia during the birthing process. In terms of the meconium, Dr Westgate explained that meconium is passed in response to hypoxia and is then inhaled by the fetus gasping for breath. This can occur in the absence of hypoxia. MAS can cause an inflammatory response. Because there was no examination of the placenta and the cord blood in this case, it was not possible to assess the level of inflammation.

[37] The next question was what recommendations Dr Westgate would have made if she had received a referral from the midwife and what recommendations she would have made. Dr Westgate answered that because of the CMDHB guidelines there was no need for the midwife to consult when the meconium stained liquor was observed. If abnormalities in the FHR had occurred Mrs Botha should have been removed from the birthing pool and continuous monitoring commenced. If there were abnormalities, the only means of expediting the delivery in a birthing unit would have been an episiotomy.

[38] Dr Westgate was also asked to comment on when an assisted delivery (with forceps for example) would have been possible after Mrs Botha had been in labour for 60 minutes. She said from 1300 hours onwards based on the position of the baby's head at that time.

[39] There were a number of questions about the fetal heart rate including if continuous monitoring was indicated. Dr Westgate said that she supported the RANZCOG guidelines and in this case because there was meconium stained liquor.

[40] In concluding remarks Dr Westgate said there was no doubt Kevin experienced an acute episode of hypoxia before delivery which resulted in bradycardia[14] which required external cardiac massage. The issue is whether this accounts for Kevin's difficulties entirely or even significantly. She said she did not know the answer to this question. But the absence of an encephalopathy and successful introduction to breast feeding suggests it was temporally related to a neurological disturbance. She suggested that a paediatric review be undertaken to try and resolve this issue.

Report by Dr Harshard Patel

[41] Dr Patel is Neonatal Paediatrician. His report is dated 12 July 2020. This was obtained by ACC as a result of Dr Westgate's suggestion of a paediatric review.

[42] Dr Patel described the events surrounding Kevin's birth and listed signs of his neurological status obtained from notes made by medical personnel caring for Kevin over the first 24 hour after his arrival at MMH. He listed five different observations and signs made of Kevin during that time. He said that none of the descriptions would fit with the presence of an encephalopathy.

[43] Dr Patel was of the opinion that Kevin's cerebral palsy was not caused by the acute hypoxic event before the delivery. His mild renal dysfunction is the only one of the criteria listed in the diagnostic table in the American Academy of Paediatrics/American College of Obstetrics and Gynaecology 1996. In particular there was no profound acidosis and the Apgar Score was not less than 3 at 5 minutes of age. He disagreed with Professor Battin that Kevin had a stage 1 encephalopathy. Features of which include being hyperalert, having a poor suck reflex and an exaggerated Moro reflex, being irritable and jittery. A baby with encephalopathy would not be feeding satisfactorily at 24 hours of age. The normal ph at 1.5 hours and an Apgar score of 7 at 5 minutes and the absence of multiorgan dysfunction rule out acute hypoxia as a cause of his cerebral palsy.

[44] He agreed that there had been a drop in liquor volume from the scan done at 38 weeks and 6 days which is a sign of intrauterine stress. He said it was difficult to say if the 13 days prior to Kevin's birth accounted for his cerebral palsy. But he was

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[14] Slower than expected heartbeat.

certain there was some placental insufficiency. He thought it was certain that whatever the cause, it was an antenatal event and not a perinatal event. He said that the midwife did an excellent job in assisting Kevin's breathing immediately after birth. He thought Kevin's hearing loss was due to the cerebral palsy and not anything that occurred perinatally.

[45] In terms of Dr Battin's concern about the temperature of the water pool, Dr Patel said it was not relevant because there was no encephalopathy. That the improved Apgar score at 5 minutes indicated Kevin responded well to resuscitation efforts and Kevin was breathing spontaneously at 2 minutes. With an Apgar score of 7 he thought it unlikely that peripartum hypoxia played a role in HIE.

[46] Dr Patel responded to two medical opinions that Kevin had a perinatal insult. One from Dr Tuck and another from a neurological registrar. In the first case Dr Tuck thought Kevin had suffered an hypoxic event during labour. Dr Patel said he did not agree because Kevin had a good Apgar score at 5 minutes and that the late arrival of the ambulance did not affect his oxygen saturations to a level to cause hypoxia. In the second case in relation to the statement that MRI findings showed a peri natal insult, Dr Patel said that the doctor had relied on incorrect information about when Kevin's breathing was established.

Second Report from Dr Malcolm Battin

[47] Dr Battin was responding to a question Kevin's lawyer had asked which was what difference would an earlier delivery of 5-8 minutes have made to Kevin's condition. His report is dated 26th January 2021.

[48] Dr Battin said that an earlier delivery by 5 to 8 minutes would mean Kevin would have been less likely to have been affected by cerebral palsy. His reasons included:

[i] Kevin was in sub optimal condition when born, specifically his Apgar score of 2 at one minute and 7 at five minutes;

[ii] he required resuscitation;

[iii] a blood test at MMH recorded an elevated lactate result suggesting a degree of compromise;

[iv] the presence of thick meconium;

[v] the postnatal documentation of nucleated blood cells in circulation; and

[vi] the renal impairment.

[49] Dr Battin explained that if cord blood gases had been taken and there was no record of the fetal heart rate during the last 15 minutes of birth, it was difficult to be certain about the proportion of injury associated with the acute episode compared to prior event. He considered that the contribution from the acute peripartum event was significant in Kevin developing cerebral palsy.

[50] He explained that in utero the fetus is able to cope with a decrease in oxygen supply. The fetus has higher haemoglobin and therefore is able to increase oxygen levels (FHR is higher than that of adults). These two mechanisms mean that the fetus can cope with a modest degree of hypoxia. Tissues extract increased fraction of the oxygen available and blood flow is redirected to the brain, heat and adrenal glands and away from the skin, gut and kidneys. The fetus can decrease activity levels to decrease the metabolic requirements in response to hypoxia. This is a late response to hypoxia. A healthy fetus can compensate for short term problems during labour, longer term, the supply needs to match the metabolic requirements to avoid compromise. Once the compensatory mechanisms are over-run, the condition rapidly deteriorates and the fetus develops problems including neurological injury. Where there is placental insufficiency, when reserve capacity is already used up, the fetus will poorly tolerate extra stress. If an insult during labour is extreme or prolonged, a previously healthy fetus may be compromised. Considering the evidence of sub optimal condition at birth and the knowledge of fetal physiology, an earlier delivery by 5 – 8 minutes would have resulted in less severe acute insult and better condition at birth.

[51] Dr Battin was asked what difference there would have been to Kevin's condition if he had been born at MMH. Dr Battin referred to the reports of Dr McEvoy and Dr Westgate in particular with regard to FHR monitoring considerations, consultation and pool water temperature.

[52] In terms of neonatal care, Dr Battin thought there would have been differences including a request for cord gases to document status at birth. This is standard practice with low Apgar scores and the need for resuscitation. Cord gases give objective evidence of status at birth and would have informed the plan for subsequent observation and detection of any encephalopathy. At the same time Dr Battin recognised that the resuscitation efforts were successful in improving Kevin's condition. If these were not performed well, the outcome would have been worse. Placental histology could also have been performed if the birth was at MMH and given more information on the potential cause of slowing growth and or low liquor.

[53] The Doctor was asked about the possibility of an earlier diagnosis of encephalopathy. Neonatal review of Kevin was at 1 hour of age. If his condition had improved over this time, previously present abnormal findings may not be manifest. That time delay would also allow blood gases to improve. The blood gas[15] result had a base excess of -11. It would have been worse if sampled earlier. If a diagnosis of encephalopathy had been made, monitoring and follow up could have been different.

[54] Dr Battin agreed with Dr Westgate's opinion that there had been a fall off in growth in the last 13 days prior to delivery. But he still thought that intra partum[16] hypoxia had a more than minimal contribution, it was significant enough to cause renal impairment.

[55] In relation to an MRI that had been taken of Kevin's brain, Dr Battin said that it was helpful in documenting abnormal appearances including the thalami and white matter consistent with hypoxic ischaemic insult. These appearances were consistent with hypoxic ischaemic injury. Kevin had been well investigated by the medical

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15 A blood gas less than -12 is a sign of acidosis.
16 During birth.

team and no other cause had been identified. Therefore, on the balance of probabilities the intrapartum events had more than a minimum contribution.

[56] Dr Battin was asked to comment on Dr Patel's opinion and the literature he had relied on. Dr Battin said that Dr Patel had referred to older publications (1996 and 2003). He said the diagnosis of intrapartum hypoxia was very restrictive and no longer accepted to reflect current knowledge. The most recent edition The Task Force on Neonatal Encephalopathy determined that a broader approach might be more fruitful. Knowledge gaps still preclude a definitive test or set of markers that accurately identifies an infant in who neonatal encephalopathy is attributable to an acute intrapartum event. He did not agree that rigid criteria were required to make a diagnosis of intrapartum hypoxia. Moreover the criteria are a guide. When a cord gas had not been obtained, it should not be assumed there is no evidence of asphyxia.[17] The blood gas was suggestive of metabolic acidosis being close to -12 threshold plus the evidence of an elevated lactate level. On the balance of probabilities it was worse at birth. The criteria of the Apgar score of below 3 at 5 minutes is too restrictive and has not been used in revisions over the last 20 years. Signs of encephalopathy can be missed or misinterpreted, such as irritability. In his opinion, Dr Battin thought that the perinatal events were more likely the origins of the cerebral palsy.

Submissions for the Appellant

[57] An hypoxic insult causing neurological injury resulting in cerebral palsy qualifies as a personal injury that is a physical injury and should attract cover.[18] In *Brown* the court directed cover be granted for brain damage caused by HIE, for a delivery at a birthing unit where tertiary care was indicated.

[58] There were the following treatment failures;

[i] Failure to adequately monitor the fetus in the ante partum period. This is a key failure that could have been avoided and it would have mitigated Kevin's injury and of itself is sufficient for a finding in favour of cover.

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17 Deficient supply of oxygen arising from abnormal breathing.
18 *Brown v Accident Compensation Corporation* [2020] NZACC 171.

[ii] The use of the birthing pool outside the guidelines, it was contraindicated due to the meconium. The temperature of the water was too high and it was not checked again for another hour.

[iii] Failure to obtain informed consent to deliver at a birthing unit.

[59] The nub of the issue is whether the failures caused the injury condition of cerebral palsy. This essentially rests on whether to accept the evidence of Dr Patel or Dr Battin. Associate Professor Battin is a practising neonatologist at New Zealand's largest children's hospital. He is also a leading researcher in the area of neonatal neurological injury and treatment. He has provided clear nuanced reasoning as to why Kevin's cerebral palsy is more likely than not caused by an acute hypoxic event.

[60] Dr Patel has referred to older research. He was incorrect to state that criteria were not present when we do not know because no cord gases were obtained. Dr Patel stated that there were no abnormalities detected on intermittent auscultation, but Dr Westgate says acute hypoxia occurred in the last 15 minutes before birth when no FHR monitoring took place. Professor Battin agreed with Dr Westgate.

[61] Dr Battin used the blood gas result taken at MMH to extrapolate backwards as to what the cord gases would have told us if taken immediately after birth. This is a measured non-absolutist approach appropriate in the Accident Compensation jurisdiction. Had the birth taken place in a hospital, cord blood gases would have been taken. This is significant given the failure to obtain informed consent to birth in a lesser equipped environment.

[62] It is therefore submitted that where treatment failure, being a departure from a standard has deprived an injured child claimant vital information to support their claim, the benefit of the doubt should be given to them. This would be consistent with the principles of equity.

[63] Dr Battin's opinion that the elevated temperature of the water in the pool may have contributed to fetal distress by increasing the metabolic demand and oxygen requirement should be accepted. This is an additional failure, causing injury.

[64] It is submitted that the Court should take the approach alluded to by Dr Westgate, where there are equally competing opinions on causation in the face of a clear treatment failure, preference should be given to the one that benefits the injured child. This is in accordance with the Court of Appeal's generous and unniggardly approach which acts as a "thumb on the scales" in such cases.[19]

[65] Courts can infer causation where experts cannot.[20] In the case of this injury there are strong policy reasons to make a finding of causation. Despite the difficult and complex medical circumstances, the claimant has marshalled reasonable evidence in support of the claim. Such cases necessarily involve some deal of speculation and theoretical analysis because we are dealing with things that did not happen as they should have.

[66] In *Black*[21] Judge Joyce, QC, found that in a borderline case, the fact of the distinctly dramatic event, coupled with the specialist's support opinion, tipped the scales in favour of the claimant. The same approach should be applied in this case.

[67] Mrs Botha was not given informed consent about the choice of birthing venues. In *Shand*[22] Cull J recently noted, on this issue:

> The patient must be given sufficient information of the anticipated benefits and risks of the proposed treatment so she can consider options and make an informed decision. Failure to obtain informed consent requires a departure from a proper standard of care, namely the provision of sufficient information about the benefits and risks of treatment.

[68] There were three points at which there was a failure to provide an informed consent:

(1) The ante-natal discussion with the midwife about where to birth;

(2) The point at which Mrs Botha's risk increased with the meconium and lack of progress by pushing; and

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19 *Accident Compensation Corporation v Ambros* [2007] NZCA 304, [2008] 1 NZLR 340. *Accident Compensation Corporation v J* [2017] NZCA 441 at para [52].

20 *Ambros* at [69].

21 *Black v Accident Compensation Corporation* [2012] NZACC 222.

22 *Shand v Accident Compensation Corporation* [2020] NZHC 2743 at [45].

(3) Entering a birthing pool.

[69] Dr McEvoy points out in his report that even in a low risk pregnancy, medical emergency such as fetal distress, cord prolapse, abruption of the placenta can occur and birthing units are potentially not able to cope. Dr Westgate said that a woman should be informed around the risks with complications in a time sensitive nature. There is no clear information in the notes about whether the pros and cons to deliver at a birthing centre as opposed to a hospital were discussed with Mrs Botha. Her evidence was that she would have been interested to know about these risks.

[70] There is no documentation of the increased risk when the meconium was observed, and no record of Mrs Botha being advised about the risk of entering the birthing pool, i.e. the difficulty monitoring the fetal heart rate, when continuing monitoring was indicated let alone the fact that entering the pool was contra indicated. It is submitted that had Mrs Botha given birth at hospital it is more likely that she would have been given continuous fetal heart monitoring and had an earlier delivery mitigating or avoiding injury to Kevin.

Submissions for the Respondent

[71] The Corporation accept that there have been failures in the care provided to Mrs Botha during labour. They are:

[a] Mrs Botha did not provide informed consent to giving birth at a primary unit and should have given birth at the hospital instead;

[b] It was inappropriate to use intermittent auscultation to monitor the fetal heartbeat during labour; it should have been constantly monitored electronically;

[c] It was inappropriate to use a birthing pool which was too warm; and

[d] Mrs Botha did not provide consent to this.

[72] It is the position of the Corporation that even if the alleged failures do constitute a departure from a standard, they do not have any causative effect for

perinatal asphyxia to cause cerebral palsy, the baby must suffer a neonatal encephalopathy.[23]

[73] Dr Patel states that the evidence does not support an encephalopathy. Dr Battin disagrees. In ACC's submission, Dr Patel's evidence should be preferred because:

(1) Dr Battin's evidence is on its face speculative;

(2) Dr Patel identifies features of Kevin's condition (in particular that he was feeding satisfactorily at 24 hours after birth) which are incompatible with an encephalopathy;

(3) Dr Battin's evidence fails to address these facts; and

(4) The other evidence of Kevin's status at birth does not support a diagnosis of severe intrapartum hypoxia. In particular he did not have profound metabolic acidosis and his Apgar score at 5 minutes of age was good; and

(5) While the events surrounding the time of Kevin's birth might been a minor departure from the standard, they did not cause Kevin's cerebral palsy.

[74] Dr Lorna Davies (PhD), a registered midwife gave expert evidence on the quality of care provided to Mrs Botha on the day of Kevin's birth, 26 January 2016, she said the care was of a good standard and she did not think a different treatment path or referral would have changed the outcome. It was appropriate to use a birthing unit rather than a hospital because Mrs Botha was a low risk patient. In terms of whether or not Mrs Botha consented to the use of the unit, Dr Davies suggested it was possible that the lack of record of informed consent was a shortfall in documentation and did not actually reflect a failure to obtain Ms Botha's informed consent. The presence of meconium was not an indication to transfer to secondary care and the fetal heartrate was satisfactory throughout, which supports the conclusion that the meconium present was benign.

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[23] A natal encephalopathy is "a clinically defined syndrome of disturbed neurological function in the earlier stages of life", in the term "infant manifested by difficulty with initiating and maintaining resperation, depression of tone and reflexes, sub normal level of consciousness and often seizures." – as explained by Dr Battin.

[75] Dr Battin provided an updated report in which he said there was a possible mild hypoxic encephalopathy, which staff had missed. The basis for this view was:

[a] The evidence of systemic renal injury during birth based on Kevin's creatinine levels on 27 and 28 January. This followed a period of metabolic stress which was demonstrated by a lactate of >4 on 26 January at 1550 (one hour and 45 minutes after Kevin's birth). He acknowledged that the severity of the stress could not be ascertained because less than 4 is a ceiling measurement and the umbilical cord gases were not taken. He said that it is reasonable to conclude that the perinatal events, poor Apgar scores and requirement for resuscitation were linked to the subsequent renal impairment.

[b] The limited neurological examinations recorded are inconsistent for moderate or severe encephalopathy. They could be consistent with a mild encephalopathy. This is much more subtle in presentation. He notes that on 28 January, Kevin was recorded as crying a lot, which could support a more subtle HIE with transient examination finding. An MRI on 27 July did not show evidence of a neurological injury but that does not exclude the possibility to HIE. A reasonable proportion of babies with mild HIE will not have an abnormal MRI.

[c] Dr Battin was critical of the use of the birthing pool on the basis it was too warm. This can cause a baby to suffer an increase in metabolic demand which may predispose the baby to asphyxia. In Dr Battin's view, the pool should not be greater than 36.5 degrees at the time the mother enters the water. The note shows the temperature was 38 degrees. It had reduced to 37 degrees at 1338 hours.

[d] He did not have access to any electronic fetal heart monitoring. However the intermittent readings were within the expected range. He said there was no avoidable delay in diagnosis or treatment that could have contributed to Kevin's injury.

[76] Dr Davies did not think electronic fetal heart monitoring was indicated. Electronic FHM is inappropriate in the absence of identified risk factors and does not meet the requirements of a good screening test of what is a legitimate problem and what is not. This can lead to unnecessary intervention. Dr Davies accepted that the water pool was too warm but did not accept this contributed to Kevin's injuries.

[77] Dr McEvoy said that:

[a] It did not appear Mrs Botha had given informed consent to the use of the pool or the birthing unit.

[b] That intermittent auscultation during a water birth is difficult. This would have been avoided had electronic FHM been used.

[c] While the temperature was one degree higher than the maternal temperature, this can theoretically increase metabolic demand for oxygen by the fetus. In an already compromised fetus this additional factor might have played a part in increasing the likelihood of hypoxic injury to the fetal brain.

[d] When meconium appeared, an obstetric referral should have been obtained and electronic fetal monitoring should have been initiated. This would have identified fetal distress earlier at 1140am and resulted in assisted delivery rather than waiting for a spontaneous delivery at 1405, reducing Kevin's exposure to any hypoxia in utero by 85 minutes.

[e] The blood cord gases should have been performed for educational purposes and because they are useful to determine medico-legal issues.

[78] Professor Westgate provided a report in which she said that:

[a] Despite the Counties Manukau CMDH guidelines, the presence of meconium meant that electronic fetal heart monitoring was indicated.

[b] To the extent that intermittent auscultation was appropriate, the fetal heart rate should have been taken every 5 minutes and this did not happen.

[c] It was unlikely that electronic FHM would have revealed concerning information prior to 1:50 pm. The heartbeat that was taken did not show any signs of fetal distress.

[d] Had electronic FHM been used or had it been taken at regular 5 minute intervals in the last 15 minutes of labour, this would most likely have identified signs of fetal distress which would have expedited

[e] delivery by 5 – 8 minutes, reducing Kevin's exposure to hypoxia.

[f] She disagrees with Dr Battin's view that the birthing pool temperature could have exacerbated Kevin's hypoxia. If there had been an increasing metabolic demand, the fetal heartrate would have increased to between 160 and 170. There is no evidence the FHR increased significantly. On that basis the pool was unlikely to have led to an increase in Kevin's metabolic demand.

[79] Dr Patel said that:

[a] Kevin's injuries were not caused by hypoxia during labour. He said "it can be said with certainty that the pathway from an intrapartum hypoxic injury to subsequent cerebral palsy, it must progress through a neonatal encephalopathy".

[b] The clinical evidence in the first days of Kevin's life did not support a conclusion he had suffered from a neonatal encephalopathy because:

[i] He was reactive to handling and his tone and movement were appropriate on admission to MMH;

[ii] At 6 pm on 26 January 2016 he was described as more settled;

[iii] On the second day he was unsettled and wanting to eat/suck;

[iv] He was assessed neurologically as being normal on 27 January. The neonatal specialist recorded "introduce breast feed and reduce IV drip and transfer to the ward this afternoon";

[v] On 27 January his grasp, suck and moro reflexes were normal. Later that day a breast feed was achieved and Kevin was transferred to the ward;

[vi] On 28 January Kevin was crying a lot, clinically alert, focuses well, following, tone normal and good suck on finger.

[c] By reference to a 1996 report, by the American Academy of Paediatrics/ American College of Obstetrics and Gynaecology, Dr Patel identified three other factors that are necessary to link hypoxia at birth to the development of cerebral palsy. As to those he said:

[i] First blood gas that was taken did not show that Kevin had suffered from profound metabolic acidosis. He had a normal PH (7.29) and his base excess of -11 is not sufficient for a conclusion of acidosis.

[ii] Although the Apgar score at one minute was poor, it was good at 5 minutes after birth (7). To link perinatal events to his injury he would have needed an Apgar score of less than 3 at 5 minutes after birth.

[iii] Kevin had mild renal dysfunction which supported a conclusion of multi-organ system failure.

[d] It was Dr Patel's view that access to paediatric care would not have made an impact on the outcome for Kevin. Resuscitation efforts at the unit were appropriate and Kevin's condition was stabilised.

[80] Dr Battin provided a second report in which he agreed with Dr Westgate and Dr Patel it was likely there was an element of chronic sub-optimal status in utero.

[81] He said that neither he, nor Dr Patel could be certain as to whether Kevin had an encephalopathy as they were reliant on their interpretation of the clinical notes. He observed that signs of encephalopathy such as irritability can be missed or misinterpreted.

[82] Dr Battin said that on the balance of probabilities the intrapartum events had more than a minimum contribution to Kevin's cerebral palsy. He disagreed it was appropriate to use the four factors identified by Dr Patel. There is a contemporary acknowledgement that a definitive test or set of markers cannot accurately or specifically identify when a neonatal encephalopathy is attributable to an intracute partum event. The use of an Apgar score of 3 or less at 5 minutes is too restrictive. The presentation at the hospital was suggestive of metabolic acidosis. There was evidence of an elevated lactate level. Kevin's base excess of -11 was very close to -12 after 90 minutes, a time in which he could have recovered. Kevin's multi-organ failure was evidenced by the renal injury. He stated his view that the use of the pool was not at the expected standard.

The Law

[83] It was said in *Adlam v ACC:*

> … in order for there to be treatment injury as a result of a failure to provide treatment it is necessary to show that an alternative treatment that would have prevented the injury suffered could and should have been given having regard to the clinical indications at the time of the alleged failure.

[84] The Court of Appeal decision in *Ambros* applied in this case. Where expert opinion diverge the Court's assessment must be entirely on the papers. ACC observes that this is an issue that has been considered by *ACC v Mehrtens,*[24] in which Judge Ongley said:

> … In relation to the medical evidence, particularly in an area where an opinion is relied upon, the Court will be influenced by the extent to which the medical opinion proceeds logically from as clear or settled a basis of fact as is possible (including the possible need for caution when significant reliance is based on a claimant's self report); appropriate analysis of that material including, where necessary, the presentation of a differential diagnosis; an appropriate level of regard for and consideration of medical research and studies bearing on the

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24 *Accident Compensation Corporation v Mehrtens* [2012] NZACC 250 at [48].

> issue at hand applied to the particular facts of the case; and a logically reasoned conclusion which takes account of any differing views or factors which might contra indicate the opinion being presented. In this respect, an opinion which is seen to absorb and respond to matters (whether matters of fact or opinion) which challenge the view offered, will often be regarded as more persuasive.

[85] In order to establish cover, the claimant will have to be able to show:

[a] A departure from the appropriate standard of treatment in the care provided to his mother during pregnancy or during labour; and

[b] The treatment provided to him and his mother caused him to suffer acute hypoxia.

[86] As is previously set out, ACC acknowledges that there were failures from appropriate standards. The question is whether the identified failures to provide treatment caused Kevin's cerebral palsy. This will largely turn on whether the Court prefers the evidence of Associate Professor Battin or Dr Patel.

[87] It is ACC's position that Dr Patel's evidence should be preferred. Dr Patel's conclusion was that Kevin's injuries are more likely to have been caused by an ante-natal event rather than treatment during his birth.

[88] It is common ground that an encephalopathy was not identified at the time of Kevin's birth or shortly thereafter. The question therefore is whether the Court can infer that there was an encephalopathy from the clinical notes as interpreted by the experts. Dr Battin suggests that there is evidence of a mild encephalopathy. In his view:

[a] Systemic renal injury is evidenced by high creatine levels following a period of metabolic stress. The timing of the creatine levels can reasonably be linked to some renal impairment.

[b] The limited neurological examinations could be consistent with a mild HIE. In particular Kevin was recorded as crying a lot at 3 days of age. Dr Battin notes this may be a sign of irritability.

[c] Kevin's MRI scan at 2 years of age showed some abnormal features.

[d] Kevin has moderate hearing loss which can occur with moderate to severe asphyxia. However, as Dr Battin acknowledges, this cannot be easily explained by a mild encephalopathy.

[89] In relation to these factors, Dr Patel:

[a] acknowledges Kevin had mild renal dysfunction which was caused by hypoxia during labour;

[b] acknowledges that irritability can be a finding of encephalopathy. He also identifies the following additional signs for a stage one encephalopathy being:

[i] being hyper-alert;

[ii] having a poor suck reflex; and

[iii] having an exaggerated moro reflex.

[c] disagrees with Dr Battin's view that Kevin crying a lot on day 3 was evidence of irritability. He could have been crying due to hunger as his feeds were being established;

[d] acknowledges there is evidence of hypoxic injury on an MRI scan. But says this was not due to a perinatal insult.

[e] he says Kevin's hearing loss was not caused by acute hypoxia.

[90] Dr Patel identifies a number of facts that are inconsistent with Kevin having suffered an encephalopathy:

[a] the admitting doctor described his reaction to handling, tone and movements appropriate for gestation at MMH;

[b] that evening a nurse records that "baby more settled";

[c] the next day the nurse records “he was unsettled and wanting eat/suck”;

[d] on the morning of 27 January Kevin was introduced to breast feed, reduced IV drip and transferred to the ward in the afternoon;

[e] on 27 January medical staff observed Kevin’s grasp, suck and moro reflexes were normal; and

[f] at age 24 hours his breathing was recorded as comfortable and a category f breastfeed had been established.

[91] Dr Battin acknowledges that Kevin’s handling tone, moro reflex and grasp reflex are inconsistent with a moderate or severe encephalopathy, but suggests they can be reconciled with a more subtly presented mild encephalopathy. He does not comment in either of his reports on the relevance of the fact that Kevin was feeding satisfactorily at 24 hours of age. This is despite the fact his second report was evidence in reply to Dr Patel’s report, where Dr Patel stated that satisfactory feeding at 24 hours of age is inconsistent with an encephalopathy.

[92] Dr Patel’s evidence should be preferred because Dr Battin’s evidence amounts to an observation that Kevin was crying a lot on the third day of life, which could have meant he was irritable, which in turn is a possible sign of a “subtle encephalopathy”. This is highly speculative. Dr Battin fails to comment on uncontroversial facts that Dr Patel explains are inconsistent with Kevin having suffered a mild encephalopathy. In particular he was breast feeding at 24 hours of age.

[93] Dr Patel identifies a range of other factors he says are necessary to link perinatal hypoxia to cerebral palsy through a diagnosis of intrapartum hypoxia. They are:

[a] profound metabolic acidosis;

[b] an Apgar score less than 3 after birth; and

[c] multi-organ dysfunction.

[94] The Court is not asked to determine whether a neonatal encephalopathy was caused by an acute intrapartum event, rather it must determine whether Kevin's cerebral palsy was caused by hypoxia suffered in the final minutes of labour and caused to some extent by the alleged departure from the standard.

[95] Both Dr Patel and Dr Battin agreed the umbilical cord gases would have been useful to determine whether Kevin had profound metabolic acidosis. In their absence, they rely on the blood gas results of around an hour of age and Dr Patel also relies on Kevin's blood pH. The doctors both agreed that the commonly accepted threshold for a diagnosis of metabolic acidosis is a base excess of -12. At an hour and a half of age Kevin had a base excess of -11. Dr Battin says that because this was taken an hour after Kevin's birth, it can be inferred he would have recovered in that period and that this base excess would have been worse immediately after birth. But he does not provide any evidence of the rate at which he would expect new born babies base rate reading to recover to within the normal range. Nor does he respond to Dr Patel's comment that Kevin's pH of 7.29 was normal.

[96] Dr Patel's evidence on this point should be preferred. He has made use of all the clinical tests available to him in forming his opinion.

[97] In terms of the Apgar score after five minutes of birth, a score of 3 is regarded as poor. Dr Battin rejects this as a criteria on the basis that an Apgar score of 3 is too narrow. He does not comment on the fact that Kevin's Apgar score at five minutes of life was 7. Dr Patel describes this as good. Both doctors agree that the evidence of renal failure or dysfunction supports the finding of multi-organ system dysfunction.

[98] On the temperature of the birthing pool, Dr Westgate's evidence should be preferred to that of Dr Battin, who is not an Obstetrician, and of Dr McEvoy. Dr Battin and Dr McEvoy's evidence goes no further than to identify a theoretical possibility that the pool's temperature could increase the fetal metabolic demand which would expose Kevin to hypoxia.

[99] Professor Westgate agrees that internal temperature can increase the fetal metabolic demand. However, she provides a detailed explanation as to why that did not occur in this case. That included that if the temperature of the pool had an adverse impact on Kevin's FHR, it would have increased dramatically and that didn't happen. This means that the use of the pool did not increase Kevin's metabolic demand or expose him to hypoxia.

Alternative cause of Kevin's injuries

[100] Dr Westgate and Dr McEvoy agree that Kevin's birth weight of 2910gms is evidence of a period of placental insufficiency which would have caused a period of chronic hypoxia in the last two weeks of pregnancy. Dr Westgate suggests that chronic hypoxia is a possible cause of Kevin's injuries. But she defers to paediatric opinion on the relative contributions of the causes of Kevin's injuries. Dr Patel states it would be impossible for him to wholly attribute Kevin's injuries to the two week period between his final scan and birth. But he accepts that placental deficiency would have contributed to Kevin's injuries. Dr Patel says that antenatal events are a far more common cause of cerebral palsy than acute perinatal asphyxia.

[101] Dr Battin agrees the evidence is consistent with Kevin having suffered some degree of chronic suboptimal status in utero. He states that perinatal events are more likely to be the origins of the cerebral palsy than an unrecognised cause occurring earlier in pregnancy.

[102] ACC submits that the uncontroversial placental insufficiency in the final two weeks of pregnancy, combined with Dr Patel's evidence on why perinatal events should not be considered the cause of Kevin's injuries, provide a plausible alternative cause of Kevin's injuries, unrelated to any of the alleged causative failures of treatment. If there was also a perinatal contribution to the injury, there is no reliable basis to conclude that it arose from any of the alleged failures of treatment.

Reply Submissions by the Appellant

[103] Where there are failures to treat that relate to protocols designed to avoid injury, then there is a prima facie case of causation. Acting on distress indicators

such as meconium, adequate FHR monitoring, correct pool temperature, not using a birthing pool when contraindicated, providing informed consent about where to birth and all appropriate points are all protocols designed to avoid fetal hypoxia and injury. FHR monitoring allows an hypoxic event to be quickly identified and birth expedited. A brief delay in delivery can make a significant difference.[25] A birthing pool should be avoided in the presence of meconium, to enable more accurate monitoring. Correct pool temperature avoids placing metabolic demands on the fetus. Delivery in a hospital allows prompt access to obstetric expertise and equipment including forceps, and cord gas measurements.

[104] ACC submits that Associate Professor Battin's evidence is speculative. Every expert opinion requires a degree of educated speculation and theorising. Professor Battin's opinion is no more speculative than Dr Patel's. Ms Peck attached a table comparing the evidence of the two neonatologist experts. A robust and pragmatic approach to the evidence allows for a finding in support of causation.

[105] Not acting on the failures meant the opportunity was lost to avoid subjecting Kevin to further metabolic stress when he had already used up his reserves. Dr Patel acknowledges he cannot wholly attribute Kevin's injuries to the two weeks prior to birth. Professor Battin says that Kevin was less likely to have been affected by cerebral palsy had he been delivered 5 – 8 minutes earlier. Cover should be granted for Master Botha.

Analysis and Discussion.

[106] I refer to the legal test for a treatment injury which is as a result of the failure to provide treatment. In *Adlam*, the Court of Appeal considered the combination of sections in the Act which are relevant to treatment injury cases. Section 20(1) and 20(2)(b) provide cover for physical injuries that is a "treatment injury". That term is defined in s 32(1) as:

> **32 Treatment injury**
>
> (1) Treatment injury means personal injury that is—
>
> (a) suffered by a person—

[25] See *Buckley v Accident Compensation Corporation* [2019] NZACC 100.

(i) seeking treatment from 1 or more registered health professionals; or
(ii) receiving treatment from, or at the direction of, 1 or more registered health professionals; or
(iii) referred to in subsection (7); and

(b) caused by treatment; and

(c) not a necessary part, or ordinary consequence, of the treatment, taking into account all the circumstances of the treatment, including—
(i) the person's underlying health condition at the time of the treatment; and
(ii) the clinical knowledge at the time of the treatment.

[107] Section 32(2) and (3) provide:

(2) Treatment injury does not include the following kinds of personal injury:

(a) personal injury that is wholly or substantially caused by a person's underlying health condition:

(b) personal injury that is solely attributable to a resource allocation decision:

(c) personal injury that is a result of a person unreasonably withholding or delaying their consent to undergo treatment.

(3) The fact that the treatment did not achieve a desired result does not, of itself, constitute treatment injury.

[108] Section 33 (d) states

33 Treatment

(1) For the purposes of determining whether a treatment injury has occurred, or when that injury occurred, treatment includes—

…

(d) a failure to provide treatment, or to provide treatment in a timely manner:

[109] In *Adlam* the Court said at para [61]:

…we consider the relevant provisions properly construed mean that in order for there to be treatment injury as a result of a failure to provide treatment it is necessary to show that an alternative treatment that would have prevented the injury suffered could and should have been given having regard to the clinical indications at the time of the alleged failure.

Failures to treat (s 33(1)(d))

[110] In this case, it is alleged that there were a number of treatment injuries which are failures to treat. They are:

[i] Failure to adequately monitor the FHR during the second stage of labour.

[ii] Failure to perform continuous fetal heart monitoring.

[iii] The birthing pool should not have been used including that Mrs Botha did not give her informed consent for this,

[iv] The temperature of the water in the pool was too high.

[v] Failure to obtain Mrs Botha's informed consent to the birth taking place in a birthing unit.

[111] There can be no dispute about the failure to perform FHR recordings every five minutes during the second stage of labour, that they were not taken is a matter of record. The respondent does not dispute that that there should have been continuous FHR monitoring electronically once meconium staining was noticed. The respondent also accepts that the use of a birthing pool that was too warm should not have occurred and that Mrs Botha did not consent to use of the pool. The respondent agrees that Mrs Botha did not properly provide informed consent to giving birth at a primary birthing unit. That leaves the third issue being that the birthing pool should not have been used at all which is closely connected to the appellant's concerns that the temperature of the pool was causative of the mild HIE which Kevin suffered at birth. I will come back to this issue shortly.

Were the Failures a departure from a standard?

[112] What standard applies can be unclear. It is the fact there was meconium staining in the forewaters that brings this issue into focus. Both Dr McEvoy and Dr Westgate agree that there should have been some action once the meconium staining was observed. Dr McEvoy said that the presence of low liquor and meconium stained liquor together and separately were an indication for a medical obstetric

review of Mrs Botha, continuous FHR monitoring, the presence of a paediatrician at delivery and the taking of cord blood. Dr Westgate said that had Auckland DHB guidelines applied or those of the RANZCOG[26] guidelines, there would have been continuous monitoring of the FHR. Continuous FHR monitoring would have noted a drop in the FHR during the better part of the second stage of labour and as Dr Westgate says (and Dr Battin agrees), Kevin would not have suffered the degree of hypoxia he did perinatally as an earlier delivery would have occurred.

[113] I pause here to say two things. First, I disagree with the Reviewer that the evidence of Dr McEvoy is not helpful. The standards of care he applies as a fellow of the RANZCOG are the same standards that apply in New Zealand. Dr Westgate makes that clear. Second, it is concerning to learn that the guidelines for midwives are different between DHBs in Auckland. It is my view that the CMDHB guideline on what should happen with the appearance of meconium stained liquor needs to be changed to at least bring it into line with the ADHB guideline. Dr McEvoy says that meconium staining occurs in 5% of all deliveries. In 5% (or 1 in 20) of those cases fetal asphyxia occurs (Dr Westgate's figure was higher 1 in 7 or 14%). While 95% of the time there is a good outcome, appropriate paediatric intervention with suction of the respiratory tract has been shown to reduce neonatal morbidity. No woman wants her baby to be the 1 in 20 who suffers from fetal asphyxia.

[114] Dr Westgate was not convinced there was a low amount of liquor in this case. But, Dr Davies said that it was documented there was little amniotic fluid present when the waters were broken. A document titled "Labour Details" in the common bundle of documents noted at 1140 "significant meconium, minimal water". Dr Davies said it was not unusual, in particular because the baby's head was still high, acting as a plug to stop liquor draining. Dr McEvoy said there was oligohydramnios (low liquor). This separately and together with the meconium staining are described by Dr McEvoy as strong indications of a possible hypoxic event. He said these signs were ignored by the midwife.

[115] I accept that Mrs Botha's pregnancy was low risk. But when there were signs of possible low liquor and meconium staining after the forewaters were ruptured, the

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[26] Royal Australian and New Zealand College of Obstetrics and Gynaecology.

midwife should have taken action to at least obtain another opinion, preferably a medical opinion about whether there needed to be any change in the birth plan. This was a departure from a proper standard of care.

[116] I turn to the issue of consent to use the birthing pool. Dr McEvoy said that this should be discussed with the mother during the antenatal period and the pros and cons explained. I ascribe no fault to Ms Donald as she did not have the time to do this in a fully informed way on the day. However, the pool was not the correct approach given the low liquor and the meconium staining. Mrs Botha should have had continuous FHR monitoring at this point and not in the pool. This also is a departure from a proper standard of care.

[117] It is conceded that the temperature in the pool was too high. The water should have been cooled before Mrs Botha got into the pool. Given what occurred on the day, I accept what Dr Battin says about the potential dangers of having a mother in labour in water that was too hot. It posed a risk both for the mother and the baby. The midwife Ms Donald accepted 38° was too high. It was a departure from a proper standard of care.

[118] However, I accept Dr Westgate's view that despite the water in the pool being too hot and that Mrs Botha's temperature rose, it was not a causative factor, for the reasons she explained, which are set out in paragraph [32] herein.

[119] The last issue is the failure to obtain Mrs Botha's informed consent to the venue for delivery. In an affidavit by Mrs Botha dated 18 January 2018, she stated that she and her husband told their midwife that they wanted the birth to happen in a hospital, where they would have everything they needed. The midwife replied that sometimes the hospital wanted the birth to happen as fast as possible. She said last time she helped a woman deliver at hospital she had to go round the hospital to find a room. She advised Mr and Mrs Botha that there was everything they would need at the birthing unit. Mrs Botha asked what would happen if she needed to have a c-section. She was assured that she would be taken to the hospital in an ambulance and there would be plenty of time to get there. Mrs Botha stated the midwife did not state any risks of not giving birth in the hospital.

[120] At the Review hearing Mrs Botha said that in China (Mrs Botha is Chinese) and in South Africa (where the couple came from) births occurred in hospitals. She had suffered one miscarriage and said, "we were obviously concerned". I am satisfied that Mrs Botha's preference was to have given birth in a hospital.

[121] Mr and Mrs Botha gave evidence about the background to choosing the midwifery model of care at the Review hearing. Mr Botha said that when they went to the Doctor's office and it was confirmed that Mrs Botha was pregnant, "… they said there was two ways to go: one was with the obstetrician, but the way most people go was with a midwifery – going to midwifery or a midwife, and she recommended someone for us as well …." He was asked if they considered an obstetrician or going through the public system. Mrs Botha answered that she did not know that hospitals provide care (for pregnant women). Mrs Botha went on to say that they were told by a lot of people that the midwives are experienced and capable and had a good name. Mr Botha said that when they met their midwife who cared for Mrs Botha during her pregnancy she said that one option was delivery at the midwifery and the other one was the hospital. He also said:

> My wife and I were pretty adamant we wanted to go to the hospital as that is the way they do it in China and … South Africa and we were really concerned about the risks to our child."

[122] As previously stated, Mrs Botha had already suffered a miscarriage and she said at the Review hearing she had waited "five years for this child". Mrs Botha said the midwife told them that the hospital had drawbacks, not giving you enough time for a natural birth as mentioned. If anything went wrong or you need to go to hospital that will be identified early. That staff in the birthing units were capable. Mr and Mrs Botha were unfamiliar with the New Zealand options for planning the pregnancy care and birth of their child. From the time they saw their GP they were pointed in the direction of the midwifery model and once they met their midwife that option was reinforced.

[123] I am satisfied that Mrs Botha was not given fully informed consent about the options for delivery and after care for herself and her unborn child. Dr McEvoy has prepared an in depth report which covers all aspects of Mrs Botha's care during pregnancy and at the delivery. On the issue of informed consent, Dr McEvoy noted

that it was not clear whether Mrs Botha understood that if she gave birth in the birthing unit that she would not have medical input if required. Dr McEvoy's said that (in this case) the decision to deliver a low intensity midwifery led care model is crucial to the outcome. He was critical of the fact there was no consent form or information leaflet that outlined the various models of care and their implications for emergency medical obstetric intervention, pointing to the fact that when there was meconium stained liquor this should have happened.

[124] Dr McEvoy is a Fellow of the RANZCOG. He has 35 years' experience as an obstetrician and gynaecologist. He is an examiner and member of the expert witness panel of the RANZCOG. He is a member of a South Australian human research and ethics committee and a member of the South Australian Health Practitioners Tribunal. He is a training supervisor for international medical graduates. Not only is he is amply qualified to give expert evidence in this case. I accept his evidence on this issue and make a finding that Mrs Botha had not given fully informed consent to give birth in a birthing unit as opposed to a hospital. This was another departure from a proper standard of care.

[125] To summarise, if the above treatment failures (excluding the 38° temperature of the birthing pool) had not happened, Mrs Botha would have either always planned to have her child in a hospital or she would have been taken to MMH at the time the meconium stained liquor occurred and a medical opinion sought at that time. Had continuous FHR monitoring been instituted as soon as possible after the meconium staining was noticed, then Kevin could have been transferred for a delivery in hospital assisted by ventouse or forceps. Had Mrs Botha remained at the birthing unit, as soon as the FHR dropped to concerning levels an episiotomy and an earlier delivery could have occurred. It is clear from the evidence of Dr Westgate and Dr Battin on this issue that Kevin would have been born in better condition and indeed may have avoided an acute hypoxic event at birth.

Did the failure cause a mild HIE

[126] Essentially there is a dispute between Dr Battin on the one hand and Dr Patel on the other as to the contribution placental insufficiency and acute hypoxic during delivery contributed to Kevin's cerebral palsy. I know Dr Patel is a Neonatal

Paediatrician but I have not been provided with any detail of his experience and qualifications. He referenced text books and three articles on relevant topics, one in 2004 and two in 2014.

[127] Dr Battin obtained his medical degree at the University of Liverpool in the UK in 1986. He has worked in the area of paediatrics all of his career. Neonatology is his primary specialty. He became a member of the Royal College of Paediatrics and Child in London in 1996 and a Fellow in 1997. He is a Fellow of the equivalent College in Australia. He came to New Zealand in 1997. He lectured in Paediatrics/Neonatology for several years. He has undertaken additional study in New Zealand obtaining qualifications in Public Health. Since 2008 he has worked with newborn babies as a neonatologist. Since 2009 he has held the post of honorary Associate Professor of Paediatrics and Child and Youth Health at the School of Medicine in Auckland. He has longstanding research in neonatal neurology. He has been the recipient of awards and has been involved in co-authoring numerous published articles in his field of expertise including neonatal encephalopathy. He states that much of his research has involved term infants with neonatal encephalopathy. He is well qualified to give expert evidence in this case.

[128] As between the two neonatal experts I am bound to take regard of Dr Battin's qualifications, published articles and experience. Published articles included one on treatment of mild encephalopathy published in a Pediatric journal in 2018. He co-authored another one on contributing factors and avoidable neonatal encephalopathy associated with maternal pyrexia 2016. I observe that Dr Patel was citing older literature and not any recent articles on HIE. I accept Dr Battin's statement that the criteria Dr Patel referred to in terms of criteria to make a diagnosis of intrapartum hypoxia as very restrictive and no longer accepted to reflect current knowledge. Further that it is not appropriate to consider something that had not been done as a negative for that criteria. Specifically cord blood gases in Kevin's case. I also accept that the Apgar score of below 3 at 5 minutes is too restrictive. Dr Battin says it has not been used in recent revisions for the last 20 years. He also states that signs of an encephalopathy can be missed or misinterpreted.

[129] There is an irony in the fact that the two things that could have settled this issue from a scientific perspective cord blood gases and placental histology did not happen because the birth did not occur in a hospital. Dr Battin says that because the FHR was not taken during the last 15 minutes prior to birth and the cord blood gases were not taken at birth, it is difficult to be certain about the exact proportion of injury associated with the acute episode at birth and prior events (during the pregnancy). Dr Battin goes on to say that based on an understanding of the fetal physiology and the acute peripartum event, his view is that on balance, the contribution from the acute peripartum event was significant in Kevin developing cerebral palsy. Dr Battin explained:

> In-utero, the fetus has a number of adaptive mechanisms that allow it to cope with a relative decrease in oxygen supplies. Specifically, the fetal blood has a relatively increased oxygen carrying capacity due to a combination of higher haemoglobin concentration and greater oxygen affinity of fetal haemoglobin. Fetal cardiac output (in proportion to the body weight) is not so much higher than in the adult. So the fetus can cope with a modest degree of hypoxia, but then this becomes more marked, there are further changes that occur in response. Firstly, the tissues extract an increased fraction of the oxygen available and secondly, blood flow may be redirected to the brain, heart, and adrenal glands and away from the skin, gut and kidneys. The fetus also decreases activity levels so decreasing metabolic requirements in response to hypoxia. However, this is a relatively late response to hypoxia. Although a healthy fetus can compensate for short term problems during labour, in longer terms supply needs to match metabolic requirements if compromise is to be avoided. Once the compensatory mechanism are "overrun" the condition rapidly deteriorates and the fetus develops adverse sequalae, including neurological injuries. So in situations of chronic placental deficiency, when reserve capacity is already utilised, the fetus will poorly tolerate any extra stress. Similarly, if an insult during labour is extreme or prolonged, then even a previously healthy fetus may be compromised.
>
> In summary, considering both the evidence of sub-optimal condition at birth and this knowledge of fetal physiology, my opinion is that an earlier delivery by 5 to 8 minutes would have resulted in less severe acute insult and better condition at birth.

[130] In his second report, Dr Battin says that in his view an earlier birth by 5 – 8 minutes would have been less likely to have resulted in Kevin being affected by cerebral palsy.

[131] Dr Battin was also asked what difference being born in MMH would have made to Kevin's condition. Dr Battin deferred to Drs Westgate and McEvoy in

terms of aspects of care during labour and delivery. In terms of neonatal care, he said:

> … in my opinion there would have been differences in postnatal care, including a request for umbilical cord gases to document status at birth, which would be standard practices following the low Apgar scores and requirement for resuscitation. Cord gases give objective evidence of status at birth and so would have informed the plan for subsequent observation and detection of any encephalopathy.

[132] Dr Battin was also asked what an earlier diagnosis of mild encephalopathy would have made. Dr Battin responded:

> Presentation for neonatal review was at around one hour of age. Previously present abnormal examination findings may not be manifest if the condition has recovered over the time. Also, the elapsed time would allow the blood gases to improve so delayed gases cannot be taken to indicate the worse degree of abnormalities. The blood gas result was noteworthy with a base excess of -11. Some people use -10 to indicate abnormal status, but it is more common to use -12 (this is used in the documents Dr Patel quotes). However, the result could have been worse if sampled earlier and closer to birth in timing.
>
> If a diagnosis of mild encephalopathy had been made, then monitoring and follow up may have been different. Although the threshold for active treatment was hypothermia this has shifted over time, the national consensus document does not advocate cooling in cases of mild encephalopathy. As far as I am aware, there are no national guidelines outside New Zealand that advocate treatment with cooling in mild encephalopathy. However, there are published reports of adverse neurological outcomes after a mild encephalopathy and recent discussion about a possible trial to document any potential benefits of coolings.

[133] As I understand it, cooling is used in cases of moderate and severe encephalopathy.

[134] Dr Battin was also asked whether the acute intrapartum hypoxia made more than a minimal contribution to Kevin's condition. He said that he thought that it had made more than a minimal contribution, pointing out that it was significant enough to cause renal impairment. He went on to refer to the MRI evidence. He said that the two scans were consistent with hypoxic ischemic injury, and that Kevin had been well investigated by the medical team and no other cause had been identified. Therefore, on the balance of probabilities the intrapartum events had more than a minimal contribution and the MRI is evidence of neurological injury causing Kevin's cerebral palsy.

[135] There is some support for Dr Battin's views in the opinions of Dr McEvoy and Dr Westgate. Dr Westgate said that Kevin had suffered a significant acute hypoxia immediately before birth. But she thought it was brief, which allowed a rapid recovery once he was provided with oxygen. She also said that Kevin suffered from chronic hypoxia in the last few weeks in-utero, but he compensated for this. She said Kevin entered labour with a reduced capacity to cope with reduced oxygen supply during uterine contractions. He was able to tolerate the first stage without deterioration. But in the second stage he had a sustained period of significant acute hypoxia, particularly in the last 15 minutes before his birth.

[136] Dr McEvoy said that Kevin's neonatal saturation of 71% was very strong evidence that an intrapartum hypoxic even occurred in this case. He pointed out that oxygen saturation was still low 85 minutes after delivery. Dr McEvoy was quite clear that there had been treatment failure and he described these as:

- No medical consultation;
- No continuous fetal heartrate monitoring;
- No request for paediatric presence at delivery; and
- No recognition that a water birth was contra-indicated.

[137] He said that the treatment was unsatisfactory and it appears that the treatment failures resulted in the injury described in this case. Dr McEvoy considered that the HIE was not wholly or substantially due to an underlying condition and that the injury condition was caused by intrapartum events.

Conclusion

[138] Taking the above 11 paragraphs together, I am satisfied on the balance of probabilities that Kevin's cerebral palsy was at least in part a result of the acute hypoxic ischemia he suffered at birth and a resulting mild HIE. I am also satisfied

that the HIE made a significant contribution to Kevin's cerebral palsy. It follows that the review decision was incorrect and it is quashed. The respondent Corporation is directed to provide Kevin with cover for his cerebral palsy and other related injuries at birth.

Costs

[139] Kevin is entitled to costs. If counsel are unable to agree, memoranda may be filed.

Judge PA Cunningham
District Court Judge

Solicitors: John Miller Law, Wellington, for the appellant
Buddle Findlay, Wellington, for the respondent