

SOUTH



AUSTRALIA

FINDING OF INQUEST

An Inquest taken on behalf of our Sovereign Lady the Queen at Adelaide in the State of South Australia, on the 30th and 31st days of March 2005 and the 2nd day of June 2005, before Wayne Cromwell Chivell, a Coroner for the said State, concerning the death of Matthew Hutchinson.

I, the said Coroner, find that Matthew Hutchinson aged 1 day, died at the Flinders Medical Centre, Flinders Drive, Bedford Park, South Australia on the 7th day of January 2002 as a result of cardiogenic shock and neonatal encephalopathy. I find that the circumstances of his death were as follows:

1. Introduction

- 1.1. Matthew Hutchinson was born on 6 January 2002 at the Flinders Medical Centre at 11:32am. He was extremely unwell at birth and required resuscitation. He began having seizures. He was transferred to the Neonatal Intensive Care Unit under the care of Associate Professor Peter Marshall, the Director of Neonatal Medicine.
- 1.2. Matthew's condition did not respond to treatment, and he deteriorated overnight. He died at 7:30am on 7 January 2002.
- 1.3. Matthew's death was not initially reported pursuant to Section 31(1) of the Coroners Act, 1975, which states:

'A person knowing of, or becoming acquainted with, the finding of the body of a dead person, or the death of a person apparently by violent or unusual cause, must immediately notify a coroner, or a police officer, of that finding or death.'

- 1.4. Instead, a report signed by Associate Professor Marshall was forwarded to the Registrar of Births, Deaths and Marriages citing the cause of death as cardiogenic shock and neonatal encephalopathy. A death certificate was issued by the Registrar on 18 November 2003 registering that as the cause of death.
- 1.5. On 14 February 2002 Dr Ginette Kremmidiotis, the General Practitioner who managed Mrs Hutchinson's pregnancy and post-natal care, wrote to me about what she described as the 'extremely poor management of this young woman's confinement at Flinders Medical Centre'. Dr Kremmidiotis was particularly critical of the fact that abnormalities (decelerations in pulse rate) were noted for several hours prior to Matthew's delivery.
- 1.6. Dr Kremmidiotis indicated that she had already spoken to at least one of the clinicians at Flinders Medical Centre about the case.
- 1.7. On 25 February 2002, Associate Professor Robert Bryce, the Clinical Director of Obstetrics and Gynaecology, wrote to me in the following terms:

'Dear Mr Chivell

Re: Notification of death – Matthew Hutchinson – Flinders Medical Centre

I advise that Matthew Hutchinson, d.o.b. 6 January 2002 at 1132 hours, died on 7 January 2002 at 0745 hours.

A post-mortem examination was not conducted as the infant's parents declined an autopsy. A review of the case by me concluded that an abnormal fetal heart rate was evident before birth.

Please advise if you require the hospital to provide anything further.'

- 1.8. By that time, of course, it was far too late to conduct a post-mortem examination of Matthew's body. I understand that Matthew's parents say that they 'declined' an autopsy on Associate Professor Marshall's advice that it would achieve nothing. If the case had been reported immediately, as the law requires, that discretion would have been mine and, on the evidence available, would almost certainly have been exercised in favour of an examination.

2. Obstetric issues

- 2.1. In view of the issues raised by Dr Kremmidiotis, Professor Roger Pepperell, Professor of Obstetrics and Gynaecology at the University of Melbourne, was asked to provide an opinion concerning the obstetric aspects of Matthew's birth.

- 2.2. Professor Pepperell is an eminent obstetrician and gynaecologist who has provided such opinions for these purposes on many previous occasions. His opinions were unchallenged at the inquest. I have no hesitation in accepting them.
- 2.3. Professor Pepperell provided a very helpful outline of the chronology of Mrs Hutchinson's labour and delivery. He recorded that she was 23 years old, and an early ultrasound examination established her estimated due date as 29 December 2001.
- 2.4. As to the ante-natal care provided, Professor Pepperell commented:

'I can find no evidence of a deficiency in the care which was provided to her during the antenatal period, and the decision to let her go a few days overdue was not inappropriate, as one would usually prefer not to induce a patient with grade I placenta praevia.'

(Exhibit C4a, p2)

Placenta praevia is a condition where the placenta has implanted adjacent to or over the cervix, which may cause problems during delivery in the form of placental haemorrhage.

- 2.5. Professor Pepperell outlined Mrs Hutchinson's progress of labour which was as follows:
- Contractions commenced at about 6:30pm on 5 January 2001;
 - Mrs Hutchinson was admitted to hospital at 10:45pm, by which time her contractions were at the rate of 4 in 10 minutes;
 - A pelvic examination at 11:45pm showed that the cervix was 3cm dilated, with the head in the occipito-transverse position, still 2cm above the ischial spines. The membranes ruptured during the examination;
 - The next pelvic examination at 3:30am on 6 January 2002 disclosed that the cervix was still only 4cm dilated. A Syntocinon drip, to encourage labour, was commenced;
 - At 4:30am the cervix was still only 4cm dilated, so the drip was continued. A cardio-tocograph (CTG), to monitor the baby's heart rate, was not commenced at this time. The heart rate was assessed by 'non-electronic' means;
 - At 6:30am pain relief was given, and contractions were noted at 4-5 every 10 minutes;

- A pelvic examination at 8:15am showed the cervix was 5cm dilated. The Syntocinon was ceased at 8:45am 'presumably because an abnormality had been detected in the fetal heart rate as determined by non-CTG monitoring'. A CTG was attached at 8:49am;
- An epidural anaesthetic was commenced at 9:15am. Professor Pepperell commented:

'At 0940 hours pelvic examination showed the cervix was getting close to fully dilated, with an anterior lip only, but the head was in the direct occipito posterior position but now 1cm below the ischial spines. Because the CTG was clearly not normal a fetal scalp pH estimation was done at 0955 hours. This showed the pH was 7.26, this being within the normal range.'

(Exhibit C4a, p2)

- Further decelerations in the fetal heart rate were noted while Mrs Hutchinson was pushing between 10:30am and 11:15am;
- At 11:32am, delivery was expedited using a Ventouse (an instrument the use of which involves placing a suction cup on the baby's head, allowing the operator to apply force and assist delivery - a technique which has largely replaced forceps);
- As to Matthew's condition at birth, Professor Pepperell commented:

'The baby was in the deflexed occipito posterior position, but rotated as it reached the pelvic floor. The baby weighted 2,990 grams, but was in poor condition at birth with Apgar scores of 2 at 1 minute, 5 at 5 minutes, and 7 at 10 minutes. Cord blood samples were taken for pH assessment. The venous pH was found to be 7.49, and the arterial pH 7.07. The base excess was within the normal range on the arterial blood sample, indicating that severe acidosis was not present. An arterial pH of 7.07, whilst not entirely normal, is rarely associated with any long-term problems in the fetus or in the neonate.'

(Exhibit C4a, p3)

2.6. CTG monitoring

Professor Pepperell gave careful attention to the information disclosed by the CTG monitoring, and in particular whether there were any grounds for earlier intervention, perhaps in the form of a caesarean section. After analysing the readouts from the instrument and other information available including blood chemistry, he commented:

'Overall, therefore, although the CTG is not completely normal, at any time, the fact the pH estimation was done at 0955 hours and was normal, the fact that there was no

significant increased problem between then and the time she was pushing, and the fact the CTG was even better when the pushing was stopped immediately prior to the attempt at the vacuum extraction, would indicate to me that the care provided was appropriate and certainly not deficient. It is the sort of care which would have been given by our own Registrars at the Royal Women's Hospital in Melbourne if they had been handling a case like this.

The fact that the arterial cord pH estimation was 7.07, slightly acidotic, but not profoundly so, would also have reassured the staff that they did interfere at the appropriate time when delivery was expedited using the vacuum extractor.

The question does need to be raised as to whether the fetal scalp pH estimation should have been repeated after 0955 hours when the labour was allowed to proceed. The rules at my own Hospital are that the pH should be repeated at about 1 hour if the pattern is continuing unchanged, or delivery should be expedited at that time. Where a deterioration in the fetal heart rate and rhythm, as detected by the CTG, occurs prior to 1 hour, the pH should be repeated at that time unless delivery can be expedited such as with the vacuum extractor, or forceps.

I therefore do not believe the staff managed Mrs Hutchinson along anything but standard lines, and am unable to explain exactly why this baby was in such poor condition at birth, and fitted during the early neonatal period, and subsequently succumbed. Syntocinon over-response could not have caused this because the Syntocinon had been ceased many hours previously, prior to the pH estimation. Sometimes unexplained deaths do occur, where no apparent cause for the death or poor infant condition at birth is able to be defined. '

(Exhibit C4a, p4)

- 2.7. I accept Professor Pepperell's opinions on these issues, and find that there are no grounds for criticism of Matthew's peri-natal care.

3. Neonatal issues

- 3.1. At the suggestion of Professor Pepperell, an opinion was sought from an experienced Neonatologist, Dr Andrew McPhee, who is the Deputy Head of Neonatal Medicine at the Adelaide Women's and Children's Hospital.

- 3.2. Dr McPhee summarised the relevant post-natal events in his report as follows:

- 'There was spontaneous onset of labour on 5.1.2002, with Mrs Hutchinson being admitted to the labour and Delivery ward at FMC at 2232 hrs. Assessment soon after revealed the cervix to be 3 cms dilated, and contractions to be occurring every 5-10 minutes.
- There was slow progress through the early hours of 6.1.2002, and a Syntocinon infusion was started at around 0400 hrs. At around 0840 hrs, fetal decelerations during contractions were noted on intermittent auscultation of the fetal heart rate. This prompted the application of an external cardiotocograph (CTG), in order to

monitor more closely the fetal heart rate pattern; this CTG was reported to show 'decelerations \downarrow upper 80s to 100. Baseline altered," and this resulted in a review by the Obstetric Registrar (Dr Weatherill), and a decision to perform a scalp pH assessment.

- At the time of this scalp pH assessment, the cervix was found to be fully dilated, save for an anterior lip, and the position of the fetal head was noted to be occipito-posterior. The scalp blood sample was analysed at 0955 hrs, and revealed a pH of 7.26, and a Base Excess (BE) of - 3 mmol/L.
- Matthew was in poor condition at delivery, and required resuscitation with oropharyngeal suction and bag and mask ventilation. This resulted in a prompt improvement in the heart rate and colour (i.e. heart rate "> 100 @ 1 min", and "pink"), but initial gasping respiration was not noted until 6 minutes, and regular breathing not seen until 13-14 minutes, following endotracheal intubation and ongoing ventilatory support at 12 minutes. Overall, 1, 5 and 10 minute Apgar scores of 2, 5 and 7 respectively were given.
- Arterial and venous umbilical cord specimens were collected for blood gas analysis. The arterial specimen showed a pH of 7.07, pCO₂ = 88 mmHg, pO₂ = 105 mmHg, and BE = -9mmol/L. Analysis of the venous specimen revealed a pH of 7.49, pCO₂ = 28, pO₂ = 42, and a BE = 0 mmol/L.
- Matthew Hutchinson's birth weight was 2990 gms (3-10th percentile for 41-42 weeks gestation), length 51 cms (75th centile), and head circumference 33 cms (3-10th centile). There is no mention in the case notes of any obvious congenital abnormalities or dysmorphic features.
- Matthew was subsequently admitted to the Neonatal ICU at FMC, where respiratory support in the form of mechanical ventilation was continued. Over the ensuing 1-2 hours his inspired % oxygen was reduced to air, based on pulse oximetry. An intravenous line was sited, and 10% dextrose was given, first as a bolus, and then as an infusion at 45 ml/kg/d.
- Cyclic movements of the arms and right leg, and intermittent generalised rigidity were noted at about 1 hour of age; these were interpreted to be seizures and were treated with intravenous phenobarbitone 25 mg/kg, given at 1250 hrs, with a further dose of 15 mg/kg being given at 1430 hrs.
- Initial investigations included a full blood examination (FBE) and a chest X-ray. The FBE was unremarkable apart for some toxic changes in the neutrophils. The chest X-ray showed acceptable endotracheal tube position, and clear lung fields.
- Based on the reasonable assumption that Matthew's poor condition at birth, his need for resuscitation and ongoing ventilatory support, and his early seizures, were due to an antenatal hypoxic-ischemic insult, a decision was made to induce hypothermia, in the hope of providing some degree of neuroprotection.
- The induction of hypothermia was commenced at around 1400 hrs, and although the target temperature was 34°C. a rectal temperature of 32.1°C was observed at 1600 hrs; subsequently, despite some rewarming efforts, the rectal temperature fell further, perhaps to as low as 30°C, before rising again. With respect to this phase of care, the clinical record is not clear as to the method of monitoring core temperature, and in

fact the Vital Signs Chart ..., has a temperature column for 'core temp' and another for "Infant A.R". with the latter obviously referring to rectal temperatures. In addition it is not clear as to whether the true core temperature was being monitored continuously or intermittently.

- The induction of hypothermia resulted in poor peripheral perfusion ('colour bluish' and 'poor perfusion'), and this was presumably one of the reasons that the pulse oximeter failed to register from about 1800 hrs ('Unable to monitor SaO₂ for most of shift'). In addition, attempts to monitor blood pressure, using a non-invasive oscillometric technique, also failed - "NIBP not reading either". Indeed, as documented on the Vital Signs Chart, there is, no recording of blood pressure until the placement of an arterial line at around 2400 hrs (i.e. at 12-13 hours of age).
- The first assessment of acid-base status was not made until 1640 hrs (i.e. at around 5 hours of age) when a capillary specimen returned the following results - pH = 7.36, pCO₂ = 16, pa₂ = 52, and BE = -15.6. A subsequent capillary specimen at 1800 hrs revealed worsening metabolic acidosis, with pH = 7.10, pCO₂ = 28, pO₂ = 54, and BE = -19.4. In response to the latter, the ventilation was increased.
- A small amount (1.5 ml) of urine was passed at around 1500 hrs; this tested strongly positive for blood and protein. There is no record in the clinical notes of any further urine output, between this time and the time of Matthew's death.
- At around 2000 hours, an umbilical venous catheter was sited, blood drawn for cultures (subsequently returning negative results), and antibiotics were commenced (penicillin and gentamicin). A repeat acid-base assessment at this time revealed further deterioration, with pH = 6.86, pCO₂ = 45, pO₂ = 34. and BE = -23.6. In response to this, ventilation was increased, and an infusion of volume (30 mls of normal saline) was given. However this had only a modest effect on the acid-base status, as demonstrated by a repeat umbilical venous gas done at 2245 hrs; this specimen revealed a pH = 6.9, pCO₂ = 36, and BE = -23.4 (see results on Respiratory Care Chart). An infusion of bicarbonate (10 mls 8.4% bicarbonate in 20 mls normal saline) was started soon after.
- Attempts to place an umbilical arterial line at the time of placement of the umbilical venous catheter were unsuccessful. This led to a decision to perform an arterial cutdown at around 2330 hours, in order to secure arterial access for monitoring and sampling. Once sited, this arterial catheter revealed profound hypotension with a maximum mean arterial pressure of 28 mmHg, and with levels in the 15-20 mmHg range for much of the time.
- An arterial blood gas analysis performed at 0030 hrs, during the latter stages of the bicarbonate infusion, again showed a significant metabolic acidosis - pH = 7.20, pCO₂ = 18, pO₂ = 61, and BE = -20. In addition, blood was drawn at this time to measure routine biochemistry and to assess the coagulation status. These tests revealed a modest elevation of plasma creatinine (0.082 mmol/L - NR 0.021-0.078), and markedly abnormal coagulation results; in this clinical scenario, these abnormal coagulation results are consistent with a diagnosis of disseminated intravascular coagulation (DIC).

- The marked systemic hypotension and the persisting acidosis resulted in a further infusion of bicarbonate (same dose), the initiation of dopamine therapy at a dose of 12-15 microgms/kg/min, and even a brief period of external cardiac massage.
- Despite these efforts, systemic hypotension and metabolic acidosis persisted, leading to a diagnosis of cardiogenic shock. Following discussions with his parents, it was decided to withdraw support, and Matthew subsequently died at circa 0730 hrs (i.e. at 20 hours of age), whilst being cuddled by his family.
- Post mortem measurements revealed a weight of 3125 gms, length of 54 cms and a head circumference of 34 cms (note discrepancies with birth measurements).'

(Exhibit C7, pp2-5)

3.3. Dr McPhee said that there were a number of factors which were consistent with a diagnosis of an ante-natal hypoxic-ischaemic neural injury. These included:

- Matthew's poor condition at delivery;
- His need for intensive resuscitation, including an ongoing need for respiratory support;
- The early onset of a moderate to severe encephalopathy;
- The later onset of renal dysfunction, profound myocardial dysfunction with progressive metabolic acidosis (cardiogenic shock), and coagulopathy.

3.4. However, Dr McPhee said that there were also a number of atypical features which should be considered.

3.5. Firstly, there is a lack of biochemical evidence of a severe perinatal hypoxic-ischemic insult. The umbilical cord arterial blood analysis performed at birth revealed only a moderate metabolic acidosis, in that the pH was 7.07.

3.6. Associate Professor Marshall argued that this should be ignored because the sample had obviously been exposed to air (indicated by the high pCO₂ reading of 88mgHg).

3.7. Dr McPhee accepted this, but argued that the base excess ('BE'), which does not change on exposure to air, was only -9 mmol/L, which indicated only a modest lactic acidosis. He said:

'For example in the Consensus Statement regarding consideration of the evidence needed to support a causal inference between a perinatal hypoxic-ischaemic insult and later cerebral palsy, a pH of <7.00 or a BE of < -12 mmol/L is deemed necessary. Overall, the

available acid-base information does not provide strong objective evidence of a severe perinatal hypoxic-ischaemic insult.'

(Exhibit C7, p7)

3.8. Dr McPhee also pointed to Matthew's prompt early response to resuscitation, the rapid early weaning of his respiratory support, and his good oximetry readings (saturations of 100% in minimal oxygen), as indicators that Matthew's myocardial function was 'quite reasonable' in the initial stage of life (Exhibit C7, p7). The inference is that deterioration may not have occurred until later, when the hypothermic treatment was commenced. If this is so, it is less likely that late intra-partum perinatal hypoxic-ischaemic event was the cause of Matthew's later cardiogenic shock.

3.9. Dr McPhee suggested that there were a number of alternative explanations for Matthew's clinical presentation including:

- An unrecognised hypoxic-ischaemic event earlier in labour. This is consistent with Matthew's early seizures, his relatively mild cardiovascular depression, his profound neurological depression, and the relatively mild metabolic acidosis at birth;
- Birth trauma leading to intracranial injury. The history of malpresentation, prolonged second-stage labour, instrumental delivery and disseminated intravascular coagulopathy are consistent with this thesis, as is the increase in head circumference from 31cms at birth to 34cms soon after his death after 20 hours of life. Matthew died before CT scanning could be done, so an intracranial injury was not excluded;
- Infection leading to sepsis. Dr McPhee said:

'This potential presentation forms the basis of the clinical dictum by which all 'sick' newborns are considered to have bacterial sepsis, and investigated and treated as such, until proven otherwise by appropriate tests. In the case of Matthew Hutchinson, although aspects of the clinical picture are consistent with bacterial sepsis, I feel that the FBE results, and the later negative blood culture results convincingly exclude this diagnosis.'

(Exhibit C7, p8)

- Metabolic disorders. Although rare, such conditions must be excluded in babies presenting atypically, as most are inherited and may complicate future pregnancies;
- Congenital heart disease. Hypoplastic left ventricle syndrome and other similar disorders should also be excluded in presentations such as Matthew's.

Dr McPhee commented:

'It must be stressed that these alternative explanations for Matthew Hutchinson's clinical presentation are offered with the wisdom of retrospect, and have been raised only because there are some atypical features with respect to the assumed hypoxic-ischaemic causation of Matthew's clinical presentation and progression. However, exclusion of these alternative explanations would only be possible via a carefully conducted autopsy ...'

(Exhibit C7, p9)

3.10. Associate Professor Marshall's attitude to all of the possibilities postulated by Dr McPhee above was as follows:

- Traumatic birth injury was unlikely because a Ventouse suction cup was used rather than forceps (T39). When asked about the increase in head size, he wondered if this was a measurement error (T40). If an increase was established, he said that he would have advocated for a post-mortem examination (T41). This is a less than useful comment after the event;
- He agreed with Dr McPhee that early onset sepsis was unlikely on the evidence;
- The exclusion of metabolic disorders can, if everything is done, take months and is not warranted because they are so rare (T43);
- He conceded that, to be 'academically complete', congenital heart disease should be excluded (T43).

I will discuss Associate Professor Marshall's attitude to post-mortem examinations later.

3.11. It is particularly significant in this case that Matthew's blood pressure was not monitored adequately after he was born, so that it is not now possible to judge the adequacy of his myocardial function. Dr McPhee said that he was 'somewhat aghast'

that an arterial catheter was not inserted at an early stage so that Matthew's blood pressure, metabolism and coagulation could be closely monitored (T82).

- 3.12. Associate Professor Marshall admitted that Matthew's treatment in the neonatal unit should have included the placement of an arterial line (T31). He also agreed that, in the absence of an arterial line, Matthew's blood pressure should have been taken manually on an hourly basis (T35).
- 3.13. Associate Professor Marshall took personal responsibility for the non-placement of the arterial line. He explained that Matthew was 'left in the corner', and did not receive adequate nursing during the mid-afternoon shift changeover (T31). He also referred to the lack of an OHIO incubator, which had the facility for electronic blood pressure monitoring (T35).
- 3.14. Mrs Hutchinson had a different recollection of these events. She told me that Matthew had been in an OHIO crib since soon after he was born (Submissions, T140). If that is so, then there is little excuse for the lack of blood pressure monitoring.
- 3.15. Associate Professor Marshall said that because Matthew looked 'remarkably well' initially (T29), and because his blood chemistry was not seriously abnormal, his treatment plan was to provide airway support, phenobarbitone for his seizures, and to observe his progress (T31). In view of the fact that no arterial line was inserted, and his blood pressure was not taken until 1pm, it could hardly be said that this plan was effectively executed. This lack of monitoring of Matthew's progress in the first few hours after his birth becomes even more significant in the context of the hypothermic neuro-protection therapy which was introduced later.
- 3.16. Hypothermic neuro-protection therapy
Dr McPhee appended to his report several articles including a report of the Infant Cooling Evaluation ('ICE') trial conducted at the Royal Women's Hospital, Melbourne. The introduction to the article is as follows:

'Intrapartum hypoxia affects 3-5 per 1000 live births with moderate or severe HIE in 0.5-1 per 1000 live births. Of these between 10-60% die and at least 25% of the survivors have neurodevelopmental sequelae. In Australia this results in at least 60 seriously handicapped children annually, at a lifetime cost approximating A\$5,000,000 each for care. This is a major problem worldwide. In 2001 there is no specific treatment

to decrease brain damage from this cause. Hypothermia is the most promising clinically feasible manoeuvre to provide neurological rescue for neonates with HIE.'

(Exhibit C7, Reference 2, p1)

- 3.17. The article explains that there are two phases of neural death following a hypoxic-ischaemic injury:

'Primary neuronal loss is related to cellular hypoxia, which leads to exhaustion of high-energy metabolism (primary energy failure) and cellular depolarisation. However, many neurons do not die during this primary phase of injury. Rather, a cascade of pathologic processes is triggered leading to further loss of neurons starting after some hours and extending over days.

This secondary loss of neurons, termed secondary or delayed neuronal death, is associated with marked encephalopathy occurring 6 to 100 hours after the injury. The mechanisms involved include hyperaemia, cytotoxic oedema, mitochondrial failure, accumulation of excitotoxins, active cell death

...

Therefore a therapeutic 'window of opportunity' exists in the interval following resuscitation of the asphyxiated newborn, before the secondary phase of impaired energy metabolism and injury.'

(Exhibit C7, Reference 2, p1)

- 3.18. Dr McPhee commented:

'Although full details of the efficacy and safety of such hypothermic therapy must await the results of these trials, the preliminary data is tantalising. These encouraging results must be considered against a depressing dearth of beneficial therapeutic interventions in this clinical scenario, particularly when, even early in the clinical course of a particular patient, the likelihood of a devastatingly poor outcome can be predicted. Thus for example, in the case of Matthew Hutchinson, the combination of a low 5 minute Apgar score, the need for intubation during resuscitation, and the early onset of a moderate-to-severe encephalopathy predicts a 70% likelihood of death or adverse neurodevelopmental outcome. For this reason, a number of centres have chosen to apply this therapy in cases that fulfil entry criteria for the various trials. In the case of Matthew Hutchinson, the application of his clinical profile to the Inclusion Criteria of the ICE trial reveals that he would have been eligible for this trial. Thus, he was term, he had an encephalopathy, and he required mechanical ventilation beyond 10 minutes of age; note however that he did not satisfy either the Apgar or the acid-base criteria. Overall therefore, although some may argue that the application of such therapy should await clear evidence of benefit, I believe that the decision to attempt hypothermic neuroprotection therapy in the case of Matthew Hutchinson was not unreasonable.'

(Exhibit C7, pp9-10)

3.19. Associate Professor Marshall said that the therapy was applied to Matthew Hutchinson, commencing at around midday on 6 January 2002. The incubator was turned off, and cool packs were applied to Matthew's head.

3.20. Dr McPhee was critical of the way hypothermic neuro-protection therapy was administered to Matthew in a number of respects:

- Most importantly, Matthew's baseline cardiovascular and metabolic status was not established before the therapy was commenced. This is important, because the therapy can aggravate existing hypotension, acidosis and coagulopathy. No attempt was made to ascertain Matthew's blood pressure until during the therapy, when it could not be ascertained. It was not until an arterial line was placed by Associate Professor Marshall (it was finally placed at around midnight, 12 hours or so later, after much difficulty), that Matthew's blood pressure could be ascertained and by then it was alarmingly low.
- No attempt was made to address Matthew's worsening acidosis until around 8am, and no attempt was made to assess his coagulation status until around noon, by which time he was moderately coagulopathic. Dr McPhee commented:

'With respect to all of these issues, it is not possible to determine whether they were present from the outset, and hence would have warranted earlier intervention, perhaps even prior to inducing hypothermia, or whether they arose de novo during the induced hypothermia.'

(Exhibit C7, p10)

- Associate Professor Marshall admitted that Matthew's treatment was deficient in these respects. He said that protocols have since been developed which are comprehensive and which would avoid such deficiencies in future (T53). The protocols are Exhibit C6d.
- Matthew's hypothermic neuro-protection therapy was also inappropriate in that at one stage he was cooled excessively to less than 32°C for some time. Dr McPhee said that it is not clear whether the temperature was monitored continuously, as it should have been, or intermittently. He said that such 'overshooting' is not uncommon, even with continuous monitoring, and so he did not regard this criticism as significant. Associate Professor Marshall also said that the overshooting was 'not an issue' (T51).

3.21. The adequacy of monitoring generally

I have already outlined that an arterial line should have been inserted soon after Matthew's birth so that the blood pressure and oxygen saturations (blood gas analysis) could be continuously monitored. Dr McPhee said:

'Such information is necessary in order to assess the post-asphyxial adequacy or otherwise of the cardiovascular system, and to adjust, if necessary, the respiratory support. If hypotension or severe metabolic acidosis is present, then treatment with volume, dopamine, or bicarbonate can be considered; thereafter, the arterial access also allows for continuous monitoring of the responses to such interventions.'

(Exhibit C7, p11)

3.22. Such monitoring is particularly important when hypothermic neuro-protection therapy is being administered because cooling makes ascertainment of the patient's condition more difficult by conventional methods. Dr McPhee said:

'In the case of Matthew Hutchinson, the absence of early blood pressure measurements could be interpreted as being due to the fact that subjective clinical assessment was reassuring regarding the circulation (ie. good peripheral perfusion, adequate capillary refill, and easily palpated pulses), or alternatively, that hypotension was already present. If the former then this argues that the subsequent cardiogenic shock was predominantly a complication of the hypothermia; if the latter, then this casts some doubt on the prudence of inducing hypothermia without first addressing the hypotension. A similar analysis could be applied to the process of monitoring and managing a metabolic acidosis.'

(Exhibit C7, p11)

3.23. I accept that all of Dr McPhee's criticisms of the hypothermic neuro-protection therapy are valid, and I find that Matthew's treatment at the Flinders Medical Centre Neonatal Intensive Care Unit was deficient in each of the aspects he has identified.

3.24. Cause of death - no post-mortem examination

As I have already mentioned, Associate Professor Marshall advised the Registrar of Births, Deaths and Marriages that the cause of Matthew Hutchinson's death was cardiogenic shock and neonatal encephalopathy.

3.25. The fact that an autopsy was not requested, and no report was made pursuant to Section 31(1) of the Coroners Act so that an autopsy could be directed pursuant to Section 13(1)(e) of the Act, meant that the atypical features of Matthew's presentation, described by Dr McPhee, and the alternative explanations postulated by him, could not be explored. They remain unresolved. This is unsatisfactory from all points of view. As a coroner I am obviously concerned with an accurate

ascertainment of the cause of death, and the parents of the child are obviously also concerned, both to ascertain the truth of what happened, and to plan future pregnancies.

3.26. Dr McPhee said that, leaving the alternative explanations which should have been excluded to one side, the cause suggested by Associate Professor Marshall remained the most likely explanation for Matthew's death. I will therefore adopt it for the purposes of these findings.

3.27. When asked about his decision not to report the matter, and not to pursue a post-mortem examination, Associate Professor Marshall said:

'But in this context the clinical scenario was not that which one would normally see with the inborn errors of metabolism and, in particular, Lee's, so I didn't chase that and I think that in the context of autopsy - let me just say a couple of things. Firstly, increasingly over the last 15-20 years there's been a declining percentage of autopsies being done in our hospitals, and the reason for that is that by and large we have a much better idea of what is taking place. We know the answers mostly before the patient dies and I think we did here. Yes, there's always potential to get a return from doing an autopsy but the return is actually remarkably low, remarkably low. There's a cost to it too. You have only got to look at the tragedy of the last 20 years and the last five, in particular, in relation to the body parts at the Women's and Children's Hospital to get a feeling for what goes on and what that does to families. And we have become increasingly reluctant to push families into autopsy; almost saying to ourselves 'Is this really something we want to push on families?'. In this situation I thought the humane thing was - I believed I knew what we were dealing with here. I believe that an autopsy was a particularly academic exercise and I didn't want to put this family through any more; they'd been through enough. So I didn't encourage an autopsy. And I actually think it's a bit of a pity that the inborn error issue has been raised for them, because it's raising an issue which is extremely unlikely and I think put them under unnecessary stress.' (T44)

3.28. Indeed, when asked whether he was aware of whether a histopathologist might share his views, Associate Professor Marshall retorted that histopathologists have a 'vested interest' in saying that autopsies are important, and asserted that a lot of it is 'intellectual voyeurism' (T55). He went on to assert that when a pathologist examines an infant brain, the brain is 'never put back into the body for burial', a claim that is demonstrably false.

3.29. I must say that I find Associate Professor Marshall's attitudes to post-mortem examinations surprisingly idiosyncratic, unscientific, and ill-informed. He referred to the 'low return' on paediatric post-mortem examinations. It seems to me that if his views were to be adopted generally, relatively uncommon disorders such as those

postulated by Dr McPhee, would remain undetected, even undiscovered. His reference to the family being unnecessarily stressed by these issues being raised is paternalistic, and overlooks the distress which they now face when they are told that these issues must remain unresolved.

- 3.30. Finally, it should be observed that it was Mrs Hutchinson, through Dr Kremmidiotis, who caused these issues to be examined, albeit by a side wind. If it were not for the fact that they questioned the quality of obstetric care, the serious deficiencies in neonatal care provided by Associate Professor Marshall and his staff would not have become apparent.

4. Conclusions

- 4.1. On the basis of the above evidence, I find:

- Matthew Hutchinson died on 7 January 2002 as a result of cardiogenic shock and neonatal encephalopathy;
- The quality of obstetric care provided to Mrs Hutchinson at Flinders Medical Centre was appropriate;
- The quality of neonatal care provided to Matthew at Flinders Medical Centre was far from adequate. In particular, his condition was not adequately monitored as it should have been;
- The decision to provide hypothermic neuro-protection therapy was reasonable;
- The application of hypothermic neuro-protection therapy was inappropriate in a number of respects. In particular, Matthew's condition was not monitored adequately either before or after the therapy was commenced, his acidosis was not addressed, and he was excessively cooled;
- Because Matthew's death was not reported pursuant to Section 31(1) of the Coroners Act, and no post-mortem examination was performed, it is not now possible to determine whether, or to what extent, the inadequacies in treatment caused or contributed to his death.

5. Recommendations

5.1. Pursuant to Section 25(2) of the Coroners Act 1975, I make the following recommendations:

5.1.1. That the administration of Flinders Medical Centre assess whether protocols developed for the monitoring of neonates, and for the application of hypothermic neuro-protection therapy are adequate and are being implemented appropriately;

5.1.2. That Associate Professor Marshall reconsider his attitude in relation to the value of paediatric autopsies, and whether his practices in relation to advising the relatives of deceased infants in this area are appropriate.

Key Words: Neonatal Treatment; Hospital Treatment; Infant Deaths

In witness whereof the said Coroner has hereunto set and subscribed his hand and

Seal the 2nd day of June, 2005.

Coroner