CORONERS ACT, 2003

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 1st, 8th, 9th, 10th, 11th, 15th, 18th and 22nd days of December 2009 and the 9th day of July 2010, by the Coroner's Court of the said State, constituted of Anthony Ernest Schapel, Deputy State Coroner, into the deaths of Jessica Lee Stemmer and Thomas William Mahar.

The said Court finds that Jessica Lee Stemmer aged 7 hours, died at Ashford Hospital, 55 Anzac Highway, Ashford, South Australia on the 26th day of November 2006 as a result of disseminated intravascular coagulation following haemorrhage.

The said Court finds that Thomas William Mahar aged 8 days, died at the Women's and Children's Hospital, 72 King William Road, North Adelaide, South Australia following transfer from the Ashford Hospital, Ashford, South Australia on the 17th day of April 2007 as a result of multiorgan failure and coagulopathy following haemorrhage and hypoxia.

The said Court finds that the circumstances of their deaths were as follows:

1. Introduction

1.1. Jessica Lee Stemmer and Thomas William Mahar were born on Sunday 26 November 2006 and Monday 9 April 2007 respectively. Each child was born at the Ashford Hospital (the Ashford). Unfortunately, both infants died within a very short time of their births. Jessica survived for a period of approximately 7 hours after her delivery

and she died on the same day on which she had been born. Thomas died on 17 April 2007. He survived for approximately 8 days post delivery.

1.2. Jessica and Thomas were both delivered at or about term and their births were both by way of vaginal delivery. Another common feature was the fact that both deliveries had been assisted by ventouse extraction which involves the attachment of a suction cup to the scalp of the child and the administration of traction applied by hand.

1.3. In each child a subgaleal haemorrhage was diagnosed following delivery. Such a haemorrhage is known to occur as the result of the forces delivered to the scalp of the child during the course of a ventouse extraction. Fortunately severe subgaleal haemorrhages are rare, but their consequences can be devastating. A subgaleal haemorrhage is a haemorrhage into the subgaleal space. The subgaleal space is the virtual space that exists within the scalp between a thin fibrous sheet known as the galea and a very tough fibrous covering of the skull bone called the periosteum. The space extends from the forehead to the nape of the neck and also down the sides of the head to a point just above the ears, in other words more or less the entire area over which the scalp covers the skull. It is an uninterrupted space, and if a situation of haemorrhage should develop and remain unchecked, the space can be infiltrated by and is able to accommodate a large proportion of the child's circulation. It is said that some infants can lose 50% to 75% of their blood volume into the subgaleal space. Subgaleal haemorrhage is the term utilised to refer to the bleeding into the space that I have described and is caused by the rupture of the emissary veins which are connections between the dural sinuses and scalp veins. A subgaleal haemorrhage is a potentially lethal condition in newborns. The morbidity and mortality associated with subgaleal haemorrhage is due to the magnitude of the potential space that the haemorrhage might infiltrate and is caused by the consequent blood loss from the child's circulation. The loss of circulating blood is naturally in and of itself but one serious complication of a subgaleal haemorrhage. The haemorrhaging process can also give rise to coagulopathy, a clotting disorder. It is believed that a haemorrhage of the kind under consideration might ultimately exhaust all of the circulation's clotting capabilities with the result that there is nothing to check the progress of further haemorrhage. In due course the haemorrhage may result in hypovolaemic shock and death. The deleterious effects of a subgaleal haemorrhage might be reversed by timely intervention and, in particular, by way of the administration of

fluid, blood transfusion and, in order to counter coagulopathy, by the replacement of clotting factors that might be achieved by transfusion of fresh frozen plasma.

1.4. The force applied to the scalp during the course of a ventouse extraction is known occasionally to result in bleeding from small blood vessels into the subgaleal space. The amount of bleeding can vary considerably and the severity and consequences of the haemorrhage can vary accordingly. On the assumption that the ventouse procedure is undertaken within the relevant guidelines, the risk of such haemorrhage occurring is said to be outweighed by the effectiveness and utility of the extraction procedure, a procedure that is now in common use. I have no reason to doubt that as an accurate proposition. The ventouse extraction procedure has, to a greater or lesser extent, become the preferred method of assisted vaginal delivery. I was told in evidence that more recently qualified obstetricians have a greater familiarity with, and prefer to use ventouse extraction over, forceps extraction which among other things is said to carry greater risk of injury to the mother.

1.5. Jessica and Thomas both suffered subgaleal haemorrhages following their respective deliveries by way of ventouse extraction. There is little doubt that in the case of Jessica her death can be wholly ascribed to the haemorrhage or blood loss sustained by way of the subgaleal haemorrhage that she suffered. Equally in her case it is clear that the haemorrhage was the direct result of the application of force sustained to her scalp during the course of her ventouse extraction. However, as will be seen in the case of Thomas Mahar, the position regarding his cause of death is more complex because of the existence of other pathology that was discovered during the course of his autopsy. There had also been clinical evidence of other pathology at work. However, there is no doubt that he too suffered a significant subgaleal haemorrhage following his ventouse extraction and that it played a substantial role in the causation of his death.

1.6. The subgaleal haemorrhage sustained by each child was diagnosed clinically during the course of their short lives and pathologically at autopsy.

1.7. In each case the deliveries of Jessica and Thomas were conducted by an experienced obstetrician and gynaecologist, Dr Melissa Sandercock, who practised privately and enjoyed practising rights at the Ashford. Also in each case, the infants were attended by the same neonatal paediatrician following their respective deliveries. This person

was Dr Christopher Barnett, a medical practitioner of some considerable experience. Dr Barnett practised privately but also enjoyed practising rights at the Ashford. Dr Barnett was not involved in the birthing process of either child except to the extent that he was present at the moment of Thomas Mahar's delivery. Otherwise, Dr Barnett had no involvement in the pre-natal course in respect of either child or their mothers. It will be noted that these births occurred months apart. There is no connection between these cases other than by virtue of the fact that the same hospital, the same obstetrician and the same neonatal paediatrician were involved.

1.8. In this Inquest I examined a number of issues. Broadly speaking, my Inquest consisted of an inquiry as to whether there was any evidence to suggest that the respective ventouse deliveries had been undertaken in circumstances that did not accord with usual and well understood clinical practice and, secondly, whether the neonatal care that was delivered following their births was appropriately and competently administered and done so in a timely manner. The broad issue is whether in either case the death of the child could have been prevented. I also examined the issue as to whether, as a result of these unfortunate events, there is any initiative or measure that can be identified that might prevent a recurrence of these events.

2. Ventouse extraction

2.1. Tendered in evidence was a College Statement dated July 2009 of the Royal Australian and New Zealand College of Obstetricians and Gynaecologists entitled 'Prevention Detection and Management of Subgaleal Haemorrhage in the Newborn'[1]. This document describes a number of matters relating to the administration of vacuum extraction and the prevention, identification and management of subgaleal haemorrhage in that context. Although this College Statement post-dates the events with which this Inquest was concerned, I did not understand it to have identified any novel development in respect of the proper administration of ventouse assisted delivery that had not been applicable in 2006 or 2007. The document cites a conclusion of Vacca (2003) that significant subgaleal haemorrhage is almost always preceded by a difficult vacuum extraction as evidenced by a prolonged extraction with excessive number or strength of pulls, multiple cup detachments and/or completion of

[1] Exhibit C7f

delivery with forceps[2]. Accordingly, the document identifies a number of matters that are said to minimise the 'shearing' forces that ventouse extraction applies to the scalp with a view to the minimisation of the risk of infliction of a subgaleal haemorrhage. These matters include the following:

- The placement of the centre of the suction cup over the flexion point which is situated on the sagittal suture 3 centimetres in front of the posterior fontanelle and 6 centimetres from the anterior fontanelle;
- The placement of the cup across the sagittal suture evenly to avoid what is known as asynclitism with traction;
- The recognition of the fact that in certain circumstances appropriate cup placement may be impossible if there is significant deflexion or asynclitism of the head;
- That the traction should be steady and applied only with contractions and only with maternal effort;
- That adequate descent should be verified during each pull;
- That traction should not be unduly prolonged.

The document goes on to identify what are considered to be proper limits and requirements as far as this procedure is concerned. They include an upper time limit of 20 minutes from cup application, an assessment after 15 minutes as to whether further traction is warranted or whether recourse should be had to caesarean section. Also included is a suggestion that experts should regard a maximum of 3 pulls together with contractions as appropriate, although it goes on to suggest that several more pulls may be acceptable if the head has descended to the level of the pelvic floor or perineum especially if delivery is attempted without an episiotomy. The document also goes on to speak of whether, and how many, cup detachments might be regarded as acceptable and whether the rapid decompression caused by detachments might predispose to the infliction of subgaleal haemorrhage. As indicated earlier, the document also deals with the identification and management of subgaleal haemorrhage if sustained and I return to that issue in due course.

[2] Exhibit C7f, paragraph 6.1

3. Jessica Stemmer's cause of death

3.1. A post-mortem examination was conducted in respect of Jessica by Dr Teck Yee Khong, a pathologist, on 27 November 2006. Dr Khong's post mortem report was tendered to the Inquest[3]. Dr Khong also gave oral evidence. In his report and in his evidence Dr Khong described the cause of Jessica's death as disseminated intravascular coagulation following haemorrhage. The reference to disseminated intravascular coagulation is a reference to a process whereby all the clotting factors that are present in the blood are used up during a haemorrhage with the result that the blood loses its ability to clot and ceases to do so. As seen earlier, this condition is sometimes referred to as a coagulopathy. This condition naturally gives rise to further difficulties in terms of ongoing uncontrolled haemorrhage. The existence of Jessica's subgaleal haemorrhage was identified during the autopsy. Dr Khong described the extent of the blood loss involved. On reflection of the child's scalp there was 50ml of liquid blood and approximately 70ml to 100ml of clotted blood. This would represent a significant proportion of the blood circulation of an infant of approximately 38 weeks gestation. The blood loss in Dr Khong's opinion occurred after the baby had been born[4]. There was some divergence in the evidence as to the precise proportion of the child's entire circulation that this accumulation of blood in the scalp represented. Suffice it to say that if it is correct as some asserted that at birth a baby has approximately 85ml of blood per kilogram, then the accumulation of approximately 120ml to 150ml in the scalp would represent approximately 50% of the entire circulating blood volume was lost by way of haemorrhage. It has to be noted, however, that the child had received transfused blood at a time after her delivery. Nevertheless, even making allowances for that, the amount of blood located within the scalp represented substantial bleeding.

3.2. In his evidence Dr Khong was not able to describe at what point or points in time Jessica's major bleed had occurred except that it occurred after delivery. From his post-mortem examination he was not certain whether there had been one major bleed or a continuous bleeding process.

3.3. During Dr Khong's evidence there was some reference to the possibility of there having been a metabolic cause of death that could not be excluded by the autopsy. A

[3] Exhibit C5
[4] Transcript, page 45

metabolic autopsy was not performed in respect of the child. Metabolic autopsies are conducted if one suspects that there was a metabolic component in the child's death. Such autopsies are extremely expensive to perform and they need to be performed within 2 hours of the child's death. The suggestion that Jessica may have died from some cause other than, or in conjunction with, the subgaleal haemorrhage was not seriously developed during the course of the evidence. Dr Khong in any event adhered to his view that the cause of death was consequent upon the subgaleal haemorrhage. In coming to that conclusion he not only relied on his post-mortem examination, but he took into account the clinical history of the child following her delivery. Dr Khong was particularly impressed by the fact that the child was born initially well and then had a rapid decline and death. It is obvious to me that the clinical picture as described by the clinicians who dealt with Jessica that the main difficulty prior to her death had been the existence of the subgaleal haemorrhage and its consequences.

3.4. Dr Khong eliminated other possibilities that may have given rise to Jessica's haemorrhage, or at least have contributed to it, such as an inherent inability to clot. There were no platelet disorders. There had been no in-utero asphyxia in his view. Dr Khong believed that if Jessica had not suffered the subgaleal haemorrhage and the consequent disseminated intravascular coagulation, from what he had seen at autopsy Jessica would have been an otherwise healthy newborn child. I accept that evidence.

3.5. I have no doubt that the cause of Jessica's death was as Dr Khong has opined, namely disseminated intravascular coagulation following haemorrhage. I find that to have been the cause of Jessica Stemmer's death. I find that the haemorrhage in question was a subgaleal haemorrhage. As will be seen there was no explanation for the haemorrhage other than that it was sustained as the result of the force applied to the child's scalp during her ventouse assisted delivery. I find that force to have been the cause of the haemorrhage.

4. Thomas Mahar's cause of death

4.1. A post-mortem examination was conducted in respect of Thomas by Dr Lynette Moore, a pathologist, on 18 April 2007. The child had been delivered on 9 April and died on 17 April 2007. Dr Moore's post-mortem report was tendered to the Inquest[5].

[5] Exhibit C6

Dr Moore also gave oral evidence. Dr Moore described the cause of Thomas' death as multiorgan failure following massive subgaleal haemorrhage. I return to the question of the description of Thomas' cause of death in a moment.

4.2. The findings at Thomas' autopsy were quite unlike those in respect of Jessica. Although Thomas had also sustained a subgaleal haemorrhage, there was other serious pathology which had complicated his presentation following delivery. There was powerful clinical evidence to suggest that Thomas had suffered a severe hypoxic insult during the course of his mother's late stage labour and his delivery. There were also abnormalities identified within the placenta and umbilical cord of the child. Thomas had also sustained a spinal injury. All of these matters distinguished his case from that of Jessica Stemmer.

4.3. As far as Thomas' subgaleal haemorrhage is concerned, Dr Moore described it as extensive. There was a subgaleal haemorrhage weighing 94 grams that Dr Moore was able to measure. There was also a similar amount of blood that was adherent to the deep sub tissues and which was not able to be weighed. This represented an extensive and significant haemorrhage. There were indications that the haemorrhage had occurred at different times having regard to the fact that the child lived for 8 days and received blood transfusions. This impression accorded with the clinical course relating to Thomas before he died. At the time of Thomas delivery, the subgaleal haemorrhage was not considered to be large and early haemoglobin tests had not suggested that he had lost blood to an alarming level. It was evident that when clinicians began administering blood and plasma transfusions to Thomas in the morning following his delivery, he bled copiously into his already existing subgaleal lesion. Nevertheless Dr Moore's findings indicate that during the days of Thomas' short life there had been extensive haemorrhage into the subgaleal space.

4.4. The child had also suffered a posterior fossa subdural haemorrhage. Whereas a subgaleal haemorrhage involves bleeding into the tissues of the scalp, a subdural haemorrhage occurs within the skull. In this case the subdural haemorrhage had occurred into the space between the cerebellum and the fibrous membranes situated at the back part of the skull. Professor Roger Pepperell, an expert obstetrician, told me in evidence that such haemorrhages are not commonly associated with ventouse delivery, but were more commonly associated with a difficult rotation forceps delivery. Professor Pepperell did say that such a haemorrhage can certainly occur in

the setting of intrapartum hypoxia (deprivation of oxygen sustained during the birthing process). In Thomas' case there was evidence of hypoxia seen during the birthing process itself and there was clinical evidence of the same seen immediately after birth in the form of a severe acidosis. The posterior fossa subdural haemorrhage itself was not to be diagnosed radiologically until the early hours of the morning of the day following Thomas' birth. Dr Moore postulated two possibilities for the existence of a posterior fossa haemorrhage. The first of these was the possibility that the child had sustained hypoxic injuries to the blood vessels involved during an episode of general hypoxia[6]. The alternative possibility was that once the baby had developed the large subgaleal haemorrhage and had become acidotic, he then developed coagulopathy which could have resulted in bleeding in many parts of the body including the brain[7]. In her report, Dr Moore suggests that it was likely that Thomas was already haemodynamically compromised as evidenced by decelerations in his heart rate prior to his delivery together with some placental abnormalities that were identified at autopsy, and that all of this resulted in an increased bleeding tendency. On the other hand, Dr Vineesh Bhatia who was one of the neonatologists involved in Thomas' treatment at the Women's' and Children's' Hospital told the Court that posterior fossa bleeds are sometimes seen following instrumental deliveries including ventouse deliveries[8]. In the event it has not been possible to choose between the possibilities. Dr Moore was clear in her view that many of the child's difficulties had in any event arisen prior to his delivery.

4.5. The child had also suffered a cervical spinal injury that involved stretching of the cervical spine and the creation of widening between the base of the skull and C1 and between vertebrae C1 and C2. A spinal injury had been suspected clinically but was not properly identified until the carrying out of the post-mortem. However, Dr Moore herself had noticed an abnormality upon performing a brief external examination that had included palpation of the neck. There was no injury to the spinal cord and no evidence of haemorrhage. In her report Dr Moore suggests that this type of neck injury is not typically associated with ventouse delivery. Professor Pepperell expressed the view that he did not think that there was any relationship between ventouse delivery and this kind of injury and told me that he had no explanation as to why it had been found in this case. There was nothing in the circumstances of

[6] Transcript, page 104
[7] Transcript, page 105
[8] Transcript, page 645

Thomas' delivery that would have exposed him to the risk of sustaining an injury of this nature. The origin of this injury and its contribution if any to Thomas' overall pathology is far from clear.

4.6. There was also evidence within Thomas' lungs that were indicative of him having an asphyxial or hypoxic episode in-utero[9].

4.7. As far as the child's placenta was concerned, it was small, being within the tenth percentile, particularly having regard to the baby's size which was 3650 grams which is quite normal. The small size of the placenta could mean that in any adverse episode during birth the child might have a depleted '*placental reserve*'[10]. The umbilical cord evidenced a number of abnormalities which could predispose to the formation of blood clots within the placenta. Foetal thrombotic vasculopathy was confirmed in Thomas' placenta. These abnormalities in themselves can give rise to adverse foetal outcomes, particularly neurological damage or damage in other organs[11].

4.8. In cross-examination Dr Moore reiterated her view that there had been an asphyxial episode in-utero. The placental abnormality may have had some role to play in this. She also acknowledged the existence of an hypoxic ischaemic injury to the child's brain that was identified was in keeping with a possible consequence of an hypoxic event during the birthing process[12]. Thus she acknowledged that even without any other pathology such as a subgaleal haemorrhage, Thomas may have been born with some significant brain impairment in any event. I add here that Professor Pepperell expressed the view that Thomas' condition at birth clearly reflected hypoxia of a marked degree and that this would clearly have adversely affected his chances of survival[13].

4.9. Dr Moore agreed that the foetal distress that the child had been experiencing may have been triggered by a haemodynamic compromise which in practice had two consequences. Firstly, the foetal distress might well trigger the need for an assisted delivery and, secondly, it might constitute a factor that might dictate a poor outcome

[9] Transcript, page 100
[10] Transcript, page 108
[11] Transcript, pages 109-110
[12] Transcript, page 119
[13] Transcript, page 941

from an assisted delivery. In other words, the fact that the assisted delivery had to be instituted rendered the child more susceptible to sustaining a subgaleal haemorrhage.

4.10. When asked in her oral evidence to evaluate and discuss her stated cause of death, Dr Moore confirmed her opinion that the cause of death was related to the subgaleal haemorrhage, although she acknowledged that there were many other complicating factors. In her view there were already problems in existence prior to Thomas' delivery that had resulted in an increased bleeding tendency with the result that Thomas might be predisposed to bleed more heavily into the subgaleal space[14]. In addition, Dr Moore suggested that an hypoxic in-utero event such as the one that Thomas had sustained could have adversely affected his clotting abilities which could have compromised an ability to stem bleeding from a posterior fossa subdural haemorrhage as well as a subgaleal haemorrhage[15]. There is significant support for this contention from the expert obstetrician Dr Neil Roy who also said in evidence that hypoxic ischaemia can lead to coagulation difficulties so that if a baby does begin to haemorrhage somewhere, such as in the subgaleal space or within the skull itself, the hypoxia will interfere with the coagulation process and may also interfere with the production of new coagulation factors such that a baby who is poorly perfused and hypoxic is more likely to bleed. Dr Roy's view was that there was a combined effect of severe hypoxic ischaemia and the subgaleal bleed. He also opined that the hypoxia ischaemia on its own did not cause Thomas' death, nor did the subgaleal haemorrhage on its own, but the fact that the subgaleal bleed was exaggerated because of the severe hypoxia had led to disseminated intravascular coagulation which was eventually the cause of death[16].

4.11. Mr Bonig who appeared on behalf of the Women's and Children's Hospital (WCH) challenged Dr Moore as to her opinion concerning Thomas' cause of death. While acknowledging that there had been an adverse hypoxic event before or during the delivery process and that many of the problems with Thomas had occurred subsequent to that, Dr Moore resisted the suggestion that it would be more accurate to conclude that the cause of death was multiorgan failure as a result of complications consequent upon an in-utero hypoxic event. She said that it was not clear as to what extent those other hypoxic changes might have been recoverable, whereas the ongoing

[14] Transcript, page 112
[15] Transcript, page 122
[16] Transcript, pages 834-835

haemorrhage was a major problem[17]. The difficulty during the child's short life, as she understood it to have been, was the severe coagulopathy and ongoing bleeding and that this was the problem that could not be controlled. It was for that reason that she described the cause of death in terms of the existence of the subgaleal haemorrhage. On the other hand, Associate Professor Ross Haslam, the principal neonatologist who treated Thomas at the WCH, expressed the view that Dr Moore's stated cause of death was poorly expressed. He agreed that at the end the child had suffered an irretrievable multiple organ failure. However, his view was that although the haemorrhaging process had begun at birth, it had not reached fatal levels at birth. It had progressed over time. He believed that the baby's progression to the final events of death had been due to ongoing haemorrhage for which a coagulopathy was a critical primary factor[18]. He also pointed to the role of the in-utero hypoxia. For Associate Professor Haslam, if one were to single out something that was critically important to the baby's cause of death it was the coagulopathy that was the result of a combination of effects, certainly the ongoing hypotension (low blood pressure) from the blood loss, the extensive tissue damage and also the hypoxic ischaemia. Nevertheless he agreed that the subgaleal haemorrhage was a significant contributing factor[19]. Indeed, he was inclined to agree that Thomas' chances of survival would have been better and he might have survived if the subgaleal haemorrhage had been removed from the equation[20]. I accept that analysis of the situation. Dr Bhatia also believed Dr Moore's expressed cause of death to have involved an over simplification of the child's pathology. He too placed heavy emphasis upon the hypoxia and upon the involvement of the posterior fossa bleed, but nevertheless agreed that the subgaleal haemorrhage was part of the overall problem[21]. I accept that analysis as well.

4.12. It seems to the Court that there is no essential difference between Dr Moore on the one hand and Associate Professor Haslam and Dr Bhatia on the other as to the cause of death. All agree that coagulation difficulties were an important factor in the child's death, and that both the haemorrhage from its differing sources and the hypoxia were also important contributing factors underlying that coagulopathy. Dr Moore would perhaps place a greater emphasis upon the haemorrhaging, and in particular the

[17] Transcript, page 123
[18] Transcript, page 728
[19] Transcript, page 729
[20] Transcript, page 729
[21] Transcript, pages 673-676

subgaleal haemorrhage as being a cause of the coagulopathy, whereas Associate Professor Haslam placed more emphasis upon the coagulopathy as having been a significant contributor to the ongoing haemorrhage. Dr Bhatia was of the view that '*it wasn't just one thing it was all these things together which caused the hypoperfusion which caused the hypotension which in turn caused the capillary leak syndrome and the multi organ dysfunction*'[22], but I do note that Dr Bhatia favoured the posterior fossa bleed as an explanation for the severe coagulopathy[23]. Two things are undeniable, however, and that is that Thomas exhibited both hypoxia and a subgaleal haemorrhage at the time of birth and that these conditions ultimately played significant roles in his deterioration and death. In this regard I note that even though the subgaleal haemorrhage was not large at birth, when Thomas was given transfusions of blood and plasma in the early hours of the morning following his delivery, the subgaleal haemorrhage bled even more to the point where it became extensive. I acknowledge as well the strong body of evidence that there was also a possible contribution to the child's coagulopathy made by the subdural posterior fossa bleed which by the morning after his delivery had also been identified as being extensive[24]. In my view the cause of Thomas Mahar's death can be described as multiorgan failure and coagulopathy following haemorrhage and hypoxia. I find that to have been the cause of his death. To my mind it is also clear that the subgaleal haemorrhage was a major component of ongoing haemorrhage and that it had as its primary mechanical cause the force applied to the child's scalp during his ventouse assisted delivery. I am unable to make any finding as to the origin of the subdural posterior fossa haemorrhage.

5. <u>The deliveries and post natal care of Jessica Stemmer and Thomas Mahar - General</u>

5.1. In these findings I shall deal in turn with the circumstances surrounding the deliveries of Jessica Stemmer and Thomas Mahar as well as their post natal care.

5.2. The ante-natal courses and pregnancies in relation to both Jessica and Thomas were uneventful.

[22] Transcript, page 673
[23] Transcript, page 678
[24] As noted by Dr Bhatia at Transcript, page 644

5.3. As indicated earlier, both deliveries took place at the Ashford and both were conducted by Dr Melissa Sandercock. Dr Sandercock gave evidence during the course of the Inquest.

5.4. Dr Sandercock is an obstetrician and gynaecologist. She received her basic medical degrees from the Flinders University in 1987. She obtained her Fellowship of the Royal Australian and New Zealand College of Obstetricians and Gynaecologists in 1997. She commenced private practice in 1998 and remains in private practice to this day. Dr Sandercock is an experienced obstetrician and gynaecologist. She is a member of the state committee of the Royal College to which I have referred. She is also Chairperson of the Calvary Hospital Perinatal Committee that reviews incidents and adverse events occurring within the maternity unit of that hospital. She is also a member of the Clinical Review Committee of that hospital. As a trainee obstetrician and gynaecologist between 1990 and 1997, Dr Sandercock performed several hundred deliveries. From 2005 to 2009 she had performed approximately 900 deliveries. Of that number 69 were by way of ventouse assisted deliveries. In the 6 years prior to 2004, which were the first years of her private practice, she performed a similar number of deliveries overall as well as a similar number of instrumental deliveries. Most of her instrumental deliveries were by way of ventouse. Unassisted vaginal deliveries account for just fewer than 50% of her overall delivery rate per year. Dr Sandercock told me that she has delivered training to other registrars and resident medical officers in respect of ventouse deliveries. Dr Sandercock acknowledged that one of the risks involved in the use of ventouse suction is the infliction of subgaleal haemorrhage. As of 2006 she had had no experience with such an outcome having occurred in any of her own deliveries.

5.5. Dr Christopher Barnett was asked to attend at the Ashford to examine both Jessica and Thomas after their respective deliveries. He was responsible for their post natal care. Dr Barnett was at that time an experienced neonatal paediatrician. Dr Barnett now works in a different specialty. Dr Barnett was conferred with his basic medical degrees in about 1990. He was admitted to the Fellowship of the Royal Australasian College of Physicians in 1997. Between 1997 and 2002 he was a consultant and senior consultant respectively in neonatal medicine at the WCH in Adelaide. Between 2002 and 2007 he was a consultant neonatal paediatrician in private practice. Since 2007 Dr Barnett's specialty has involved clinical and metabolic genetics, both

in Canada and South Australia. Dr Barnett's curriculum vitae[25] reveals extensive clinical experience in neonatal paediatrics and, as well, demonstrates his wide academic contribution to this specific field of medicine.

5.6. Dr Barnett's experience at the WCH in the period between 1997 and 2002 involved practice in neonatal medicine in a neonatal intensive care environment within that hospital. The environment involved in that setting was referred to as a Level 1 environment that involves the delivery of neonatal care to newborns who require special care and consultant opinion. The Ashford did not provide Level 1 care. It did not have a neo-natal intensive care unit. Rather it provided Level 2 care. Neonates who required intensive care could be transferred from the Ashford to the WCH if the need arose. Such a process is known as retrieval.

5.7. Jessica Stemmer died at the Ashford. Thomas Mahar died at the Women's' and Children's Hospital some days after his death. He had been retrieved to the WCH from the Ashford.

6. Jessica Lee Stemmer

6.1. The delivery of Jessica Stemmer

In the course of the Inquest two witnesses gave oral evidence about the circumstances of Jessica Stemmer's delivery on 26 November 2006. Those witnesses were a registered midwife, Ms Helene Maddern, and Dr Sandercock. Ms Maddern told me that Mrs Stemmer had been admitted to the Ashford at approximately 10pm on 25 November 2006. Ms Maddern commenced her shift at 7am the following day. At 11:30am Ms Maddern conducted a vaginal examination which established that Mrs Stemmer was fully dilated. Dr Sandercock was notified of that result. Dr Sandercock told Ms Maddern to instruct the mother to commence pushing. However, in due course Mrs Stemmer became quite fatigued and little progress had been made with pushing. Dr Sandercock came to the hospital and arrived at or around 12:30pm whereupon Dr Sandercock decided to administer a ventouse assisted delivery. Ms Maddern had a role to play in the delivery. She administered an epidural top-up and set the pressure on the ventouse. Owing to a wrist injury, Ms Maddern told me that she had some difficulty reaching or maintaining an appropriate pressure but ultimately did obtain and set a pressure at 60. The ventouse delivery then proceeded.

[25] Exhibit C9

6.2. When asked in evidence as to whether she could recall how many attempts (or pulls) were made to administer the ventouse delivery Ms Maddern said that she could not absolutely recall but that her recollection was that there were '*at least 3*'[26]. An episiotomy was performed as well. The delivery was effected at 1323 hours. According to Ms Maddern, Jessica appeared to be flat upon delivery insofar as she was pale and cold and not making any noise[27]. Ms Maddern herself believed that Jessica should be accorded a certain Apgar which Dr Sandercock did not agree with. Any discrepancy is not material. In Ms Maddern's cross-examination she was pressed as to a number of matters. Specifically, she recalled that the difficulty associated with her wrist injury was that she did not have enough strength and had difficulty raising the pressure to a level of 50 (mm of mercury) but that once she attained a pressure of 60 she was able to maintain that level. Ms Maddern was definite in her assertion that the pressure stayed at the appropriate level during the delivery[28]. She asserted that the cup remained attached to the foetal scalp at all times during the delivery. She maintained that there were at least three contractions involved in the delivery. When pressed as to whether she disagreed with Dr Sandercock's assertions that there were in fact just the three, Ms Maddern said:

> 'There may have been more.' [29]

Ms Kereru, counsel assisting, in cross-examination invited Ms Maddern to comment on Dr Sandercock's notations in the Ashford clinical record that had included the following assertion:

> 'Delivery over three contractions, no difficulty.' [30]

Ms Maddern agreed that the delivery had occurred over about three contractions that had coincided with three pushes by Mrs Stemmer. As to the accuracy of the observation noted by Dr Sandercock that there was no difficulty in the delivery, Ms Maddern said that her recollection was that it had been a difficult delivery. When asked to extrapolate on that assertion she said:

> 'No, I don't know why. It was just the force of the delivery having to - the strength required with the contractions. Mrs Stemmer's contractions might not have been strong enough. She was exhausted.' [31]

[26] Transcript, page 149
[27] Transcript, page 150
[28] Transcript, page 178
[29] Transcript, page 179
[30] Transcript, page 183

When asked to explain further what she meant, Ms Maddern said that the force used by Dr Sandercock could have been regarded as '*appropriate force*'[32]. She said:

> 'I don't know if it was - it could have been appropriate for the situation that she needed to pull that hard with the contractions.' [33]

Ms Maddern said that she could not say whether there had been more force used than was necessary and agreed with counsel assisting that she may not have had sufficient experience to be able to compare the relative use of force in respect of other ventouse deliveries that she had seen. In any event, Ms Maddern maintained that the pressure within the cap was no more than 60 which was to be viewed as acceptable, although she said that some doctors might view it as slightly high. There was no evidence to support her contention that views as to the appropriateness of a pressure of 60 might differ. Ms Maddern reiterated that on no occasion did the cap come off, notwithstanding the amount of force that she said she had seen administered[34].

6.3. Some of Ms Maddern's statements as given in her oral evidence gave me the impression that at the time of Jessica's delivery she had viewed the amount of force that had been administered by Dr Sandercock as something beyond the norm, but she then disinfected those statements by reference to her relative lack of experience with the procedure. Ms Maddern at times was also less than clear about the number of tractions that Dr Sandercock had applied and whether it had exceeded three or not. In the event, while I had some misgivings about Ms Maddern in terms of what she said she recalled and what she seemed to be prepared to openly discuss, there is no evidence that would have contradicted anything that Dr Sandercock was to say in her own evidence about the circumstances of Jessica's delivery, particularly in respect of the amount of force used and the number of times that traction had to be applied before delivery was successfully effected.

6.4. In her evidence Dr Sandercock told me that when she arrived at the Ashford at about 12:50pm, Mrs Stemmer was uncomfortable and was distressed with pain having been pushing for some period of time. Mrs Stemmer had also been vomiting and had been awake all night. She was quite exhausted. Upon vaginal examination, Dr Sandercock detected a mild degree of asynclitism where the baby, instead of presenting in a direct

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[31] Transcript, page 184
[32] Transcript, page 185
[33] Transcript, page 185
[34] Transcript, page 185

position, has the head rotated slightly to the side. Given the mother's state of exhaustion, the lack of progress and some evidence of foetal heart rate deceleration, and having regard to the result of the vaginal examination, Dr Sandercock felt that she should hasten the delivery of the baby by way of ventouse extraction. When asked as to why she chose that method she said simply that it was the instrument with which she was most familiar and which she used most frequently. She said there was nothing in the positioning of the baby that contraindicated a ventouse delivery. In particular, the asynclitism was not excessive and indeed was mild.

6.5. Dr Sandercock told me that she utilised a Mityvac vacuum cup. The pump was operated by the midwife, Ms Maddern. When asked about the circumstances surrounding the raising of the appropriate pressure, Dr Sandercock gave evidence that the pressure was applied in the manner that she had instructed and that once traction was applied there was no loss of pressure. As to the position of the cup, Dr Sandercock described a position relative to the child's scalp that accorded with usual practice. The cup remained attached at all times during the delivery and the pressure never dropped out of the appropriate range. According to Dr Sandercock the delivery took place over three contractions. This figure accords with her clinical notes. She said that the first two contractions brought the foetal head onto the perineum and whilst the head was distending the perineum, the foetal heart rate dropped down to about 100 beats per minute. To her this signified possible onset of bradycardia. With the next contraction, which was the third contraction, Dr Sandercock cut an episiotomy and delivered the baby. When compared to other ventouse deliveries that she had conducted, Dr Sandercock said that it was not a difficult delivery and that she had no recollection of having to apply more force than was usual.

6.6. The child was delivered at 1323 hours. Dr Sandercock has recorded that the Mityvac cup was applied at 1317 hours. This would mean that there was a 6 minute delivery time which is well within acceptable limits.

6.7. Dr Sandercock also said there had been no rotation of the child during the procedure[35].

6.8. In cross-examination by Ms Kereru, counsel assisting, Dr Sandercock described what one might experience in a ventouse delivery that could be characterised as difficult. A difficult delivery, which indeed she has experienced with other ventouse deliveries

[35] Transcript, page 297

she has performed, might involve rotation of the head, a delay in delivery or a delivery over 7 or 8 contractions that might involve the cup removal and the eventual use of forceps. I did not understand any such features to have been involved in Jessica's delivery.

6.9. When a ventouse delivery is effected, a feature known as a chignon on the baby's scalp is usually the result. The chignon is effectively the temporary reshaping of the scalp caused by the suction and traction applied by the ventouse cup. Dr Sandercock told me that in this case she believed that the chignon was more obvious than normal and that she believed at that point that the child had suffered a cephalhaematoma. A cephalhaematoma represents bleeding in the scalp but is by no means as serious as subgaleal haemorrhage and is usually confined to one particular area of the scalp. In the event it proved not to be a cephalhaematoma but a subgaleal haemorrhage.

6.10. I asked Dr Sandercock whether there was any explanation for the subgaleal haemorrhage having been sustained in Jessica's case having regard to the fact that there had been no cup detachment, that no other difficulty that one might typically associate with a ventouse delivery had been experienced and that there had been an absence of any identified previous disposition for the infant to have bled. Dr Sandercock said:

> 'I can only say that it is a rare and unexpected complication.' [36]

Dr Sandercock agreed that in terms of the literature that relates to ventouse delivery and the possible complications in its administration, of which there were none in this case, Jessica Stemmer's subgaleal haemorrhage appeared to be anomalous[37]. It certainly seemed to the Court that if all of what Dr Sandercock said about the circumstances of the delivery was to be believed, the infliction of a subgaleal haemorrhage of the severity sustained was significantly against the run of play.

6.11. Dr Barnett was summoned to examine the child. Dr Sandercock told me that following Jessica's delivery, she was concerned for the baby's wellbeing. The baby was quite pale and she had a low heart rate initially. She entered a description of the child in her notes as having been the subject of '*shellshock*'[38]. This signifies in her

[36] Transcript, page 323
[37] Transcript, page 323
[38] Transcript, page 256

view that the baby was pale and floppy, did not have a lot of voluntary movement and did not have a good tone. It was for those reasons that she called for Dr Barnett.

6.12. I add that the circumstances of Jessica's and Thomas' deliveries and neonatal care were examined by experts in these fields and reported on by those experts. I will describe their reports and evidence in other contexts in this finding, but as far as the management of Jessica's delivery is concerned, neither Dr Roy who is a neonatal specialist and expert nor Professor Pepperell who is an obstetric specialist and expert, could identify any material area of criticism that could be levelled at the quality of care given to Mrs Stemmer and Jessica during the birthing process. The only matter of materiality was raised by Dr Roy who made the observation that Dr Sandercock's clinical notes suggested that there had been a marked degree of asynclitism and that this slight asymmetry of the presenting part of Jessica's head may have increased the risk of foetal scalp trauma, although it would not in his view have acted as a contraindication to the use of ventouse extraction. This view appears to have been based upon a possible misinterpretation of Dr Sandercock's clinical note. Dr Sandercock gave evidence that her note as to asynclitism was not intended to indicate a marked degree of the same. She told me on oath that there was a mild degree of asynclitism. Dr Sandercock's notes are open to different interpretations. In any event I accept Dr Sandercock's evidence, corroborated as it is by Dr Barnett's evidence that to his eyes, from the position of the chignon, there was no suggestion that the ventouse cup had been placed in other than the normal position. Professor Pepperell in his report[39] specifically states that he could find no evidence on the documentation that the techniques used by Dr Sandercock were inappropriate or that there were significant difficulties in performing the procedure that increased the risk of subgaleal haemorrhage. He also states in his second report[40] that Dr Sandercock's decision to deliver the child by way of ventouse was appropriate in all of the circumstances.

6.13. There is no or no sufficient evidence that any of the risk factors that might typically explain a subgaleal haemorrhage were in existence in this case. There is no evidence that Jessica had any predisposition to haemorrhage. Accordingly, aside from a suspicion that the traction applied in the administration of the ventouse was accompanied by insufficient maternal effort due to exhaustion, a matter in respect of which Mrs Stemmer can naturally be accorded no blame, and while it is clear that the

[39] Exhibit C11, paragraph 4.1
[40] Exhibit C11a, paragraph 3

subgaleal haemorrhage had as its primary cause the force delivered by the traction of the ventouse, there is really no available explanation as to why Jessica sustained a subgaleal haemorrhage of such severity.

6.14. Jessica Stemmer's post natal care

Dr Barnett was not present at the Ashford at the time of Jessica's delivery. However, he was on call. He received a call on his mobile phone to attend the Ashford in respect of Jessica. He had been attending a sporting event at Forestville and, after he received the call, he took approximately 10 minutes to travel to the Ashford by which time Jessica had been transferred from the delivery suite to the nursery. Jessica had been delivered at 1:23pm. It appears that Dr Barnett examined Jessica for the first time at approximately 1:40pm.

6.15. After initial resuscitative efforts, Jessica maintained a picture of outward stability for a time until she collapsed at about 4:15pm. Jessica died later that same day at the Ashford. Her death was certified at 8:28pm. Resuscitative efforts had been ceased at 7:38pm. Except for a period of less than 30 minutes, Dr Barnett had been in attendance at the Ashford during the afternoon and evening in question. In addition, when Jessica's condition had been identified as critical, a retrieval team from the WCH attended the Ashford and the members of that team also delivered treatment to Jessica. As events transpired, Jessica was not to be retrieved to the WCH.

6.16. I now describe Jessica's neo-natal care. Dr Barnett made notes of his initial observations of Jessica. He noted that she had a moderately sized subgaleal haemorrhage with typical boggy swelling. She was hypotonic at first. He noted that Jessica was pale and queried whether she had anaemia secondary to blood loss. That was Dr Barnett's clinical impression. He ordered the administration of 30mls of normal saline 'stat', which means urgent, and this was then administered. Dr Barnett ordered this because normal saline is the universally accepted volume replacement strategy for a neonate. Given intravenously it bolsters the child's perfusion and it will typically render an improvement in a descriptively flat baby as its vascular system fills with the fluid. The dose of 30mls was calculated on the basis of Jessica's weight of almost 3 kilograms at 10ml per kilogram. Saline was administered in bolus form which means that it was given quickly as opposed to a continuous infusion. Dr Barnett noted that the baby's appearance improved after the administration of the 30mls of saline.

6.17. Dr Barnett obtained arterial blood gas results in respect of Jessica post delivery. The blood gas results obtained at around 2:26pm revealed a number of matters of concern having regard to Jessica's initial presentation and in the light of her clinically diagnosed subgaleal haemorrhage. After noting those results Dr Barnett obtained an analysis of the child's arterial cord gas at birth which should reflect the child's various values at the time of birth. The purpose of this exercise was to compare her condition at birth at 1:23pm with her condition at approximately 2:25pm when the later samples were taken. A comparison of the values from the two blood gas results revealed that there had been a descent of some significance in the child's haemoglobin from 18.1 at the time of birth to 11.2 as reported at 2:26pm. An average haemoglobin level would have been 16.5 plus or minus 3. This means that the haemoglobin in Jessica's case had descended from within the normal range at birth to a level that was significantly below normal and this could not wholly be explained by dilution of her circulation by the administration of saline. Rather, it was reflective of significant blood loss.

6.18. There were other results that were of concern. The child's pH value was 7.08 which is below the normal range of 7.35 to 7.45. The arterial cord gas at birth revealed a pH of 7.37 which is within the normal range. Therefore there had been a descent in the pH value. The level of 7.08 represented a level of acidosis. Other readings suggested that the acidosis was of a metabolic as opposed to respiratory origin. Other matters eliminated the possibility that Jessica had experienced a foeto-maternal haemorrhage. By a rather belaboured forensic route, Dr Barnett acknowledged in his evidence what must have become rapidly evident at the time of these events, and that was that Jessica had suffered significant loss of blood by way of a subgaleal haemorrhage sustained at delivery. There can in my view be no suggestion other than that this state of affairs was very clear to everyone on the day.

6.19. Dr Barnett did not regard the haemoglobin level descent to 11.2, which was a level reflective of Jessica's blood loss, as giving rise to a need for Jessica to receive a blood transfusion urgently.

6.20. Jessica was not to have another blood gas test until after her eventual collapse at about 4:15pm. In particular, there was no haemoglobin measurement taken in the intervening period beyond the test that at 2:26pm revealed that it had descended to that level of 11.2. It is said that this was a significant omission having regard to the significant descent from its level of normality at birth. I return to that issue in due

course but make the observation that a debate that ensued in the Inquest as to whether Jessica had continued to bleed by way of a subgaleal haemorrhage during the course of the afternoon or whether she had a very acute re-bleed at the time of her collapse may have been all the less complex if haemoglobin monitoring had taken place beyond the level that was established at 2:26pm. I add that there was also no further analysis of Jessica's acidosis before her collapse.

6.21. On the other hand, from a clinical point of view, Dr Barnett genuinely, I find, entertained the belief that Jessica, notwithstanding the existence of the subgaleal haemorrhage and the abnormal results of the 2:26pm blood gas test, was stable and in particular enjoyed haemodynamic stability as evidenced by oxygen saturations of 100% and a heart rate of 160 which Dr Barnett regarded as very reassuring[41]. Dr Barnett believed this to be normal and not tachycardic as one might expect in a child who has suffered a significant blood loss. During this period no monitoring of Jessica's blood pressure had taken place. Blood pressure monitoring by way of invasive monitoring was not available in a Level 2 hospital such as Ashford and monitoring by way of cuff was said to be an imperfect exercise. In any event, Dr Barnett believed that blood pressure monitoring was not indicated. It is in respect of this contention that there is also some debate.

6.22. Having regard to the anaemic state of the child and also the need to maintain a satisfactory circulating blood volume, Dr Barnett decided that Jessica required a transfusion of blood in the form of packed cells. As I have mentioned, he did not believe that the transfusion was required immediately and, indeed, an immediate transfusion of cross-matched blood at that point could not have been logistically achieved. Dr Barnett ordered a transfusion of 30mls of packed cells over 2 hours with lasix which is a diuretic. He telephoned the WCH transfusion service to find out how long it would take for cross-matched blood to be transported to Ashford. Dr Barnett also completed a written transfusion request in respect of the supply of 30mls by way of a neonatal quad pack. The written request[42] refers to the need for a transfusion 'stat'. I have already referred to the fact that 'stat' signifies a need for urgency, although Dr Barnett was of the view that the transfusion was not urgent in the sense that it needed to take place immediately. The transfusion would involve the administration of 30mls of packed cells and it would also involve the need to obtain

[41] Transcript, page 358
[42] Exhibit C3a, page 42

cross-matched blood having regard to the child's blood group. An alternative would have been to have simply administered blood group O negative there and then which is a measure that, it is said, will meet a need for a transfusion in an emergency. As well, there are certain difficulties associated with its administration including the need to warm the blood. There are also risks occasioned by the fact that O negative status may not accord with the blood grouping of the recipient. The administration of O negative blood was not indicated in the immediate circumstances that prevailed. It came to be administered after Jessica collapsed and the reason for that was that the cross-matched blood that Dr Barnett ordered did not arrive in time. In the event, I find that Dr Barnett was genuinely satisfied that it was appropriate to wait for the delivery of cross-matched packed cells which from his telephone conversation with WCH he noted would be 'available within 1 hour'[43]. Dr Barnett made the verbal request on the telephone at approximately 2:30pm. Dr Barnett told me that he believed it was implicit in the fact that he had telephoned as well as had transmitted a written request for blood that he did not want the whole process '*to take 3 hours*'[44]. He wanted to know how long it was going to take. He did not want the process to be slow but at the same time he did not believe the need for blood to be '*screamingly urgent*'[45]. He told me that he estimated that the urgency was situated somewhere in the middle between the two extremes and he was reassured when they told him that the blood would be matchable and deliverable within an hour.

6.23. There are two other matters that I should mention that are relevant to the question of the child's transfusion. Firstly, the proposed transfusion was to be by way of packed cells which refers to haemoglobin loaded blood with the other parts removed such as the plasma. The haemoglobin and the red cells are the elements of blood that carry oxygen to the tissues. The blood transfusion would not in a significant way replace or enhance clotting factors within the circulation. If a need for clotting factors to be administered arose, as it ultimately did, this would be achieved by way of a transfusion of fresh frozen plasma, separate from the transfusion of red blood cells. Dr Barnett did not at this early stage contemplate the administration of fresh frozen plasma. In the event, fresh frozen plasma came to be administered having regard to the obvious development of a coagulopathy in the child after her collapse. The other matter that was drawn to my attention was that with neonates particularly, there was a

[43] Exhibit C3a, page 16
[44] Transcript, page 360
[45] Transcript, page 361

system whereby a blood pack could be divided into four sections (a quad pack). The individual packs are smaller than might be required for an adult transfusion. The quad pack configuration is designed to avoid wastage in the context of a neo-natal transfusion and it also accommodates the potential to give more blood to a neonate beyond that originally prescribed. Dr Barnett believed that the most appropriate source for the blood would be the WCH as opposed to the services embedded in Ashford such as Clinpath and Gribbles. Dr Barnett also believed that Clinpath and Gribbles would not have been able to supply quad packs but would have supplied bags of approximately 500mls which was much more than what he believed Jessica required in the first instance. He also believed the WCH would have had a greater familiarity with the process that was required. In addition, Dr Barnett believed that the turn around time would have been very similar irrespective of whether the blood was supplied by WCH or by the onsite services. In any event Dr Barnett told me that he was reassured in Jessica's case that the blood would be there within an hour[46].

6.24. In the event, the blood had not arrived by the time Dr Barnett came to leave the hospital at approximately 4:00pm and had not arrived by the time of Jessica's acute collapse that took place shortly after that time at about 4:15pm. There was no attempt made to chase up WCH for the blood which did not in fact arrive until sometime after Dr Barnett returned to the hospital at about 4:25pm, having been called back after Jessica's collapse. We know this because when Dr Barnett arrived back at the hospital the child was given an urgent transfusion of O negative blood. The giving of O negative blood is explicable on no basis other than that the cross-matched blood had not yet arrived and that there was an absence of any reasonable expectation that it would turn up imminently. I am uncertain of the exact time it arrived, but it had been ordered at about 2:30pm, an indication had been given that it would be available within the hour and it had not arrived by the time Dr Barnett left the Ashford at 4:00pm nor by 4:15 pm when Jessica collapsed and nor by 4:25pm when Dr Barnett returned to the Ashford. This state of affairs meant that Jessica did not receive her 'stat' transfusion of cross-matched blood, or for that matter any blood at all, at a time at which it might have had some meaningful therapeutic benefit.

6.25. As indicated earlier, Dr Barnett was of the view that following the administration of normal saline Jessica had achieved a level of haemodynamic stability. This

[46] Transcript, page 523

impression was to dictate his course of management regarding Jessica and indeed his own movements during the afternoon. Jessica was monitored by the nursing staff from birth and the recorded heart rate and respiratory rate in respect of Jessica were within normal limits between the first recorded time of 1327 hours (which is four minutes after her delivery) and 1530 hours. The oxygen saturation rates at and after 1355 hours are satisfactory and at 1530 hours the oxygen saturation rate was at 100%. Dr Barnett attended to some other duties while he was at Ashford that afternoon but looked in on Jessica regularly. Dr Barnett left the hospital at 4:00pm which was about 15 minutes before Jessica's collapse. Dr Barnett believed that Jessica had maintained an appearance of haemodynamic stability until the time that he left. Asked by counsel assisting as to why he left at 4:00pm he said:

> 'Well, when I assessed her I came back to the nursery to assess her at 4 o'clock and she looked significantly better. She looked less pale, her heart rate was normal, her saturations were 100 and I made the decision that she was haemodynamically stable and that it was appropriate to leave.' [47]

As I understood Dr Barnett's evidence he would have expected the blood to have ultimately arrived and have been administered without incident. The acute deterioration that Jessica experienced not long after he left the hospital was, to him, unexpected and he told me quite genuinely that he would not have left the hospital if he had foreseen the possibility that Jessica would have rapidly deteriorated[48].

6.26. Jessica's collapse at approximately 4:15pm was marked by a dramatic descent in her heart rate, her oxygen saturation levels and her clinical wellbeing. When Jessica collapsed, measures were undertaken to improve her wellbeing. It is clear that clinically she had deteriorated quite markedly. I do not need to go into the details of that, nor of the details of the measures that were adopted in an attempt to reverse the deterioration that had taken place. It was clear that Jessica had developed a coagulation difficulty as evidenced among other things by the fact that, upon Dr Barnett's examination, sites of puncture marks were now bleeding. Jessica ultimately passed away that evening.

6.27. In this Inquest I examined at considerable length, and in considerable detail, a number of contentions to the broad effect that Dr Barnett's care of Jessica was less than optimal. One such contention involved an alleged failure on Dr Barnett's part to

[47] Transcript, page 545
[48] Transcript, page 550

monitor Jessica's blood pressure and other signs that might have signified her deterioration from the effects of a clearly diagnosed subgaleal haemorrhage of some severity. There is the suggestion that Dr Barnett should have monitored more closely and on an ongoing basis signs such as Jessica's haemoglobin levels and acidosis, rather than relying on a clinical assessment of haemodynamic stability. As well, the suggestion is made that Dr Barnett's leaving the hospital without having been satisfied that proper arrangements were in place to administer the necessary blood transfusion in accordance with his own already established timeline was not appropriate in the circumstances. An associated question of course is whether Jessica's death could have been prevented by more timely and vigorous intervention than was delivered at Ashford. I deal with those issues now.

6.28. The evidence of Dr Neil Roy concerning the neo-natal management of Jessica Stemmer

Dr Roy is an experienced neonatal paediatrician. He was a consultant neonatal paediatrician at the Melbourne Royal Children's Hospital (1975-1977) and the Royal Women's Hospital (1975-2007). During his tenure at the Royal Women's Hospital he was Director (Medical) of Neonatal Services from 1995 to 2007. Dr Roy is now Emeritus Consultant Paediatrician at the Royal Women's Hospital in Melbourne. Dr Roy was the inaugural Director of the Victoria's Newborn Emergency Transport Service (NETS) from 1976 to 1999. Dr Roy was not involved in the delivery of either Jessica Stemmer or Thomas Mahar. He is an expert in the field of neonatal paediatrics. Dr Roy examined the circumstances surrounding the births and deaths of both Jessica Stemmer or Thomas Mahar. In this section I deal with his analysis concerning Jessica Stemmer. Dr Roy prepared a report for the Coroner in relation to Jessica Stemmer[49]. He also gave oral evidence in the Inquest.

6.29. In his report concerning Jessica, Dr Roy outlines the course of Jessica's neonatal management. He referred to two striking changes between the original cord gas analysis and that taken at around 1 hour of age. In his view the acidosis that developed in the first hour of Jessica's life was severe and was, for the most part, due to a metabolic component. Dr Roy also viewed this change as having been a very rapid one. The other notable feature of the blood gas comparison was the fall in haemoglobin from 18.1 to 11.2 which represented a fall of over one third of the

[49] Exhibit C8

baby's red cell mass. He viewed the haemoglobin level of 11.2 as not necessarily being life-threatening in itself, but the difficulty with the haemoglobin level was the rapidity of the fall of one third of the baby's red cell mass in approximately an hour or less. These results suggested that Jessica's blood loss was very rapid and the accompanying acidosis suggested strongly that there was poor peripheral tissue perfusion as a result. The one other distinctly relevant matter for Dr Roy was the fact that there was a clear clinical explanation for all of this in the form of the subgaleal haemorrhage. In Dr Roy's view where there was evidence of rapid bleeding in the first hour of the child's life, an urgent blood transfusion was required. The view expressed in his report was that Dr Barnett had incorrectly interpreted the blood gas and haemoglobin level results and not appreciated just how rapid the loss of the blood into the subgaleal space had been.

6.30. Dr Roy was convinced that Jessica had died as a result of the complications of the subgaleal haemorrhage. In essence, in his view the nursery observations of a normal heart rate were misleading. I took Dr Roy to mean by that observation that they had been falsely reassuring. In his view blood pressure monitoring should have taken place. While the initial management by fluid resuscitation and the ordering of blood for the purposes of a transfusion was correct, it was insufficient in the circumstances in order to correct the complications in Jessica's presentation.

6.31. In his evidence Dr Roy pointed out that his views had to be examined in the light of the fact that he himself had very limited exposure to Level 2 clinical care such as that provided by the Ashford. However, he believed that he possessed a reasonable perspective in respect of such care because of his experience over the years as Director of the Victorian NETS. Nevertheless, in expressing his views he kept in mind that he has always dealt with the very worst cases in intensive care settings. That said, he acknowledged that severe subgaleal haemorrhage, at least as far as the Victorian experience was concerned, is relatively rare.

6.32. Dr Roy expanded on some of his opinions in respect of the management of Jessica's subgaleal haemorrhage. During his evidence in chief it became apparent that he held the belief that the baby's heart rate that had been recorded for a period of time prior to her collapse and which was within the normal range, had at one point risen to a level of 160 which Dr Roy regarded as of concern. The evidence as to the point in time at which that heart rate was identified suggests that it was not reflective of a rise after

the normal readings had been recorded by nursing staff, but had been a level that had been determined at one point by Dr Barnett and had been regarded by him as normal and not of concern. To my mind the evidence was not clear as to when exactly this rate had been identified and what its significance was. I have decided to ignore the evidence concerning the heart rate of 160. Suffice it to say, it was in any event not a heart rate that was unduly tachycardic if at all and one which was in any event not sustained. The evidence is clear enough that following Dr Barnett's administration of normal saline, the nursing observations of heart rate had remained within the normal spectrum until the child's eventual collapse at approximately 4:15pm.

6.33. Dr Roy's assertions that the child's blood pressure should have been monitored in the light of its diagnosed bleed, and preferably administered by way of intravascular monitoring, could also be endlessly debated, particularly in the context of a Level 2 nursery such as that at Ashford. In the event I did not find the debate about that issue to be particularly helpful having regard to the fact that such monitoring did not appear to be available at Ashford in any event. However, irrespective of whether or not invasive blood pressure monitoring was indicated or feasible in this particular case, the Court would simply make the observation that the evidence is reasonably clear that in cases of severe subgaleal haemorrhage as this was, particularly where there is evidence of clinical deterioration as a result of it, the availability of invasive blood pressure monitoring would be highly desirable. This at any rate is the clear view of Dr Roy. In his report he strongly expresses the view that there is a need for blood pressure monitoring whenever bleeding is suspected. Dr Roy did acknowledge that in a Level 2 hospital like Ashford, blood pressure monitoring would relatively rarely be required and that it was expensive. He believed it would probably only have significant utility where a baby required retrieval as intra-arterial blood pressure monitoring was beyond the scope of nursing staff in Level 2 hospitals in any case. While a paediatrician could set that up, Dr Roy believed that it was more the role of a retrieval team to perform such a task. I note that in the Royal Australian and New Zealand College of Obstetricians and Gynaecologists' new College Statement of July 2009[50] that blood pressure monitoring is absent from those suggested management strategies involved in both Level 2 or Level 3 neonatal surveillance. Dr Roy would suggest that although there appears to be some debate about the need for blood

[50] Exhibit C7f

pressure monitoring in these circumstances, his view is firm that it should be instituted[51].

6.34. Dr Roy expressed the opinion that it was sometimes extraordinarily difficult to visualise actual changes in a subgaleal haemorrhage and that even substantial bleeding can continue to increase the size with little immediate apparent change. In his view for there to have been a subgaleal haemorrhage of a moderate size that was identifiable on first examination, there must have been a reasonably fast bleed from the time of birth. It will be remembered that Dr Barnett himself noted the size of the subgaleal haemorrhage as moderate when he first examined Jessica. There was some considerable debate in the evidence as to whether on the one hand notwithstanding an appearance of haemodynamic stability Jessica had continued to bleed into the subgaleal space to a point where she inevitably collapsed, or whether she had re-bled acutely causing that collapse. Dr Roy favoured the view that the vast majority of subgaleal haemorrhages behave by way of a slow leak of blood into the subgaleal space which at some stage may or may not stop itself. He believed that is what happened here. He said that at the worst end of the spectrum one might encounter babies who have a large subgaleal haemorrhage from very early on in which the bleeding is profuse and which consumes coagulation factors at a huge rate[52]. As to whether in Jessica's case her presentation and deterioration could be consistent with an initial bleed stabilisation and then a further sudden bleed, he conceded that this was possible, but that a subgaleal haemorrhage was more usually characterised by continued bleeding with compensatory changes to the baby's circulatory system which eventually become so compromised that the baby is no longer able to maintain her circulation integrity[53]. Dr Roy did not believe the behaviour of a subgaleal haemorrhage to be one where the bleeding stopped and then re-bleeding recurred. I add here that Dr Barnett himself favoured the view that the apparent haemodynamic stability indicated that it was more likely that the baby had stopped bleeding and that the deterioration was reflective of a re-bleed. This view was shared by a professional colleague, Associate Professor Ross Haslam, whose evidence I will come to in a moment. As to this scenario, Dr Roy expressed the view that the favourable signs such as might lead a clinician to an impression of haemodynamic stability such as satisfactory oxygenation and heart rate might be falsely reassuring. He said that an impression of haemodynamic stability with heart rate levels being satisfactory, and

[51] Transcript, pages 846-846
[52] Transcript, page 894
[53] Transcript, page 867

perfusion also appearing to be satisfactory, did not necessarily mean that the child would stay that way in the face of a clear source of blood loss and evidence that there had already been significant blood loss and evidence that something had caused a deterioration in the metabolic status of the baby as evidenced by the concerning acidosis[54]. Dr Roy, on more than one occasion, pointed out that in all of this the acidosis which had a strong metabolic component had not at any time resolved. I would also add to that it was never again checked before the collapse. There was also the fact that in Dr Roy's view the significant descent in the haemoglobin level from 18 to 11 had not been rectified at the time of Jessica's collapse. There was also a question about whether the potential for the child developing a coagulopathy had been properly taken into consideration. For Dr Roy the appearance of normality in the child following the saline infusion administered by Dr Barnett was misleading in the sense that although appearances were normal, they may simply have been covering the problem[55]. Dr Roy again stressed the view that the child may continue to bleed to a point where all of the coagulation factors had been consumed and that is when coagulopathy can occur and appear very rapidly. In the event it was established that Jessica had developed a coagulopathy and indeed this was ultimately responsible for her death.

6.35. Dr Roy did agree that, as far as the urgency for any planned blood transfusion was concerned, the plan not to administer an absolute immediate transfusion was reasonable[56]. Nevertheless, in the light of the haemoglobin drop and the change to a severe acidosis, in Dr Roy's view there was a sense of urgency required here. He was not suggesting that the transfusion had to be administered right there and right at that time, '*but there is a sense of urgency that something is rapidly changing unless that blood gas is wrong*'[57].

6.36. As to the appropriateness of Dr Barnett waiting for the blood to arrive before a cross-matched transfusion could be administered, Dr Roy was asked by Dr Barnett's counsel, Mr Evans QC, whether in all of the circumstances that included firstly an indication that blood would be available in an hour, secondly continued monitoring, thirdly an assessment of clinical improvement in the child and finally an assessment

[54] Transcript, page 862
[55] Transcript, page 870
[56] Transcript, page 863
[57] Transcript, page 863

of haemodynamic stability, it was reasonable to wait for the blood to arrive. To this Dr Roy said:

> 'Only with the proviso that we have made two observations, one of which I think you've covered, and that is that we have the appearance, early, of a moderate sized subgaleal haemorrhage, so we have flagged that this is a high risk situation which may stop of its own accord, but which is in danger of further deterioration. The second one which is unresolved is that we have a severe acidosis which remains unresolved and, until that is resolved, I'm not happy that this baby really is stable.' [58]

Dr Roy also reiterated his view that the blood gas should probably have been repeated within 1 to 2 hours and that one would desire to administer that further blood gas test before the practitioner left the baby. Dr Roy also said that having identified a deterioration in blood gas pH from 7.3 to 7.08, which is significantly below normal, he would have been very concerned about that and believed that one should not leave the child until it was established that the blood gas was '*better again*'[59]. In addition, before one left the baby, one would want to be '*certain that the blood was about to commence*', being a reference to the planned blood transfusion[60]. He also said that one might have considered administering O negative blood by way of transfusion without the need for cross-matched blood, but that if Dr Barnett thought that the cross-matched blood would arrive '*at any minute*' then he would understand him waiting for that[61]. That observation of course has to be examined in the light of the fact that Dr Barnett had an expectation that the blood would be available within an hour of him ordering it. Clearly that proved not to be the case and no alternative plan for transfusion within that timeframe was made.

6.37. Dr Roy expressed the belief that in hindsight Jessica had been retrievable. He said:

> 'I believe that Jessica, in hindsight, was retrievable, yes. If we had known that she was going to continue to bleed and continue to deteriorate, then if one intervenes extremely early in a subgaleal haemorrhage, and in Jessica's case if one had done that straightaway, I think the chances are she would have survived.' [62]

Other views about this were expressed and I will come to those in a moment. I take into account that Dr Roy's opinion is premised on the need for very early intervention.

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[58] Transcript, page 879
[59] Transcript, page 810
[60] Transcript, page 811
[61] Transcript, page 811
[62] Transcript, page 816

6.38. The evidence of Associate Professor Ross Haslam

Associate Professor Haslam is a neonatologist at the WCH. He is the Head of Neonatal Medicine at that hospital, having been so since 1979. He is also the former Coordinator of the South Australian Neonatal Emergency Transport Service and is Clinical Senior Lecturer, Coordinator of Neonatal Program. Associate Professor Haslam is on the Paediatric and Neonatal Advisory Committee for the Medstar Service.

6.39. Associate Professor Haslam was concerned in the treatment of Thomas Mahar but was not in any way involved in Jessica Stemmer's management. However, he was asked to express his opinions about certain aspects of Jessica's management and I naturally regarded him as an expert witness for these purposes. Associate Professor Haslam had an opportunity to examine the clinical record relating to Jessica.

6.40. Associate Professor Haslam regarded the heart rate initially recorded by Dr Barnett at 160 as being a little high although within normal range. He naturally viewed the drop in haemoglobin from 18 to 11 as necessitating a transfusion, particularly so if there was clinical evidence of blood loss, which there was in the form of a subgaleal haemorrhage. The question would then become how quickly that should be given and whether un-cross-matched blood, which would be cold, should be given. If the baby was not haemodynamically stable, Associate Professor Haslam would administer un-cross-matched blood (ie. O negative). However, the correct approach would be to arrange for properly cross-matched blood in the shortest time possible, and the shortest time possible is an hour.

6.41. Associate Professor Haslam believed it would be important to closely watch a baby clinically in these circumstances with a '*senior reliable observer*'[63] and also by way of actual monitoring to ensure that the child remained haemodynamically stable while waiting for the blood. Associate Professor Haslam believed that as far as observations were concerned, if one had the ability to accurately measure blood pressure one would do so, but one would also rely on clinical experience by watching the child to see that he or she was breathing comfortably, that its colour was good and that blood was flowing through its veins and capillaries. Associate Professor Haslam regarded it as an ideal for an arterial line to be administered to continuously monitor blood pressure but recognised that such was only available in an Intensive Care Unit which the

[63] Transcript, page 749

Ashford did not have. Other methods of measuring blood pressure were notoriously inaccurate and could be misleading.

6.42. Associate Professor Haslam believed that what had happened in Jessica's case was that she had suddenly re-bled and this had accounted for her collapse. He thought it unlikely for a mature baby in whom there was a slowly accumulating haemorrhage with hypovolaemic hypotension to have suddenly reached a point where it collapsed. In other words, he did not believe the theory that Jessica had continued to bleed. He pointed to the readings that included those of the heart rate that had been measured from just after birth to approximately 3:30pm as being perfectly normal, as were the respiratory rate and oxygen saturations. He believed that the readings were those of a baby who was quite stable[64]. Associate Professor Haslam did not believe that giving an urgent O negative blood transfusion, as opposed to waiting for the cross-matched blood to arrive, had necessarily been indicated even after the cross-matched blood had not arrived within the anticipated hour[65]. That said, Associate Professor Haslam agreed that if he had been in Dr Barnett's position, one thing that he may have done differently would have been to make a call at around about 2 hours of age to chase up the cross-matched blood that had been ordered. I observe that even that did not happen in this case. Associate Professor Haslam also believed that he likely would have performed another arterial blood gas. He said:

> 'I personally would want that comfort of confirmation of which direction things are going. I would have expected given the clinical picture that has been described that metabolic acidosis would have been improved and the haemoglobin would have been about the same. That is what I would have expected to have seen but certainly no worse and, yes, I think it is likely I would have wanted to look at that again if that was possible.' [66]

6.43. In the event we know that the cross-matched blood did not arrive until sometime after approximately 4:30pm because it was at about that time that Dr Barnett returned to the Ashford and instead of giving cross-matched blood, he administered O negative blood sourced from that hospital. Although Associate Professor Haslam resisted the idea that O negative should have been given after the expiration of the anticipated 1 hour, he acknowledged that he would have had a dilemma on his hands if he was told that the blood was another hour away from that point but suggested that on the other hand, the dilemma was ameliorated by the fact that the baby at that particular point in

[64] Transcript, page 755
[65] Transcript, page 757
[66] Transcript, page 770

time was still stable. In any case he himself would have waited at the hospital for the cross-matched blood to arrive. Associate Professor Haslam agreed with the proposition that, as a clinician, one would stay to ensure that the blood had arrived or if it had not, that one was still there and available to make a decision as to what to do next. He said that there was a case for staying clinically in touch and to strongly consider expediting blood of some kind.

6.44. Associate Professor Haslam did not wholly agree with the proposition that Jessica's survival would have been highly likely if alternative and more timely action had been taken. However, he thought it was fair to say that it was possible that Jessica may have survived. In this context, Associate Professor Haslam spoke of the complicating effects of co-morbidities such as asphyxia or intracranial haemorrhage, but I observe that none of those considerations applied in the case of Jessica, although it did with Thomas Mahar. Nevertheless, the literature in his view made it plain that even with aggressive blood replacement and optimal intensive care the mortality of a severe subgaleal haemorrhage is still significant. Where there is an inability to control the disseminated intravascular coagulopathy that is especially so. I note, however, that the evidence of uncontrollable coagulopathy only emerged after Jessica's acute collapse. Associate Professor Haslam gave this evidence in response to a question from counsel assisting:

> 'Q. And I think you have already answered this question, but if baby Jessica have been given the transfusion within the hour of it being ordered, as expected, or 10 or so minutes after, so around 1530 to 1545 hours, it's your evidence that the outcome may have been different.
>
> A. It may have been different, yes. I certainly couldn't say with absolute certainty that it would have, but it may have been.' [67]

6.45. <u>The evidence of Dr Christopher Barnett</u>

I do not need to recite every detail of Dr Barnett's own analysis of Jessica's management as the basic factual matters are not in dispute. He dealt with the criticisms of his management. Dr Barnett recognised that the child's arterial cord blood gas at birth was markedly more favourable than the arterial blood gas results that he obtained an hour after the child's delivery. In particular the comparison indicated to him that there was had been the development of a metabolic acidosis and a haemoglobin decline which indicated that there was a source of blood loss. He

[67] Transcript, page 774

naturally also recognised the existence of what he described as a moderate subgaleal haemorrhage upon his first examination. Dr Barnett told me in evidence that he believed the haemoglobin value of 11.2 was not life-threatening[68]. He said that although the level indicated anaemia, it was not a level that dictated that one must do something about it urgently[69]. This meant that in his view, and given the clinical improvement upon the delivery of a normal saline infusion, that although it was appropriate to transfuse by giving blood, there was no need to do so urgently, meaning '*not in the next 5 to 10 minutes*'[70]. I do observe, however, that in the written request he used the expression 'stat'[71] which does rather suggest that it was within his contemplation that the transfusion would occur without undue delay. Dr Barnett used the word 'reassured' in respect of his state of mind when he was told that the blood that was required was matchable and deliverable within an hour[72].

6.46. Dr Barnett also acknowledged that the administration of a blood transfusion by way of packed red cells was not designed to rectify any coagulopathy that might exist[73]. What would be required in those circumstances would be a transfusion of fresh frozen plasma that also would have needed to have been cross-matched and obtained from the transfusion service. He did not order any such plasma.

[68] Transcript, page 351
[69] Transcript, page 351
[70] Transcript, page 359
[71] Exhibit C5a, page 42
[72] Transcript, page 361
[73] Transcript, page 373

6.47. In answer to the suggestion that at the time he left the hospital at approximately 4pm he should have upgraded his request for the blood transfusion to an urgent request, he said:

> 'No. The situation is you know the wheels are in motion so probably the blood is coming. There is nothing you can really do to get it there faster. The process is happening so -' [74]

6.48. Dr Barnett was of the view that the child's sudden deterioration was probably accounted for by a re-bleed. The circumstances indicated an acute event of some sort had taken place, given the previously stable presentation and the sudden drop in the child's parameters such as oxygen saturation. When asked as to whether the baby's erstwhile stability had meant in his view that the bleeding had stopped or had slowed down, he said:

> 'I don't think you can be sure that it's stopped, there's no way of knowing that, but certainly, being haemodynamically stable means that the baby is coping and the assumption there is that it has slowed down or stopped, because the baby is telling me from heart rate and appearance that it's coping.' [75]

6.49. In answer to the suggestion that blood pressure monitoring should have been administered prior to the baby's collapse, he suggested that the means by which that would have been taken via a cuff would have been a poor substitute for central line blood pressure monitoring and that, in any event, one assesses a baby using other parameters.

6.50. As to the suggestion that the impression of haemodynamic stability engendered by a normal heart rate was misleading, Dr Barnett suggested that one would only view it as such in the light of the known outcome, but at the time of these events the heart rate had been telling him that the baby was haemodynamically stable.

6.51. Dr Barnett also rejected the suggestion that he had not had sufficient regard to the rapidity of the fall in haemoglobin. He believed that he had adequately taken that into consideration[76]. Dr Barnett rejected the suggestion that he had not fully taken into account the child's accompanying acidosis which had indicated poor peripheral tissue perfusion. He said as follows:

> 'Yes, I don't think it's correct. I think that it's not that it wasn't taken into consideration, it's that I am standing there looking at the baby with a normal heart rate and a normal

[74] Transcript, page 377
[75] Transcript, page 391
[76] Transcript, page 470

> saturation and improved after the bolus of fluid. I made a conscious decision that the fallen haemoglobin had occurred but that the baby was telling me that the baby had coped with that fallen haemoglobin and that Jessica needed to be given blood but with a normal heart rate, a normal saturation and improved perfusion to look at, that providing O negative blood to the baby didn't seem appropriate at all at the time, and potentially not without risk.' [77]

6.52. As to Dr Roy's suggestion that in a more slowly accumulating subgaleal haemorrhage it might be appropriate to await routine cross-matching of blood for transfusion, but that where there was evidence of rapid bleeding an urgent blood transfusion is required, Dr Barnett argued that even with the bleeding that had taken place, Jessica appeared to have adequately coped with it cardiovascularly and had done so for a period of hours before she had an acute collapse. Dr Barnett argued that having regard to a haemoglobin level of 11 which was not in and of itself indicative of extreme urgency, and taking into account the normal heart rate and an improved tissue perfusion, his waiting for the blood had been appropriate[78]. Dr Barnett went on to say that in his view the unpredictability of what happened to Jessica was rare.

6.53. In answer to Ms Kereru, counsel assisting, Dr Barnett agreed that if he had conducted further tests before he left the Ashford, this may possibly have given him a better picture of her presentation, but he stated that when babies acutely deteriorate, as had Jessica, the test results could be absolutely normal minutes before that sudden deterioration and therefore the result may not have been helpful. In other words, they may have been no reliable predictor of what was tragically to take place.

6.54. In answer to Ms Kereru, counsel assisting, Dr Barnett suggested that if it had been his assessment that the child looked as if she was about to experience an imminent collapse he would have certainly telephoned again to find out where the blood was but that had not been his assessment.

6.55. As to the fact that he had conducted no test for coagulation factors, Dr Barnett suggested that this could have been done, but the child was looking better and, in any event, he posed the question as to how testing for coagulation factors may have altered the outcome.

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[77] Transcript, pages 471-472
[78] Transcript, page 474

6.56. When questioned about why he chose to leave the hospital, Dr Barnett pointed out that he had not been away from the hospital for very long before Jessica collapsed. It was about 15 minutes. The point can be made, however, that the question is not so much why Dr Barnett left the hospital or whether he should have, the question really is why, prior to leaving the hospital, he had not followed through in respect of a transfusion that he had himself had requested and organised.

6.57. Conclusions relative to the circumstances of the death of Jessica Stemmer

Jessica's cause of death was disseminated intravascular coagulation following haemorrhage. The haemorrhage was a subgaleal haemorrhage was caused by the force applied to her scalp in the course of a ventouse delivery.

6.58. As to the circumstances of her deterioration and death, there is no dispute that Dr Barnett's management of Jessica was appropriate up to a point. There is an undeniable fact, however, that Jessica did not receive a cross matched blood transfusion or, for that matter, any blood transfusion at all prior to her collapse at 4:15pm. Dr Barnett had clearly considered that a cross matched transfusion was necessary in the short term, albeit not immediately. Dr Barnett had ordered the cross matched blood from the WCH at approximately 2:30pm and did so in the light of the blood gas results that had indicated a significant drop in Jessica's haemoglobin level. He had taken the trouble not only to forward to the WCH written documentation requesting the delivery of 30ml of cross matched blood for the purposes of it being transfused 'stat', but had telephoned the relevant service and had sought and been given an assurance that the blood would be available within 1 hour, which assurance he noted in the clinical record. I can only infer that Dr Barnett did make that phone call because he wanted to be, to use his own terms, 'reassured' that the blood would be available in the timeframe within which he considered it necessary for the transfusion to be administered. There is no evidence to explain why it was that the cross matched blood was not made available within 1 hour, nor indeed is there evidence to explain why it was not made available within 2 hours and more, given that it had not arrived by 4:25pm when Dr Barnett decided to administer the O negative blood after all. Equally unexplained in a satisfactory way in my opinion is the failure of Dr Barnett to make any enquiry as to the whereabouts of the blood when it had not been made available within 1 hour and had not been made available by the time he left the Ashford at 4:00pm which was 1.5 hours after he had made the request

for the blood. It was not as if the need to transfuse the child had in any way been ameliorated or had disappeared. Nor was there any reasonable basis to conclude that the blood transfusion could legitimately be postponed to a later point in time, a point in time which owing to the failure of the blood to arrive was becoming increasingly uncertain. Dr Barnett's explanation for not chasing up the blood or upgrading his request to an urgent one to my mind bore an air of resignation on his part that such action would have been futile. I here refer to his explanation that he knew the wheels were in motion so that the blood was probably coming and that there was nothing he could really do to get in there any faster. To him '*the process was happening*'. At 3:30pm when the blood had not arrived within the hour he did not make any further enquiries as to where it was. He said that given his personal experience with the blood bank and their excellent record he assumed that it would be arriving '*any minute now*'[79]. It will be noted that Dr Barnett remained within the precincts of the hospital for a further half an hour before he left so his expectation that the blood might arrive any minute now had not been realised at 4:00pm, the time that he left. When asked whether prior to his departure he should have called the hospital to enquire as to the whereabouts of the blood, Dr Barnett did agree that it was something that he could have done but that when he returned to examine Jessica he was still reassured by her improved appearance and felt that the blood would be coming[80]. He specifically said that he was reassured by the oxygen saturations of 100% and the normal heart rate.

6.59. I note that Associate Professor Haslam said in his evidence that the one thing that he may have done differently if he had been in Dr Barnett's position would have been to have made a call when the child was around 2 hours of age to chase up the cross matched blood. This would have been at a time around 3:30pm which was in fact an hour after the blood had been ordered and the time within which it had been suggested it would be available.

6.60. In my view it makes little sense that Dr Barnett would telephone the blood bank and seek and obtain assurances that the blood would be available within 1 hour, consider this to be a satisfactory arrangement, if not a clinically necessary one, and then not follow through with the request that was after all marked as 'stat' on the requesting

[79] Transcript, page 546
[80] Transcript, page 647

paperwork. It is even more perplexing that he would not have sought some further assurance about the blood's arrival before he left the hospital premises.

6.61. Dr Barnett's explanation for this behaviour is premised on two things. Firstly, there was his genuinely held belief that the child was haemodynamically stable as evidenced by satisfactory heart rate, respiratory rate and oxygen saturation levels and secondly, his genuine belief that all of this reflected a true picture of stability insofar as it indicated that there was no ongoing bleeding into the subgaleal space. This latter notion involves the debate as to whether or not Jessica's collapse can be explained by an inevitable deterioration following a continuous bleed or as a result of an acute episode of bleeding at the time of her collapse. The evidence differed about that and the only point that can be made is that there is insufficient evidence to positively establish which of the two processes was at work. The question is whether Dr Barnett's belief that the child was haemodynamically stable and therefore not in any imminent danger of rapid deterioration was a reasonable belief. In this regard, Dr Barnett was not merely reassured by the picture of haemodynamic stability. His actions are more in keeping with those of a man who was convinced to an absolute degree that there was no risk of such a deterioration and that the child's continued stability was virtually guaranteed. One questions whether such a state of mind could ever be reasonably entertained in a setting such as this, especially without further blood gas testing for haemoglobin and acidosis.

6.62. On the question of whether the child's apparent haemodynamic stability was in any sense reasonably to have been viewed as reassuring, it seems to me that I have to take into account Dr Roy's observation that if there was independent evidence that the bleeding had actually stopped, one might more legitimately take comfort from one's clinical observations. Such evidence would consist of repeated haemoglobin level tests and repeated blood gas tests to show that the baby's peripheral circulation had now recovered[81]. As well, one would need to look at the baby's level of coagulation factors. In this case there was no further haemoglobin or blood gas testing beyond that conducted by Dr Barnett sometime around 2:30pm which, in and of themselves, revealed a non-reassuring picture. As well, there were no coagulation studies undertaken at any point in time prior to the child's collapse.

[81] Transcript, page 814

6.63. In short, Dr Roy was of the view that relatively little could be inferred from a picture of haemodynamic stability, particularly having regard to the fact that other more scientific means of establishing the child's wellbeing would have been available, namely further haemoglobin and blood gas testing and the testing for coagulation factors.

6.64. To my mind there is considerable weight to be accorded to Dr Roy's views that in the absence of further testing, in essence it would not be reasonable to place significant reliance in outward signs of stability as being truly reflective of the child's wellbeing. The best evidence of that would have been further testing which was not undertaken. I take into account Associate Professor Haslam's views that given Jessica's clinical picture of stability, further testing would have been otiose as he would have expected it to have revealed no more than an improved metabolic acidosis and no change in the haemoglobin levels and that further testing would merely have given those treating Jessica an improved sense of comfort. I also take into account Dr Barnett's evidence that further testing might not have been helpful in a situation where an imminent acute collapse was about to occur as it may even at a late stage have revealed no more than a picture of normality and stability. There is an element of circularity involved in these arguments, however, because the presupposition that further tests in Jessica's case would have indicated normality is naturally based on the premise that Jessica's clinical picture was an accurate reflection of her actual wellbeing which is the very question at issue. The fact of the matter is that one will never know the answer to this question because no such further tests were conducted. What is known is that Jessica collapsed without any further intervention beyond the resuscitative measures that Dr Barnett initially put in place.

6.65. Jessica was never given the cross matched blood transfusion that Dr Barnett had contemplated and ordered. The child did need a transfusion and the fact that she did not receive it in a timely manner, a situation that in my view ought to have been avoided, is a serious matter. The evidence would suggest in this regard that had the child been given a transfusion at a time before her collapse at 4:15pm the chances of her survival would have been greater. Whether she would have survived or not in reality is a matter that cannot be known with any degree of certainty. Dr Roy was of the view that early intervention would have meant that Jessica was retrievable, even on the basis of his favoured theory that she had continued to bleed. Associate

Professor Haslam on the other hand was less confident about the chances of her survival had she been given the transfusion within the hour of it being ordered or, say, between 3:30pm and 3:45pm. But as seen earlier he suggested the outcome may have been different if the transfusion had been so given.

6.66. My conclusion is that before he left the Ashford at 4:00pm Dr Barnett genuinely believed that Jessica Stemmer had a picture of haemodynamic stability and that this meant in his eyes that there was no need to give O negative blood or that there was a need to urgently chase up the blood from the blood bank. I am of the view that in the absence of further testing in relation to acidosis and haemoglobin level administered before Jessica's collapse, these beliefs cannot be said to have been reasonably held. In my opinion Dr Barnett ought to have made further enquires about the whereabouts of the cross-matched blood and as to its time of arrival. However, I note in this regard that there is no evidence before me as to the reason for the delayed delivery and there is no evidence that repeated requests for the blood if made by Dr Barnett would necessarily have accelerated its delivery to Ashford. The evidence does not allow me to reach any conclusion as to those matters. Nevertheless I also take into account that if it had been established by repeated enquiry that further delay in the delivery of the blood would be experienced, consideration inevitably would have been given to administering O negative blood at a time before Jessica's collapse.

6.67. In my opinion it is open to conclude on a balance of probabilities that Jessica's chances of survival would have been better if she had received a blood transfusion at a time before her collapse and I so find.

7. Thomas William Mahar

7.1. The delivery of Thomas Mahar

The estimated due date of Thomas' birth had been 2 April 2007. When Mrs Mahar had not come into labour by 9 April 2007, plans were made to induce labour. She was therefore admitted to hospital and, following the performance of a CTG (cardiotocograph) which was normal, induction was commenced. At approximately 10am that day an artificial rupture of the membranes was performed. An intravenous drip was inserted and a Syntocinon infusion commenced at approximately 10:30am. For the most part, Mrs Mahar continued to be monitored by the CTG. CTG monitoring measures and records the rate and quality of the foetus' heartbeat.

7.2. Mrs Mahar's labour was described in some detail in the reports of Professor Roger Pepperell who examined the circumstances surrounding Thomas' birth. I do not need to go into the entirety of the detail described. However, the latter part of Mrs Mahar's labour was of some note. The circumstances of the delivery of Thomas were investigated having regard to two things. Firstly, when the child was delivered he was in a depressed state, had no tone and had made no initial respiratory effort. Following bag and mask ventilation with oxygen for one minute, there was still evidence of apnoea, and respiratory effort commenced only after about two minutes with no limb movements for 20 minutes. The test revealed that the child was significantly acidotic. His condition was in keeping with in-utero ischaemia prior to his delivery. Indeed, the indications during the course of his delivery were that he might be experiencing an hypoxic episode given non-reassuring CTG traces. Secondly, like Jessica, Thomas was diagnosed with a subgaleal haemorrhage following his delivery. Therefore, my inquiry concerned whether the hypoxia that Thomas experienced in-utero was identifiable and could have been prevented or minimised and, secondly, I examined the possible causes of the infliction of the subgaleal haemorrhage.

7.3. Dr Sandercock had been at home during the latter part of Mrs Mahar's labour which had proceeded into the evening. Mrs Mahar's labour had meanwhile been managed by the Ashford midwifery staff.

7.4. It is first necessary to refer to the evidence of Professor Pepperell in relation to his view of the relevance and significance of non-reassuring CTG traces that were identified during the course of the evening. According to Professor Pepperell, between 8:20pm and 8:30pm the CTG demonstrated that decelerations in the child's heart beat were becoming deeper and slightly broader and that in the light of that revelation Dr Sandercock should have been advised at the time. He also expresses the view that the Syntocinon infusion that had been administered as part of the induction process should have been turned off or turned down. In the event, Dr Sandercock was not to have been notified of any adverse developments until 9:10pm and the Syntocinon infusion would not be turned off until 9:20pm by which time the hypoxic picture had become even more concerning. I digress to observe that Dr Sandercock possessed a facsimile machine at her home and that it would have been possible for CTG traces to have been faxed to her for her interpretation. Professor Pepperell felt that an examination of the CTG traces as they existed between 8:20pm and 8:30pm

may well have dictated a decision on Dr Sandercock's part to have expedited the child's delivery. His own personal experience in these matters would have dictated him being notified in those circumstances at 8:30pm at the latest. I return to Dr Sandercock's own attitude to these contentions in a moment.

7.5. The CTG trace from 8:44pm to 9:10pm represented a worsening of the situation. In fact Professor Pepperell suggested that there was marked deterioration from 8:50pm.

7.6. Professor Pepperell was of the view that when Dr Sandercock was ultimately notified of these worrying developments by phone at 9:10pm, the urgency of the situation was not effectively conveyed to her as a result of which Dr Sandercock may not have proceeded to the Ashford with the necessary haste. In the event, Dr Sandercock did not arrive until about 9:45pm and the child's delivery was commenced 25 minutes after that. This further delay was partially explained by the need for Mrs Mahar's epidural to be topped up. However, much of the earlier delay could have been avoided in Professor Pepperell's opinion if a proper analysis had been made in respect of the non-reassuring aspects of the CTG prior to 8:30pm. In fact the delay was not only caused by a failure to notify Dr Sandercock appropriately by 8:30pm, but further delay was occasioned by the fact that a proper sense of urgency was not conveyed to her even when Dr Sandercock was notified of the situation at 9:10pm. Professor Pepperell also expressed the opinion that most midwives would have turned off the Syntocinon earlier than it was as the decelerations at an earlier time were very much of concern[82]. Professor Pepperell suggests that with an abnormal CTG it is routine for the Syntocinon infusion to be turned off or at least turned down, and only reinstituted when the CTG abnormality has been resolved[83].

7.7. Professor Pepperell conceded that with a CTG trace such as that of Thomas Mahar, the majority of neonates or foetuses would be delivered with no deficit. He also conceded that the worst abnormalities that might involve the likelihood of hypoxia, such as prolonged bradycardia, complete loss of short term variability or late decelerations occurring after contractions had not applied in this case. However, he added, importantly in my view:

> 'The problem is if you have this sort of pattern in clinical practice you don't know whether the baby is going to be adversely affected or not and in 20% it will have

[82] Transcript, page 953
[83] Transcript, pages 957-958

> evidence of hypoxia, in the others when it's born it will be in perfectly good condition and you will wonder why you expedited delivery, and that's the problem that the obstetrician has to cope with in terms of these sorts of abnormalities. And in general terms because we don't know, we expedite delivery, using either ventouse or forceps, where this pattern is evident.' [84]

In other words, Professor Pepperell was clearly of the opinion that one does not view the risk of hypoxia as an acceptable one simply because of the absence of the worst possible abnormalities that one may see. One expedites delivery regardless. We know in hindsight from the severe acidosis that was detected after the child had been delivered that Thomas had been experiencing an hypoxic episode prior to his delivery. Of course this outcome was not known with certainty at a time prior to delivery, but clearly there had been indications in Professor Pepperell's view that an hypoxic baby was on the cards.

7.8. Registered midwife Linda Lewis gave evidence before me. Ms Lewis told me that she had been observing the CTG trace and had noticed regular decelerations subsequent to 8:15pm, but noticed that they had a good return to the baseline. In her assessment, he variability was normal. At approximately 8:30pm she discussed the CTG trace with another midwife. Ms Lewis told me that she and the other midwife reached a joint conclusion that the CTG revealed a possible cord compression pattern and they formulated a plan to firstly to examine the patient to ascertain whether she was progressing in labour and then notify Dr Sandercock. Ms Lewis then carried out a vaginal examination and recorded in a note that she made at 8:40pm that the CTG showed decelerations occurring regularly with good return to the baseline and good variability in between. The vaginal examination was noted to have revealed blood and mucus and it also records that Dr Sandercock was informed of all of the above. In a retrospective note made by Ms Lewis the following day, she added to the observations that she had noted at 8:40pm the night before that at the time of her vaginal examination Mrs Mahar had been 9cm dilated. She records in this retrospective note that she had phoned Dr Sandercock at approximately 9pm. The impression that Ms Lewis' contemporaneous note and retrospective note in combination conveys is that she saw no real urgency and identified no significant concern at the time of her vaginal examination and telephone conversation with Dr Sandercock.

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[84] Transcript, pages 931-932

7.9. However, by 9:30pm Ms Lewis had become so concerned and anxious about the irregularity of the decelerations and their type that this prompted her to arrange for another midwife to call Dr Sandercock to ask her to attend immediately. As it happens, Dr Sandercock was already on her way to the hospital at that time. She arrived at about 9:45pm.

7.10. In cross-examination by Mr Lindsay on behalf of Dr Sandercock, Ms Lewis agreed that the note that she made timed at 8:40pm, which includes reference to her informing Dr Sandercock of the current situation, does not record any concern or sense of urgency on her part. That said, Ms Lewis said in answer to me that she had told Dr Sandercock that she was concerned about the fact that the decelerations on the CTG trace were becoming more prolonged and that Dr Sandercock had asked her about the nature of the decelerations and had then said that she would be on her way[85]. However, she also agreed in cross-examination that the concern and sense of urgency that she was experiencing was only reflected for the first time in a further note timed at 9:30pm where decelerations had now deepened to a level of 72 and were lasting longer[86]. She agreed that she did not think that it had been necessary to fax through the concerning CTG trace to Dr Sandercock at the time of her phone conversation because she had verbally described what she had believed to be the abnormalities. She agreed that she did not convey any sense or urgency as such, but conveyed a level of concern[87]. Ms Lewis agreed that she would have noted the fact if she had requested Dr Sandercock to actually attend the hospital when she had called her at what she had believed to have been about 9:00pm and agreed that the absence of any such note signified that she had not told Dr Sandercock that it was time for her to come to the hospital. Counsel assisting, Ms Kereru, asked Ms Lewis as to why at the time of that telephone conversation she did not ask Dr Sandercock to come in. In answer Ms Lewis said that her recollection was that in the conversation with Dr Sandercock, Dr Sandercock had asked her questions about the type of decelerations and also as to the nature of the variability between the decelerations. She said:

> 'In view of the fact that the woman was close to delivery - close to fully dilated Dr Sandercock informed me that she would be on her way very soon. I cannot remember in detail whether I expressed any concern verbally at that time.' [88]

[85] Transcript, page 219
[86] Transcript, page 220
[87] Transcript, page 221
[88] Transcript, page 235

7.11. Dr Sandercock's version of these events is as follows. By reference to her own pager records, she was able to establish that registered midwife Lewis had notified her of her concerns at 9:10pm, not 9:00pm as thought by Lewis. Later that evening Dr Sandercock made a handwritten note in the clinical record relating to Mrs Mahar[89]. Dr Sandercock told me that her recollection of the conversation, as supported by the note, was that Ms Lewis expressed concern about the CTG tracing of the baby from about 8:30pm, that Ms Lewis had conducted a vaginal examination 10 minutes prior to the conversation, that on that examination the patient had been found to be 9cm dilated and that Ms Lewis had felt that it was difficult to assess the position of the baby. Ms Lewis had reported to Dr Sandercock that the trace had a normal baseline and normal variability with decelerations down to a heart rate of 80. Dr Sandercock told me that her interpretation of that information was that the decelerations were explained by and were reflective of the rapidity of progress and the descent of the baby's head into the pelvis. On that basis no particular concern was ever conveyed to her about the decelerations[90]. In cross-examination by Ms Kereru, counsel assisting, Dr Sandercock was asked in essence what her attitude would have been had she been fully aware of, or had seen, say by way of fax to her home address, the CTG trace as it existed prior to the time that she was telephoned at 9:10pm. In answer Dr Sandercock said that the trace in the 20 minutes immediately prior to the phone call from Lewis, that is between 8:50pm and 9:10pm, certainly showed a broadening of the decelerations and a change in the baseline variability of the baby's heart rate and that this was something that she had not been made aware of. When asked what difference it would have made if she been apprised of this development by way of a faxed copy of the trace, Dr Sandercock stated that she would have attended the hospital more quickly[91]. As it happened she did not leave her home address immediately to travel to the Ashford which was only about 5 to 10 minutes away by car. She had advised Ms Lewis that she would be at the hospital in around half an hour, which would mean that she had delayed her departure from her home by approximately 20 minutes. In my view there is little doubt that Dr Sandercock would have left straight away if she had seen the CTG trace and had appreciated its significance at 9:10pm. It therefore appears that at least some of the delay, representing as it does a period of hypoxia, could have been avoided. This would

[89] Exhibit C4, page 42
[90] Transcript, page 267
[91] Transcript, page 318

mean that much of the more serious non-reassuring aspects of the CTG trace and hypoxia could have been avoided. That said, Professor Pepperell was of the clear view that Dr Sandercock should have been notified even earlier in respect of the non-reassuring CTG traces prior to 8:30pm.

7.12. Dr Sandercock also said that from 9:20pm specifically, which was 10 minutes after her telephone conversation with Ms Lewis, the trace over the following 15 minutes, which she was not to see until her arrival at the Ashford at about 9:45pm, was of increasing concern. The foetal heart rate decelerations were deeper to a lower heart rate of below 70, the periods of return to baseline were short and there was a decrease in variability, all of which indicated foetal distress. Dr Sandercock acknowledged that she was telephoned at approximately 9:30pm when she was enroute to the hospital about these worrying matters. When she arrived at the hospital she was very concerned about the CTG and decided to deliver the child as soon as was practicable by way of ventouse. It thus appears that much of this rather worrying period of abnormality in the trace specifically between 9:20pm and 9:45 pm when Dr Sandercock arrived, reflecting as it did hypoxia, could have been avoided if an appropriate level of concern had been conveyed to her in the phone call of 9:10pm and if as a result she had immediately come in to the Ashford.

7.13. As to the circumstances of Thomas' actual delivery, there is no question but that an expedited delivery was appropriate having regard to the non-reassuring aspects of the CTG trace. Dr Sandercock had her reasons for not conducting a caeserean section, which I do not need to go into, but there is no suggestion that they were unreasonable. There is also no suggestion that a delivery by way of ventouse extraction in particular was not appropriate. Professor Pepperell did not suggest otherwise.

7.14. According to registered midwife Lewis, the delivery was effected over no more than three contractions. It is accepted that there was a delay between the second and third applications of traction owing to the fact that Mrs Mahar did not experience another contraction. A pressure of 60 was applied. Ms Lewis said that Dr Sandercock at no stage appeared to struggle and that the appropriate amount of force was utilised. As far as she was concerned the baby's head was in the correct position for a delivery and that no twisting had been necessary. As far as Ms Lewis was concerned there was

nothing different about this ventouse delivery when compared to any other delivery that she had seen Dr Sandercock administer[92].

7.15. According to Dr Sandercock, the cap remained attached during the entire delivery process. The entire process took 8 minutes. The Mityvac cup was applied at 10:10pm and the baby was delivered at 10:18pm. Dr Sandercock spoke in her evidence of the delayed last contraction. She told me that the foetal heart rate at that point was quite low, in fact on 50, and which was very concerning so she asked Mrs Mahar to push without the contraction to enable her to deliver the baby sooner. Notwithstanding the lack of an actual contraction, Dr Sandercock told me that she did not experience pressure of any different magnitude[93]. Dr Sandercock did say, however, that there was some maternal effort, notwithstanding the absence of a contraction. Dr Sandercock rejected the proposition that the subgaleal haemorrhage sustained by Thomas may have occurred because of the non-existence of a contraction at the time of the third pull. She said that the head was well on the perineum and that she had cut an episiotomy and had asked Mrs Mahar to push. The traction applied with the device at that stage was minimal[94]. Dr Sandercock was not able to explain the cause of the spinal stretching injury that was identified at autopsy, but I have already referred to the fact that there does not appear to be any known association between that kind of injury and ventouse delivery. Dr Sandercock told me that she had not needed to rotate the child's head to effect delivery. In short, according to both Ms Lewis and Dr Sandercock no unusual amount or type of force had been used in order to effect this ventouse extraction. The injury will thus have to be regarded as unexplained.

7.16. Professor Pepperell in his evidence did not comment unfavourably on the fact that there had been no contraction at the time of the third pull. He believed it to have been an appropriate way to carry out a delivery that was urgent[95]. Professor Pepperell found no deficiency in the obstetric care as far as the child's delivery is concerned[96].

7.17. As with Jessica, there does not appear to be any available explanation as to why Thomas sustained a subgaleal haemorrhage. However, its ultimate severity and consequences have to be examined in the light of the fact that he had also suffered a

[92] Transcript, page 233
[93] Transcript, page 273
[94] Transcript, page 324
[95] Transcript, page 932
[96] Transcript, page 933

severe hypoxic episode during the course of his delivery and that this played a role in the subgaleal haemorrhage assuming more significant degree of severity.

7.18. There is no evidence that Dr Sandercock departed from any standard or usual operating procedure in the delivery of Thomas Mahar.

7.19. Thomas Mahar's post natal care

Dr Barnett attended at the Ashford at the very end stage of Thomas Mahar's delivery. He was responsible for Thomas' post natal care until such time as Thomas was retrieved to the WCH. I have already described Thomas' condition at birth from a clinical view point. Throughout the course of Dr Barnett's care prior to the retrieval, he performed a number of blood gas tests. They included a blood gas taken immediately after birth from the umbilical cord artery. A number of other blood gas tests were conducted.

7.20. To begin with, Dr Roy, who also examined the appropriateness of Thomas' post natal care, was critical of a lack of recognition of the severity of blood loss and the lack of any urgency in conducting a blood transfusion in relation to Thomas at the Ashford. It will be observed that this criticism was very similar to that which Dr Roy had voiced in respect of Dr Barnett's treatment of Jessica Stemmer. The criticisms insofar as they related to Dr Barnett's handling of Thomas Mahar, were articulated in the first instance in Dr Roy's report[97]. In essence, what Dr Roy had been suggesting was that the subgaleal haemorrhage that was diagnosed upon delivery had clearly been the main cause of Thomas' poor condition and subsequent death and that insufficient attention had been bestowed upon that aspect of Thomas' presentation. In his report Dr Roy acknowledged that perinatal hypoxia may have made a small contribution to his presentation and have exaggerated the effects of the hypoperfusion and that it had 'distracted' his carers, specifically Dr Barnett, from being clear about the problem and, by extension, had distracted him from an appropriate form of management which ought to have included an expedited blood transfusion at Ashford.

7.21. During the course of the Inquest it became apparent that Dr Roy had misidentified the precise source of what he described as an arterial blood gas result taken at 10:27pm. I speak here of a blood gas result that is contained within Exhibit C4a, which is Thomas' clinical record at Ashford, and in particular the document at page 21. Dr

[97] Exhibit C8a

Roy was under the erroneous impression that this was an arterial blood gas test that had been taken some minutes after delivery whereas in fact it related to a sample taken from the child's umbilical cord artery which would have been reflective of his condition at birth. Naturally the results of this sample would have more accurately reflected Thomas' condition at the time of delivery and not have represented any deterioration in the short period of time post delivery. The underlying premise of Dr Roy's original critical opinion as to the appropriateness of Thomas' management was his belief that at birth Thomas' results had been reflective of a less severe picture than had been the case and that a severe worsening of his clinical picture since delivery had not been properly taken into account. This premise was false insofar as Thomas' condition at birth was one of severity.

7.22. In the event, a proper interpretation of the blood gas result of 10:27pm leads one to the conclusion, which Dr Roy now accepts, that the child was in fact born with a severe acidosis and that this was reflective of a significant hypoxic ischaemic episode during the delivery process. In the event, Dr Roy in his evidence very properly and appropriately withdrew the significant criticisms that he had levelled at Dr Barnett in respect of Dr Barnett's handling of the matter. Specifically, Dr Roy said that his amended view that at the time of birth Thomas' presentation had been one of hypoxic ischaemia was now very much in line with the concern that had been identified by Dr Sandercock on the CTG trace prior to delivery and was very much in line with Dr Barnett's assessment that the baby's presentation and condition contained a very significant component of hypoxic ischaemia. The fact that hypoxic ischaemia was now considered to be a major contributor to Thomas' condition at birth meant that Dr Roy's opinion as to the contribution made by the subgaleal haemorrhage, and the need to deal with it, needed to be revised[98]. Further testing administered by Dr Barnett confirmed an even more severe metabolic acidosis than had been apparent at birth.

7.23. On the other side of the ledger, whereas although the child's haemoglobin level at birth had been at the high end of normality, the further tests revealed that the level had descended but were nevertheless still within the limits of normality. Later testing revealed, if anything, a slight improvement in the haemoglobin level. Originally Dr Roy appears to have been of the view that the descent in haemoglobin levels after Thomas' delivery had signified a bleeding difficulty caused by a subgaleal

[98] Transcript, page 820

haemorrhage which was not adequately dealt with by way of fluid replacement and transfusion. Dr Barnett on the other hand had always been adamant that there had been no requirement on his part to administer any blood while the child was under his care at Ashford. Dr Roy acknowledged that his original view that after the blood gas tests had been made available at Ashford a blood transfusion had been indicated and necessary prior to the child's retrieval to the WCH now had to be revised. This was because Dr Barnett's focus would have had to have been placed upon the issue of the child's hypoxic ischaemia. In fact, Dr Barnett's attention had been so focused. Thus Dr Roy had to concede that it was reasonable for the child to have been transferred to the WCH before any transfusion, particularly having regard to the fact that the haemoglobin level on consecutive blood gas results seemed to have stabilised, had remained within the limits of normality and, if anything, had slightly increased from 16.0 to 16.9 which in a sense was reassuring. This view was also that shared by Associate Professor Haslam. In fact, it was universally accepted that circumstances in which a transfusion would be indicated where haemoglobin levels were at or round 16 were difficult to envisage.

7.24. In the event, Dr Roy agreed that the contribution of the subgaleal haemorrhage to Thomas' deteriorating condition had been given the appropriate attention and had been properly recognised and discussed as between Dr Barnett and Dr Bhatia who was a member of the WCH retrieval team. Dr Roy was of the view that the care at Ashford was appropriate.

7.25. Dr Roy did, however, remain of the view that monitoring of the child's blood pressure at Ashford would have been appropriate in Thomas' case. This was the same view that he had offered in relation to Jessica Stemmer. It is fair to say that his view and that of other practitioners who gave evidence in this case very much differed as to that and there is no point in this Court endeavouring to resolve the issue.

7.26. The other matter that Dr Roy raised was that in his view Dr Barnett's request for the attendance of a retrieval team from the WCH could have been undertaken earlier. Again it has not been necessary for me to come to any conclusion about this in view of the fact that irrespective of when the retrieval team was called, the treatment of Thomas Mahar in the first 87 minutes of his life before the arrival of the retrieval team would not have been any different.

7.27. In my view there is no basis upon which Dr Barnett's management of Thomas Mahar at Ashford can be criticised. The same is to be said for the performance of Dr Bhatia who was the principal member of the retrieval team on the night of 9 April 2007.

7.28. The treatment of Thomas Mahar over the following days of his short life was examined by counsel in some considerable detail, particularly as it was described by Associate Professor Haslam and Dr Bhatia. The recitation of all of this clinical history for the most part served little purpose except to the limited extent that it helped to elucidate the child's cause of death. There is no aspect of the child's treatment at the WCH beyond the events of the early hours of the morning of 10 April 2007 that is the subject of any adverse comment by any person who has examined this matter. Indeed the contrary is the case.

7.29. There was, however, one area of concern and that involves the timing of the administration of the first blood transfusion and fresh frozen plasma that took place in the early hours of the morning following Thomas' delivery. The first blood transfusion commenced at 3:15am when 50mls of packed red cells were given over 1 hour. This transfusion was administered in excess of 2 hours since Thomas' admission to the WCH and approximately 5 hours since delivery when the subgaleal haemorrhage was first noted. These timeframes are identified by Dr Roy in his report[99]. Dr Roy notes that the first haemoglobin test performed at WCH revealed a level of 12.6 which was considerably down on the readings established at the Ashford and to Dr Roy this represents a significant loss of circulating red cell mass, that is to say significant bleeding.

7.30. As alluded to earlier, the principal member of the retrieval team from the WCH was Dr Vineesh Bhatia who gave evidence in the Inquest. Dr Bhatia obtained his Fellowship of the Royal Australasian College of Physicians in July 2005 and has worked in the Neonatal Unit of the WCH for the last 10 years. He is current a consultant in the Neonatal Unit. His role as such involves the primary care for patients admitted to the Unit. Dr Bhatia attended at the Ashford on the night in question. He noted the haemoglobin levels that had been taken at various times. The level of 16.9 that was up slightly from the previous test was reassuring insofar as it indicated that the subgaleal haemorrhage was not large and was not the cause of the poor perfusion that the baby was experiencing at that point in time. His view, like

[99] Exhibit C8a

that of Dr Barnett, was that the child's condition was explained by significant hypoxia. I have already indicated that this assessment of the situation is now accepted as being correct. Dr Bhatia decided that Thomas required retrieval to the WCH. One reason for this was his assessment that the child needed an arterial line put in so that his blood pressure could be accurately monitored. Secondly, his blood tests needed to be repeated in order to determine whether his acidosis was improving or worsening. Thirdly, it needed to be determined whether his haemoglobin level would drop. Fourthly, a coagulation profile needed to be obtained. In addition to all of that, the child needed inotropic support which would require the insertion of a central venous catheter. There were other reasons why the child required retrieval and more intensive care than the Ashford could provide.

7.31. When Thomas was retrieved to the WCH, Dr Bhatia telephoned the blood bank advising them that they were going to send a sample for the purpose of blood grouping and cross matching in anticipation of the possible need for a blood transfusion and administration of plasma. This took place at approximately 1:30am. The samples were taken at about that time.

7.32. Dr Bhatia told me that at approximately 1:50am some blood gas results were made available to him. Thomas' pH level had improved a little when compared to the Ashford results. The lactate had come down slightly as well. However, the haemoglobin level had dropped from approximately 16 at Ashford to 12.5. Nevertheless, while Dr Bhatia still regarded this as a reasonable haemoglobin level for a baby[100], he considered the possibility that the baby could be bleeding and so a decision was made to transfuse when the blood that he had ordered became available. After Dr Bhatia had completed putting a line in the child he made an enquiry as to the whereabouts of the blood. This occurred at approximately 2:10am. It was revealed that the necessary samples had not even reached the laboratory. The blood bank was on the fourth floor of the WCH building and the laboratory is very close to that. Dr Bhatia instructed the nursing staff to then fast track the samples and get them delivered to the laboratory as soon as possible. Dr Bhatia personally rang the blood bank again and urged them to contact him as soon as the blood was ready. When asked in evidence as to whether the failure to deliver these samples to the laboratory in the first instance could be explained, Dr Bhatia said essentially it was due to the

[100] Transcript, page 623

exigencies of the then very busy workload during the course of that night[101]. As a result of the discovery that the blood samples had not arrived at the laboratory Dr Bhatia took steps to ensure that it did and it appears from the documentation that the laboratory received the necessary samples at 2:18am.

7.33. At approximately 2:10am at which time Dr Bhatia had discovered that the blood samples had not been delivered, he had two options. He could either wait for the cross match and for the blood to arrive or he could have given O negative blood then and there. He did not give O negative blood at that time. His reason for waiting included his view that although the haemoglobin level had dropped, it was still at a reasonable level for a baby. As well, inotropes were being given to the baby which would have boosted his blood pressure. As well the baby had been given fluid. The baby was oxygenating satisfactorily. In addition, Dr Bhatia was concerned about the prospect of giving the child too much fluid and so he decided to wait for the appropriately matched blood. As well, there was the possibility that fresh frozen plasma might have to be given in the light of coagulation status and Dr Bhatia believed that the blood and plasma might have to be given together. All that said, it is clear that Dr Bhatia would have given the blood if it had been made available earlier. At 3:00am results pertaining to the baby's coagulation factors were made available and these indicated a significant derangement. This could have been explained by the perinatal hypoxia as well as by an exhaustion of coagulation factors due to haemorrhage[102]. In the event, as indicated earlier the blood transfusion of cross matched blood commenced at 3:15am. A transfusion of fresh frozen plasma was commenced at 3:40am. Dr Bhatia had to send a separate request for fresh frozen plasma and this explains why the plasma was not commenced until 25 minutes after the commencement of the blood transfusion.

7.34. There were subsequent transfusions administered, the details of which do not need to be recited.

7.35. Unfortunately the subgaleal haemorrhage increased in size as the night progressed which suggested that Thomas was continuing to bleed, notwithstanding the transfusion of blood and fresh frozen plasma. Indeed, the administration of the fluid volume was one factor that gave rise to the increase of the size of the subgaleal

[101] Transcript, page 629
[102] Transcript, pages 634-635

haemorrhage[103]. There was also the question of the subdural posterior fossa haemorrhage which was reported on the following morning when a CT scan of the brain revealed that the haemorrhage was extensive. Dr Bhatia was of the view that both that haemorrhage and the intrapartum hypoxia had contributed to the coagulopathy as well as the subgaleal haemorrhage[104].

7.36. In the event, there appears to have been a delay of 30 to 40 minutes[105]. Dr Bhatia agreed that if the samples had not been lost the blood would have been given sooner. There is no evidence that an earlier blood transfusion than 3:15am or that an earlier transfusion of fresh frozen plasma that was commenced at 3:40am would have made any difference to Thomas' outcome.

7.37. I do not need to recite the remainder of Thomas' treatment at the WCH that included further involvement of Dr Bhatia and also of Associate Professor Haslam. Suffice it to say there is no area of criticism identified.

7.38. Thomas passed away on 17 April 2007 at the WCH after intensive treatment that can only be described as having been of the highest order.

7.39. Conclusions relative to the death of Thomas Mahar

Thomas Mahar's cause of death was multiorgan failure and coagulopathy following haemorrhage and hypoxia. Thomas was born with a subgaleal haemorrhage that was the result of the force applied to his scalp by way of ventouse extraction. However, his propensity to haemorrhage was heightened by an hypoxic insult that he sustained during the birthing process and as a result, the severity of the subgaleal haemorrhage that contributed to his death was probably greater than it otherwise might have been but for the hypoxic insult.

7.40. I find that if in the phone conversation at 9:10pm Dr Sandercock had been advised with precision as to the abnormalities that had been detected in the CTG trace to that point in time, Dr Sandercock would probably have immediately departed for the Ashford and have arrived there about 25 minutes earlier than she did. Therefore, a period of hypoxia represented by that delay probably could have been avoided. It is

[103] Transcript, page 641
[104] Transcript, page 642
[105] Transcript, page 699

not possible to determine whether and to what extent death or a serious intellectual deficit in Thomas could have thereby been prevented.

7.41. There is no basis to bring into question the appropriateness of Dr Barnett's management of Thomas at the Ashford or the level of skill that he devoted towards Thomas' neonatal care. He did everything that he could for Thomas.

7.42. There is no basis to bring into question the appropriateness of Dr Bhatia's management of Thomas at the Ashford or the level of skill that he devoted towards Thomas' neonatal care. He did everything he could for Thomas.

7.43. There is no basis upon which the neonatal care delivered to Thomas by either the Ashford or the Women's' and Children's' Hospital can be criticised. However, the first blood transfusion delivered to Thomas at the WCH was delayed by the fact that the blood samples required for cross-matching purposes were not delivered to the laboratory in a timely manner. There is no evidence that this delay affected Thomas' outcome.

7.44. There is no evidence that the death of Thomas Mahar could have been prevented by any alternative neo-natal care that could have been administered at the Ashford or at the WCH.

8. Recommendations

8.1. Pursuant to Section 25(2) of the Coroners Act 2003 I am empowered to make recommendations that in the opinion of the Court might prevent, or reduce the likelihood of, a recurrence of an event similar to the event that was the subject of the Inquest.

8.2. I have already referred to the July 2009 College Statement of the Royal Australian and New Zealand College of Obstetricians and Gynaecologists entitled 'Prevention Detection and Management of Subgaleal Haemorrhage in the Newborn'[106]. Although this document was promulgated by the professional body of obstetricians and gynaecologists, much of its content is in essence directed to the attention of neonatal carers as opposed to those practitioners such as obstetricians and gynaecologists who are concerned for the most part in the assisted delivery of children, particularly by

[106] Exhibit C7f

way of ventouse extraction. It was suggested in the course of the evidence from a number of quarters, including counsel, that a document such as this would very appropriately reside in the papers of the Royal Australasian College of Physicians to be specifically drawn to the attention of neonatal specialists. I come back to that issue in a moment.

8.3. What this case has demonstrated in my view is that the unpredictability of the behaviour of subgaleal haemorrhages may not be completely understood. Dr Barnett was a very experienced neonatologist and the sudden deterioration in Jessica Stemmer, I find, genuinely took him by surprise. It has not been necessary for me to resolve the debate as to whether or not Jessica's deterioration was the product of an ongoing bleeding episode in which her coagulation factors were being depleted, or whether her bleeding had stopped and she suffered an acute episode of bleeding at the time of her eventual collapse. Suffice it to say, whatever process was at work her picture of haemodynamic stability was clearly one that was falsely reassuring insofar as it was thought to ameliorate the risk of any further deterioration in her presentation. All of this demonstrates in my opinion that special care is required in the management of subgaleal haemorrhages sustained at the point of delivery, and that the possibility of either continued bleeding or a re-bleeding needs to be very much kept in mind. As I understand the position there is no equivalent College Statement promulgated by the Royal Australasian College of Physicians that deals with the management of subgaleal haemorrhages. The College of Obstetricians and Gynaecologists' document[107] sets out in some detail the matters that need to be addressed as far as the monitoring and treatment of a subgaleal haemorrhage is concerned. To my mind a similar document ought to be promulgated by the Royal Australasian College of Physicians and directed to the attention of neonatologists. Dr Roy expressed the view that such a document insofar as it might be promulgated by the professional body of neonatal specialists was overdue.

8.4. Indeed, it would be useful for the wider medical profession to have its attention drawn to the circumstances of this case with a view to the education of practitioners, especially in relation to the unpredictability of and severity of consequences of subgaleal haemorrhages.

[107] Exhibit C7f

8.5. The circumstances in the death of Jessica Stemmer indicated that there was a lamentable level of timeliness in the delivery of the cross matched blood that Dr Barnett had ordered. As indicated earlier I do not know the reason why an assurance that the blood would be cross matched and available within an hour of his request was not honoured, and less still do I have an explanation as to why its delivery was delayed as long as it was. It seems to me that the potential for delay can be avoided if, immediately upon the identification of a subgaleal haemorrhage of whatever severity in a recently delivered infant, that steps are immediately taken, even in a Level 2 hospital, to ensure the availability of cross matched blood in quad packs and cross matched fresh frozen plasma should the transfusion of either become necessary. Associate Professor Haslam agreed that such a measure would be very prudent[108].

8.6. There is an issue concerning the availability of invasive blood pressure monitoring capabilities in a Level 2 hospital like Ashford. Dr Roy was of the view that blood pressure monitoring of some form ought to be utilised in these scenarios. Dr Roy, however, could not deny that there was room for debate about the utility of blood pressure monitoring in clinical scenarios such as these and particularly as to its necessity and accuracy when the blood pressure monitoring is by way of cuff. When asked as to whether the more accurate invasive blood pressure monitoring ought to be available in a Level 2 hospital Dr Roy suggested that the objections would probably be based upon the rarity of it being required and its expense. As well, the nursing staff in these environments would not be expected to have been trained in the use of such monitoring. I say no more about invasive blood pressure monitoring.

8.7. Dr Roy in his evidence also made a suggestion that there was a need for collective knowledge and collective experience concerning the behaviour of subgaleal haemorrhages to be collected and developed.

8.8. I make the following <u>recommendations</u>:

1) That the Minister for Health and the Medical Board of South Australia draw these findings and recommendations, and in particular those relating to the circumstances surrounding the death of Jessica Stemmer, to the attention of the wider medical community;

[108] Transcript, page 766

2) That the Royal Australasian College of Physicians draw these findings and recommendations to the attention of its members, and in particular those members who are neonatologists;

3) That the Royal Australasian College of Physicians promulgate and circulate for the benefit of its members a College Statement that replicates that of the Royal Australian and New Zealand College of Obstetricians and Gynaecologists document dated July 2009 and entitled 'Prevention Detection and Management of Subgaleal Haemorrhage in the Newborn';

4) That the Royal Australasian College of Physicians draws to the attention of its members, and in particular neonatologists, the following matters:

   a) That practitioners should recognise that subgaleal haemorrhages can behave in unpredictable ways and can have devastating consequences;

   b) That undue reliance should not be placed upon a clinical picture of haemodynamic stability alone as the clinical picture may be falsely reassuring;

   c) That regular monitoring of acidosis and haemoglobin levels, among other parameters, is essential;

   d) That upon a diagnosis of a subgaleal haemorrhage in a neonate, practitioners should have regard to the potential need for cross matched blood transfusion and transfusion of fresh frozen plasma and that they should immediately take the necessary steps to ensure that cross matched blood and fresh frozen plasma is available to be administered at short notice;

   e) That if a decision is made to administer a blood transfusion or a transfusion of fresh frozen plasma that practitioners should ensure that it is administered without delay.

5) I draw recommendation d) to the attention of the Minster for Health for transmission to the Chief Executive Officers of all hospitals in South Australia that provide obstetrics services, be they Level 1 or Level 2 hospitals;

6) That the Chief Executive Officer of the Women's and Children's Hospital take the necessary steps to ensure that in future, assurances given to medical practitioners as to the availability of cross matched blood are met.

Key Words: Subgaleal Haemorrhage; Ventouse Extraction

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 9th day of July, 2010.

Deputy State Coroner

Inquest Number 36/2009 (1762/2006, 0493/2007)