

**BEFORE THE ACCIDENT COMPENSATION APPEAL AUTHORITY
AT WELLINGTON**

[2014] NZACA 5

ACA 100/96

IN THE MATTER of the Accident Compensation Act
1982

AND

IN THE MATTER of an appeal pursuant to s.107 of
the Act

BETWEEN

TE RANGIHERIA POTAE
Appellant

AND

**ACCIDENT COMPENSATION
CORPORATION**
Respondent

HEARING: Evidence taken and appeal part heard at Auckland on 27 November 2013

AUTHORITY:

Robyn Bedford

COUNSEL:

Mr Hesketh, amicus curiae; Mr Barnett, counsel for respondent

DECISION AND INTERIM DIRECTIONS

[1] This appeal concerns an application for cover for personal injury by accident through medical misadventure alleged as occurring to the appellant (“Hiria”), an infant born on 31 December 1988, through an adverse reaction to vaccinations and initially attributed to her third Hepatitis B vaccine on 29 March 1989 at age 3 months and her third DPT vaccine received a week later on 5 April 1989. The claim was declined by ACC on 1 July 1991, on the grounds that ACC, having considered the medical facts on file, decided that there was no correlation between the vaccinations

given as medical treatment and the development of encephalitis¹. Mr and Mrs Potae applied to review ACC's decision. Ms Perry-Claridge dismissed the application in her review decision dated 25 June 1996, although by this time the claim had evolved to one involving only the pertussis component of the DPT vaccine as the alleged cause of Hiria's diagnosed West syndrome. A notice of appeal was filed in July 1996.

[2] For various reasons the appeal languished, despite the Authority's best attempts to have it heard. In 2008, Mr Cartwright directed ACC to obtain an opinion from Dr Stanley of the Middlemore Hospital, to try to resolve the conflict between the experts who had prepared reports for the review concerning the alleged causal connection between pertussis vaccine and Hiria's West syndrome. Dr Stanley's opinion was ultimately obtained in November 2009. Dr Stanley relied on modern research and did not support the causal connection, but as with the earlier experts, he too was unable to identify an alternative cause. The appeal came to a halt once more until was called before me for a directions conference in late 2012. Mrs Potae's then lawyer, Mr Miller, was given leave to withdraw and in 2013, the Authority appointed Mr Hesketh as *amicus curiae* to represent Hiria's interests and assist the Authority.

[3] Dr Stanley's evidence was heard on 27 November 2013. Before his evidence was taken, I asked counsel if they had considered the possibility that the appeal could concern accident *simpliciter*, as in *Mitchell v ACC*,² rather than medical misadventure as had been asserted in the claim for cover, as *Mitchell* had been relied upon in the alternative by Hiria's lawyer at the review, but it was not considered by Ms Perry-Claridge in her decision. There was general consensus that it was not possible to convert the claim from medical misadventure to one of accident *simpliciter* in the absence of a related claim and decision by ACC, but the questions put to Dr Stanley by counsel and by the Authority took into account a possible referral back to ACC for the claim to be considered under s 2 (a)(i) of the 1982 Act. At the conclusion of Dr Stanley's evidence, directions were made by consent that counsel

¹ It is agreed by all medical opinion that Hiria did not suffer encephalitis, but an encephalopathy as a result of which she developed West syndrome. "*Encephalitis*" is an inflammation of the brain usually caused by a viral infection; "encephalopathy" is the term covering certain conditions in which there are signs of cerebral irritation without any localised lesion to account for them; Black's Medical Dictionary, 42nd Edition (Marcovitch 2010).

² (unreported, AP NO. 193/89, HC Wellington Registry, 18 December 1990, per Grieg J; upheld by the Court of appeal in *ACC v Mitchell* [1992] 2 NZLR 436.

would confer when the transcript of evidence was available to try to agree as to the best way forward.

[4] On 23 January 2014, I made the following directions:

[1] The appeal was set down on 27 November 2013 for hearing the expert evidence obtained from Dr Stanley at the direction of the Authority (Mr Cartwright in 2009 and Ms Bedford in 2013). Dr Stanley's evidence was led by Mr Barnett and he was cross-examined by Mr Hesketh, and he also answered questions put to him by the Authority.

[2] By the conclusion of Dr Stanley's evidence it became apparent that as an alternative to the medical misadventure claim under s 2(1)(a)(ii) before the Authority, it may be possible that Ms Potae suffered a personal injury by accident such as suffered by the appellant in *Mitchell v ACC* which would entitle her to cover under s 2(1)(a)(i) of the Accident Compensation Act 1982.

[3] As ACC had only considered the claim under medical misadventure, this being the claim before it, by consent I directed counsel to consider Ms Potae's situation after receiving a transcript of the evidence, and revert to the Authority to advise whether a claim under *Mitchell* was to be pursued and if so, whether ACC would agree to consider the claim as brought under the 1982 Act.

[4] At the conference Mr Hesketh reported that he had met with Ms Potae and discussed the medical evidence with her and that she had decided that if ACC would consider Ms Potae's claim under the 1982 Act personal injury by accident criteria, then she would be happy to withdraw the appeal against ACC's decision to decline cover for medical misadventure.

[5] Mr Barnett confirmed that ACC was willing to investigate a personal injury by accident claim and the application of the *Mitchell* decision to Ms Potae's circumstances and it was agreed that rather than Ms Potae withdrawing the appeal, the Authority would dismiss the appeal subject to directions to ACC concerning the consideration and determination of Ms Potae's claim for personal accident by injury under s 2(1)(a)(i).

[6] Mr Hesketh's ability to represent Ms Potae after his role as amicus is functus officio was discussed and I cannot see any reason why he should not be able to do so. Indeed, I think it would be of great benefit to Ms Potae and would assist with implementing the Authority's directions and ACC's investigations if he were acting for Ms Potae in a private capacity.

[7] Subject to Mr Hesketh providing written confirmation from Ms Potae that she consents to the appeal being dismissed and ACC's medical misadventure decision being confirmed rather than withdrawing the appeal as has been discussed with Mr Hesketh, by consent, counsel are to file a joint memorandum within 14 days which is aimed at assisting the Authority to frame appropriate directions to ACC for the consideration of the claim for cover under s 2(1)(a)(i) of the 1982 Act in light of the *Mitchell* decision.

[8] If all matters that I consider relevant have been included in the memorandum, then I will issue my decision and remit the claim back to ACC for investigation and determination accordingly. If not, then I will issue a memorandum concerning matters still to be addressed and the reasons for this, and a further telephone conference will be convened before my directions are made final.

[5] On 13 February 2014, Mrs Potae agreed in a letter addressed to the Authority to the appeal based on medical misadventure being dismissed on the condition that

Hiria's claim for cover will be determined under s 2(a)(ii) of the 1982 Act as personal injury by accident (other than by medical misadventure) and that ACC will issue a fresh reviewable decision on the claim.

[6] On 17 February 2014, counsel filed a joint memorandum and agreed as follows:

3. On the understanding that Ms Potae is to provide written confirmation that she consents to this appeal, founded as it is on a claim for medical misadventure, being dismissed, the respondent would not oppose the Authority dismissing the appeal with a direction that the matter be referred back for separate consideration as a claim for cover for "*personal injury by accident*" (other than medical misadventure) In light of the Court of Appeal decision in *ACC v Mitchell* [1992] 2 NZLR 436, and that a fresh reviewable decision be issued by the Corporation on such a claim.
4. In the event of the Authority dismissing the appeal and making such direction, counsel do not ask for or require the Authority to elaborate on or add to the terms as detailed in paragraph 1 above.
5. If the Authority has in mind making a referral back on terms additional to that proposed here, then counsel would first require to be heard.

[7] The appeal decision is therefore intended to form the basis for the investigation of Hiria's claim under s 2(a)(i) and records the relevant background facts and medical information that have a bearing on the consideration of the claim in light of *Mitchell*, and the directions that will need to be made for the referral back to ACC under s 109(8). I have not covered the pertussis debate as conducted in the expert reports, as it is accepted on the basis of Dr Stanley's evidence that the medical misadventure claim must fail and will be dismissed.

Background

[8] Dr Jamison, a Paediatric Neurologist at the Princess Mary Hospital where Hiria was treated from May 1989, was asked by ACC to prepare a report for the review. The relevant parts of the report dated 30 December 1991 are repeated below:

As indicated, I had perused the file for Dr J Newman who had been the paediatrician responsible for Hiria's management in Princess Mary

Hospital in May 1989, and subsequently, as indicated in Dr Newman's letter of 23/4/91, saw little correlation in time or in consequence between immunization and the conditions described. After referral by Hiria's general practitioner, Dr Julia Peters, as she was concerned about Hiria's poor fit control, I saw her in Paediatric Neurology Clinic at Princess Mary Hospital on the 21st October 1991 and on the 12th December 1991.

From the contemporary Princess Mary case record and the family diary 29th March 1989 to 19th May 1989 (some days not decipherable on the photocopy I have -copy enclosed) the salient details seem to be:

- 1 The pregnancy, delivery, antenatal and neonatal period, apart from mild hypothermia initially, is regarded as normal.*
- 2 No problems are alleged in regard to immunization until after the second injection of hepatitis B vaccine and first injection of triple (diphtheria, pertussis, tetanus -DPT) vaccine at six and a half weeks of age (15th February 1989) after which Hiria was grizzly.*
- 3 The family diary entry 29/3/89 (no previous entries available to me) states: "doctor's for injection (hepatitis B), decided to split (them because of last time. Didn't work, Hiria's grizzly again".*
- 4 According to the diary note 30th March 1989 an "ugly rash on Hiria tonite ...mostly in neck and armpit" and the following day "PHN reckons its a rash caused by something in breast milk. Go to chemist. Using Alpha Keri and Polaramine today". By April 4, "quiet tonight, Alpha Keri is working, rash not so red, starting to dry up". April 5, "doctor's injections (DPT) talked about rash, keep (an) eye on it". The Princess Mary Hospital admission on the 1515/89 records "over the last month has had widespread eczema".*
- 5 The second injection of triple (DPT) and first dose of oral polio vaccine was given 5/4189.*

On 6/4/89 the family diary states: *...Hiria's playing up tonite" and on April 7, "small sores on hands and head. Different to rash, looks like pimples. She rubs her eyes and nose a lot causing redness. Unhappy girl today". On April 8, "sores are spreading across her hands. Creme looks like it is working but only slowly. Rash looks very uncomfortable. Baby only a little grizzly tonite" . By April 12, "that's two quiet days in a row, unusual for Hiria, usually she is either laughing or crying (makes a nice change to her whinging ..)". April 13, "back to normal, a lot of noise from madam".*

6. *The Princess Mary notes at admission on 15/5/89 indicate that "about a month ago she had a cold and widespread rash over her body. Has had hydrocortisone cream for the rash -now only the neck and ears. Had been on SudomyI and Amozil for her cold". This reference to a cold correlates with April 16 diary entry. "Looks like someone's getting a chill. Bootees don't seem to be doing the trick, mittens might not be any good for sores?? anyway they look funny". April 17, Freda will take baby to doc's tomorrow. Cold has gotten worse". April 18, "doctor's visit: baby is chesty, very runny nose. Has spewed up some milk earlier. Got some medication".*

7. *Thirty-four days after second DPT immunization the diary states on May 9, "Freda reckons baby daydreams a lot (father must be a spaceman)" . In the discharge letter relating to the admission to Princess Mary Hospital on 15/5/89 it is stated: "This babe was flown across from Waiheke Island with a one week history of intermittent activity indicative of fitting". it is also stated: "The child had a flu-like illness approximately one week before admission but no other problems" .*

At the time of admission to Princess Mary Hospital on 15/5/89 she was regarded as having:

- a. Fits in previous 3 days.*
- b. Infected eczema.*

c. Delayed motor development with hypotonia with no obvious cause.

On 16/5/89 an electroencephalogram (BEG) was reported as normal and on 19/5/89 computerised tomography (CT) of Hiria's brain (without contrast media given) showed no specific abnormality, but when repeated 25/8/89 Dr G Dodd, Radiologist, reported "no significant alteration since examination in May 1989. There is suspicion of a little patch decreased attenuation superficially in the posterior part of the left cerebral hemisphere due to some previous minor damage ? ischaemia. No other abnormalities detected apart from slight dilatation of the lateral ventricles and Sylvian fissure".

The fitting responded initially to Epilim medication.

- 8. Her seizures increased 10-30 times a day, and her developmental progress became significantly delayed. Dr S. Ameratunga on 21/8/89 noted that Hiria had ceased developmental progress in that previously Hiria "was almost rolling from supine to prone and now lies supine, fairly inactive most of the time". She had "ceased to reach for toys and plays with hand. Now little interest in beads/feet/toys". By 21/8/89 West's syndrome (infantile spasm syndrome) with developmental delay was apparent. An EEG on 22/8/89 was reported as being severely abnormal (hypsarrhythmic) with bursts of attenuation, probably electrodecremental seizures. By 4/9/89 the EEG continues to show a hypsarrhythmic pattern. After a course of steroids, by 21/8/89 the EEG had improved.*

A number of investigations attempting to demonstrate the aetiology of her encephalopathy were found to be negative and included, over the two admissions in May and August, toxicology screen, blood lead and copper, serum sodium, potassium, urea, calcium, magnesium, amino acids, urine organic acids, blood ammonia, blood liver function tests, syphilis serology, and white blood cell cerebral enzymes.

Discharge diagnoses on 5/9/89 at Princess Mary Hospital are listed as:

- 1. Seizure disorder -infantile spasms.*
- 2. Developmental delay.*
- 3. Conjunctivitis.*
- 4. Upper respiratory tract infection.*

9. It is difficult to date the onset of her developmental delay as in the Princess Mary Hospital notes at the time of admission on 15/5/89 it is recorded that there was "some head lag" and that when examined on 16/5/89 she was noted to be hypotonic with perhaps a spastic catch in the legs. The parents indicated that Hiria's floppiness was not new. The Occupational Therapist on 17/5/89 noted abnormal posturing with extension of the neck especially in supine, flexor posturing of the upper limbs, predominant extensor tone in the lower limbs. The paediatric medical registrar on 18/5/89 considered there was evidence of motor delay present. Dr J. Newman described Hiria on 30/11/89 as significantly delayed with skills scattered between a 3-5 months developmental level at a chronological age of 11 months.

10. Currently Hiria remains severely developmentally delayed with poorly controlled epilepsy.

I would summarize the above history as indicating a child with apparently normal developmental progress until about May 1989 (although the findings at the time of admission in May could suggest that there was some prior developmental abnormality not noted previously), when 1-2 weeks after an upper respiratory tract infection treated with antibiotic for a week, Hiria manifested fits. In mid-May 1989 abnormalities of posture and tone were noted. Subsequently she went on to demonstrate West's syndrome of infantile spasms with a hypsarrhythmic EEG and associated developmental regression.

It is recognised with the syndrome of infantile spasms, which is an encephalopathy occurring in infancy, that about two-thirds will continue to have epileptic fits and that only 13 % in some series have the mentality to be able to attend normal school. The onset of this syndrome is usually between the ages of 3 and 9 months with a peak at 3-5 months of age. Developmental cessation and deterioration occurs at the time of the onset of this form of epilepsy. The term 'hypsarrhythmia' is a descriptive one applied to the EEG pattern and this pattern has a poor prognosis for future intellectual normality even if there are no clinical fits seen. Drs. Friedman and Pampiglione, Children's Hospital, Great Ormond Street in London reviewed 105 children who had had this EEG pattern and found that the mortality in this group was in the order of 1 in 4 and the instance of mental subnormality in survivors was 77 % . Only 18 children attained fairly normal standards of mental development and could attend ordinary schools. It seemed that whatever the clinical picture at the time, the presence of hypsarrhythmia in the EEG of an infant with physical signs, had grave prognostic implications. (Prognostic implications of electroencephalographic findings of hypsarrhythmia in first year of life. E. Friedman and G. Pampiglione. British Medical Journal (1971) 4: 323-325).

In regard to aetiology, it seems that infantile spasms represent the way the brain can behave in response to a number of factors and they have been associated with cerebral malformations which are inherited e.g. tuberous sclerosis, while others are sporadic, e.g. neuronal heterotopias; inborn errors of metabolism; prenatal intrauterine infections, e.g. cytomegalovirus infection; perinatal insult to the brain; and postnatal insults, e.g. viral encephalitis, trauma etc. In some, as in Hiria, no cause is able to be established. It should be noted that neuronal heterotopias when present may not be able to be disclosed by CT brain imaging.

[9] The only other contemporaneous record, apart from the diary notes that Dr Jamison referred to is Hiria's Plunket book. Hiria was seen at home by the Plunket public health nurse weekly for her first 6 weeks, then fortnightly until age 3 months and her records described Hiria as a large healthy and contented baby who progressed with large weight increases to being “an active responsive happy 3

months old infant” who passed all her milestones and was holding things and close to rolling over at age 14 weeks, the last time she saw Hiria before the DPT injection. Ms Evans recorded that Hiria had good muscle tone, and the only negative thing she noted was that Hiria then had an allergic rash. She next saw Hiria on 15 May 1989, after Hiria had been vaccinated with the DPT, and observed the rash still present but no sign of the twitching that caused her hospitalisation. The next time Ms Evans saw Hiria was on 19 July 1989, and she was floppy and totally unresponsive, with low muscle tone.

[10] Dr Reid, General Practitioner and Member of the Communicable Disease Control Advisory Committee, was instructed by Ms Clarridge Perry to provide a further report because of the differing opinions expressed by Dr Morris and Dr Jamison as to the causal connection between the pertussis vaccine and the West’s syndrome. In his report dated 13 June 1995, Dr Reid agreed with Dr Jamison that the pertussis vaccine was unlikely to be the cause. Regarding the onset of Hiria’s West syndrome, Dr Reid said:

Between 13/4/89 and 9/5/89 Hiria suffered symptoms of respiratory tract infection and eczema but there is no comment in the parent’s diary which indicates the onset of ‘encephalopathy’ until the entry of 5/9/89

...The contention as I understand it, of the Potaes, is that the pertussis component of the second dose of DPT vaccine administered to Hiria on 5/4/89 caused encephalopathy which resulted in West’s syndrome of infantile spasms with a hypsarrhythmic EEG and associated development regression. The date of the onset was 9/5/89 when Hiria was noted to ‘day dream a lot’, 34 days after the administration of the relevant dose of DPT vaccine. It could be argued that the first sign of Hiria’s illness was on 11/4/89. I consider this unlikely as there was a 29 day gap until further symptoms suggestive of the onset of West’s syndrome were noted.

[11] Neither Dr Jamison nor Dr Reid were able to identify a cause for the encephalopathy in May that led to the later diagnosis of West syndrome, and this was the one area in which they both agreed with Dr Morris. Dr Jamison’s comment that Hiria suffered mild hypothermia as a neonate was not considered in terms of its

possible contribution or cause, and the focus was the causal nexus with the pertussis vaccine.

The evidence on appeal

[12] Dr Stanley's report dated 1 November 2009 is the only other specialist evidence considered for the appeal. As with the other reports, I have ignored the aspects dealing with pertussis. Dr Stanley described Hiria's situation in 2008 as follows:

"I have had the opportunity of reviewing her extensive case notes. They confirm that she has had multiple admissions to the ward with complications of her cerebral palsy. Although the type of cerebral palsy appears to have been variably defined (including spastic quadriplegia), it is clear now that her signs are those primarily of atheoid cerebral palsy. She also has severe developmental delay, epilepsy and scoliosis. She is wheelchair bound. Although I mention atheoid cerebral palsy, I suspect that in fact she has a mixed atheoid spastic picture. This is not uncommon."

[13] Dr Stanley subsequently clarified his diagnosis for the Authority in his letter of 13 July 2013, and said:

"I believe on the balance of probabilities she has idiopathic³ West syndrome but as mentioned in my report from 2009 there are a number of conditions that lead to this which had not, at that time at least, been excluded." (Dr Stanley confirmed this diagnosis again in his evidence)

[14] The only difference of any significance between Dr Stanley's observations in his report concerning Hiria's history and the earlier medical evidence, is that while Dr Jamison summarised Hiria's history *"as indicating a child with apparently normal developmental progress until about May 1989"*, Dr Stanley felt there was no definite evidence of this but he did say that prior to Hiria's seizures, *"no definite developmental regression had occurred and she had no prior history to suggest she had suffered from an encephalic or encephalopathic process (brain inflammation or acute brain disorder as defined very well by Dr Jamison)."*

³ "Idiopathic" means of an unknown cause.

[15] However, in his evidence, Dr Stanley explained that he had based his opinion that there was some doubt as to whether Hiria was actually developmentally normal prior to the first worries her father recognised on the lack of clinical information concerning Hiria that he was given and said:

"I have to say that the Plunket record that is now available to me makes me much more happy that she was developmentally normal.

... If the developmental milestones were abnormal, that would be highly relevant of she was already developmentally abnormal before she presented, that would suggest already that she's showing symptoms of an abnormal brain."

[16] Earlier in his evidence, when asked what was suggested, if anything, when you have a child who is developing normally and reaching normal milestones with a normal weight gain/growth, a normally thriving child who then seems to have a sudden stop and a diagnosis of severe developmental delay at a point in time merely weeks after a normal observation, Dr Stanley said:

"It suggests that an event occurred around the time when she presented with her spasms that was associated with significant injury."

[17] Dr Stanley said that this didn't necessarily discount a genetic predisposition, but it was impossible to say whether in Hiria's case there was an outside event, or a genetic cause. Dr Stanley discounted a febrile seizure and Dravets, because Hiria did not fit the scenario for such a child, and he was clear that there was no evidence of an acute neurological illness as the cause and no evidence of encephalitis. Nor could he say whether the infantile spasms were a cause, or an effect of the beginning of Hiria's physical and intellectual disability, but he did say that the atetoid cerebral palsy that Hiria is described as having in her post 1989 treatment notes was a chronic condition Hiria has developed as an outcome of her earlier neurological injury. Dr Stanley also explained that the terms "*atetosis*" and "*spasticity*" are not medical diagnoses, they are descriptions of clinical signs and simply signal that a child has a damaged brain.

[18] Dr Stanley said, in the context of discussing cases of children with epilepsy who present as normal up to the time of their first epileptic seizures, that out of 10,000 or 15,000 births in the Wellington Hospital area each year, the rate of children with unexplained serious neurological presentations would probably be “*one or two of these children a year, maybe three that present in this sort of way*”. He said that West syndrome is relatively rare and in his opinion, the number of cases of West’s syndrome in whom no cause is found is somewhere between 25% and 30% of presenting cases.

[19] Regarding the investigations that could now be performed, Dr Stanley confirmed that Hiria had had EEG and CT scans, but no MRI scan. Dr Stanley said that if an MRI scan could be carried out, this could demonstrate if the cause of the damage was some form of secondary brain injury, or if it was a primary developmental abnormality that Hiria had from the start. Hiria could then have a blood test to see if she had a recognised genetic or metabolic disease that explains her progress. However, in his report, Dr Stanley identified various problems associated with subjecting Hiria to an MRI scan and said that at this stage it was very likely that she would have an abnormal brain scan as a result of her subsequent brain deterioration and that the scan would probably show signs of brain shrinkage. He thought that Hiria *may* still show signs on an MRI scan of an underlying brain malformation that was present prior to the onset of her seizures, but that she was likely to require a general anaesthetic and he could “*understand her caregivers’ reticence in considering such an investigation.*”

[20] Dr Stanley based his opinion in part on research published by Olivier Dulac and Ingrid Tuxhorn in 2005⁴. Regarding the incidence of West syndrome, they said at page 53 that according to studies they cited, this ranges from 2.9 to 4.5 per 100,000 live births. With respect to cause, Dulac and Tuxhorn do not state the incidence of failure to find a cause, but state at page 54 that pre-existing brain damage is seen in 90 per cent of cases and at pages 59 and 60 they describe other causes (which may be coincident) such as vascular malformations and inborn errors of metabolism as rare, and less rare are tumours, a family history of epilepsy or febrile convulsions. This suggests that the incidence of unknown cause is in the vicinity of 1 – 10% at the

⁴ Epileptic Syndromes in Infancy and Adolescence (4th edn), Chapter 4: Infantile spasms and West syndrome.

very highest, and probably less than that, given the overlapping of causes identified in the research.

[21] With respect to radiological examinations that are the major source of investigation of West syndrome, Dulac and Tuxhorn differ with Dr Stanley that delayed MRI scans would be of benefit, and they said on this point at p 59:

“...Pre-, peri- or postnatal damage may be disclosed by history, and clinical examinations may demonstrate signs of a neurocutaneous syndrome, but neurological examination at the time of diagnosis is seldom contributive. Neuroradiology is therefore the major source of investigation, but it must be performed at the start of steroid treatment which produces shrinkage of the brain that is difficult to distinguish from atrophy which is present in half the cases and from the normal variations of pericerebral space size in infancy. Magnetic resonance imaging (MRI) may overlook abnormalities in the grey matter in the second semester of life after 18 months of age.”

Mitchell v ACC

[22] *Mitchell* concerned a claim for personal injury by accident, where the accident could not be identified. The appellant was born on 9 February 1987 and he suffered what was described as an apnoeic episode on 9 May 1987. Apnoea is defined as “*suspension of breathing; cessation of respiration*”. On 30 May 1987, the baby’s apnoea alarm sounded and his mother took him to hospital. The attack was prolonged and was followed by severe and persistent fitting which lead to metabolic and circulatory changes, which in turn led to large areas of his brain becoming severely damaged and eventually dying. He was declared brain dead, but survived and would be profoundly handicapped for the future. The paediatrician’s opinion was that there was no clear reason for last apnoeic attack although it could have been caused by virus infection, metabolic disturbance or epileptic trigger resulting from the previous attack and there was no clear reason for the earlier attack.

[23] The appeal turned on the definition of personal injury by accident in terms of whether an external action or cause was a necessary part. Section 2 (1)(a)(i) provides that “*Personal injury by accident*” includes the physical and mental

consequences of any such injury or accident; paragraph (b)(ii) excludes damage to the body or mind caused exclusively by a disease, infection or the aging process.

[24] Greig J said at page 17:

“In my view authority supports the conclusion that it is not necessary to show, for personal injury by accident, any outward, external action or cause, whether occurring by some outward means or by some action of the person concerned. This is not to say that there will never be a need to show some incident or circumstance identified as the cause of the damage or injury. I do not suggest the condition resulting from a gradual process over a period now becomes personal injury by accident.⁵ That question is not in issue in this case and is not for decision by me.”

[25] The process Grieg J approved is described at page 18:

“Adopting the analysis proposed by Lord Wilberforce, but without the reference to the connection of that with any element of employment, one decides what happens first. What that was was a cessation of breathing. That was involuntary. Although there may be a possibility of disease causing it is not the sole cause and it certainly is now shown that it was the cause or even a cause. This must be treated as an untoward event, an unexpected happening. The result of that is the tragic state in which that child now lives. That clearly has followed from the apnoea and the physiological effects and injuries which followed. If one looks at the matter without subtlety I think the only conclusion that can be made is that this was injury by accident, an unexpected untoward mishap. The fact that it came from a stoppage in an essential human activity does not alter the real effect, the real meaning of the events and occurrence.”

Interim directions

[26] I do not intend to pre-empt ACC’s consideration of the claim or limit the evidence it should consider, but I direct that ACC conscientiously applies the approach adopted by Greig J in *Mitchell* and puts aside completely the definitions of

⁵ The authorities Greig J considered involved gradual process work injuries.

accident that have developed under the later legislation in light of the background facts and the medical evidence that I have discussed above. As part of its investigation, ACC should also consider the role, if any, that may have been played by the hypothermia episode noted by Dr Jamison.

Decision

[27] The appeal is dismissed.

[28] The question of whether the appellant's West syndrome amounts to personal injury by accident under s 2(a)(i) of the Accident Compensation Act 1982 is remitted back to the Corporation under s 108(9) of the Act to issue a fresh decision with review rights.

[29] Counsel have 14 days in which to comment on the interim directions before they become final.

[30] There is no order as to costs.

DATED at Wellington this 7th day of March 2014

.....
R Bedford