IN THE CORONERS COURT
OF VICTORIA
AT MELBOURNE

Court Reference: COR 2007 003907

FINDING INTO DEATH WITH INQUEST

Form 37 Rule 60(1)
*Section 67 of the **Coroners Act 2008***

Inquest into the Death of: DISHITA MADAMSHETTY

Delivered On:	16 July 2012
Delivered At:	Coroners Court of Victoria Level 11, 222 Exhibition Street Melbourne
Hearing Dates:	16, 17, 18 and 19 April 2012
Findings of:	K. M. W. PARKINSON, CORONER
Representation:	Mr D. J. Wallis on behalf of the family; Mr J. P. Constable on behalf of Western Health.
Police Coronial Support Unit	Senior Constable K. Ramsey

I, K. M. W. PARKINSON, Coroner having investigated the death of DISHITA MADAMSHETTY

AND having held an inquest in relation to this death on 16, 17, 18 and 19 April 2012
at Melbourne
find that the identity of the deceased was DISHITA MADAMSHETTY
born on 30 August 2007
and the death occurred on 30 September 2007
at Royal Children's Hospital, Flemington Road, Parkville, Victoria 3052

from:

1a. HYPOXIC BRAIN INJURY IN THE SETTING OF MECONIUM ASPIRATION SYNDROME AND CHORIOAMNIONITIS/FUNISITIS

in the following circumstances:

1. Baby Dishita Madamshetty was born to Mrs Shashi Madamshetty and Mr Vasudev Madamshetty at the Sunshine Hospital on 30 August 2007. Dishita was born with severe hypoxia and she died in the neonatal intensive care unit at the Royal Children's Hospital on 30 September 2007. The following witnesses gave evidence in the inquest: Mrs Shashi Madamshetty, Midwife Michelle Dibasi; Obstetric Registrar, Dr Nita Dhupar; Associate Professor Glyn Teale and Forensic Pathologist Dr Mathew Lynch. An independent expert opinion was provided to the court by specialist obstetrician and gynaecologist Dr Christine Tippet, who also gave evidence.

Background and Antenatal Attendances

2. Mrs Madamshetty had conceived the pregnancy through IVF procedure, which took place in India. Mrs Madamshetty had an 8 year history of infertility. The pregnancy was confirmed by ultrasound at 7 weeks gestation and because the pregnancy was IVF the due dates were certain at 20 August 2007. She returned to Australia for her antenatal and obstetric care.

3. Mrs Madamshetty attended regularly at the Sunshine Hospital for antenatal care and on her first clinic attendance on 3 May 2007 at 24 weeks gestation, consulted Dr Michael Sedgley. His correspondence to the GP dated 3 May 2007, advised that at this consultation Mrs

Madamshetty had expressed concern regarding the high risk nature of her pregnancy and whether a caesarean section would be performed and that she was advised that many IVF pregnancies were born vaginally, and that if there was an indication for caesarean section, one will be performed. She attended a midwife antenatal clinic from that date in accordance with scheduled appointments.

4. Mrs Madamshetty stated that on antenatal visits she requested delivery by caesarean section, as she was concerned for the baby and wished to deliver a healthy child. On 20 August 2007, she was at term and attended for her 7th antenatal appointment. Her evidence was that at that appointment she indicated she was very concerned for her baby because it was an IVF baby and she did not want any complications. She states that she had cold and flu like symptoms at the time and was concerned about her own health and that of the baby. Foetal monitoring did not identify any problems with the foetus or pregnancy.

5. Mrs Madamshetty states that both she and her husband again requested a caesarean delivery and they were told by the medical staff that they would not normally undertake caesarean unless it was an emergency, however that if the pregnancy continued to 42 weeks then a caesarean would be undertaken. Mrs Madamshetty was advised upon cold and flu medication and another appointment was made for prenatal clinic.

6. On 23 August 2007, (at term plus 3 days) Mrs Madamshetty presented to the hospital with intermittent period like pains, which had commenced the previous day, and continuous pain with some vaginal bleeding and a headache. Observations on presentation were that she was afebrile, she had a tachycardia of 110, normal respirations, was normotensive and urinalysis was positive for leucocytes, protein ketones and blood. Examination of the fundus was recorded as 37cms. It was a longitudinal lie, a cephalic presentation, and the head was engaged with two-fifths of head palpable abdominally. Contractions were recorded as tightening 'two and every ten minutes'.

7. A CTG[1] was undertaken, which was reported as reassuring. Comments were made that (1) there was some clear fluid seen on underwear (2) back pain increasing (3) contractions hard to palpate. A second CTG was requested and reported as normal. A vaginal speculum

[1] Exhibit 8 - Cardiotocograph is a machine which measures the foetal heart rate and provides an indication as to foetal well being by reference to heart rate, its response to contractions and its recovery time to a pre determined baseline.

examination was undertaken and no liquor was seen, and a digital examination revealed that the cervical os was closed, the cervix was 2cm along, and the vertex was 3cm above the spines. Mrs Madamshetty was discharged home, six hours after presentation with analgesia for right sided pain.

8. On 27 August 2007 Mrs Madamshetty attended for her scheduled antenatal appointment. She was then at term plus 7 days. She reported continued upper respiratory infection and discussions were held with the medical registrar, emergency department and the obstetric resident as to appropriate management. A CTG was performed and was reported as normal. An amniotic fluid index was 5.2. A handwritten note of the AFI reported minimal fluid. Foetal dopplers showed a low resistance blood flow in the umbilical arteries and low pulsatility index in the middle cerebral artery.

9. Mrs Madamshetty was advised to obtain cough medicine from her chemist and to see her GP if there was no improvement. Arrangements were made for induction of labour on 30 August 2007. These arrangements are documented in the maternal health record against the appointment of 27 August 2007 and so it would seem that this arrangement was made at that attendance.

10. On 30 August 2007, when she was term plus 10 days, Mrs Madamshetty attended the pregnancy day stay unit (PDSU) for Prostin induction. Admission time to the PDSU was 0845 hours and she was admitted under the care of Midwife Michelle Dibasi. Shortly after admission the CTG was commenced.

11. Midwife Dibasi reports that the CTG was commenced at approximately 0850 hours. She stated that before commencing the CTG she would have ensured that there was a foetal heart rate within normal limits and the foetal heart rate was recorded at 132bpm. Her evidence is that after commencing the CTG she would have ensured the trace was working and that there was a foetal heart rate within normal limits[2]. In relation to the foetal heart rate and the application of the CTG she stated[3]:

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[2] Exhibit 3 Statement dated 14 March, 2012 at 2.13 and T38.5

[3] T22.18

"I probably would have written it I think- we normally check the foetal heart rate before we put it on and I've made a note that it was 132 and then I would have put it on and just made sure that – I don't recall exactly, I don't have a recall of what it was, but I've looked at the trace and normally as long as the heart rate is between 110 and 160 for us it's considered a normal foetal heart rate and that's considered reassuring because it is within normal range."

12. She is unable to state how long it was after the CTG monitoring was commenced that she left the room. However her evidence is that she did not think she was in the room long enough to make any assessment about the trace itself because *"traces don't start or instantly meet criteria and take time to run their course"*. When asked to comment upon the trace in her evidence Ms Dibasi stated that for the first 10 minutes (from 0850 to 0900 hours) there was what is considered to be a normal foetal heart rate. She then noted at about 0900 hours a deceleration which she described as the first and down to 115/120 lasting for approximately 90 seconds. Then the foetal heart rate went up to 160 and remained there. The variability decreased after that deceleration and there was a rise in baseline between 160 and 170. She also stated that the heart rate had initially been fine and within normal range but that the initial persistent deceleration was evidence that the baby was possibly in trouble[4].

13. Ms Dibasi stated that the PDSU was very busy and she then left the room to attend to other patients including a patient requiring an iron infusion and attendance for at least 30 minutes after commencement of that infusion. She stated that there was no specific time frame within which the CTG must be checked and that the day was an extremely busy day and she got back to Mrs Madamshetty as soon as she could after dealing with all of the other tasks required of her in the unit at that time[5].

14. Dr Tippet was asked about this approach to the CTG monitoring. Her evidence was that the trace was abnormal from commencement (which Dr Tippet put at 0900 hours as she said the trace was illegible prior to that time) did not require any time to assess, and warranted

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[4] T28.20

[5] T42. 25

immediate response[6]. It was also her evidence that the CTG ought to have been reviewed within 10 minutes of commencement.

15. When Ms Dibasi reviewed Mrs Madamshetty's CTG at 0925 hours she recorded that the CTG was non reassuring with a rising baseline (from 140 to 175bpm), no variability (<3bpm) and prolonged deceleration (x2 down 60 beats from baseline lasting 90-120 seconds). She then notified the RMO of the non-reassuring trace. It does not appear that the resident attended and at 0930 hours she paged the obstetrics and gynaecological registrar and called the birthing suite. She was advised to transfer Mrs Madamshetty to the birthing suite, as the registrar was unable to attend PDSU to review her. Mrs Madamshetty was transferred to the birthing suite at 0935 hours. Ms Dibasi stated she did this in order to have her immediately reviewed by a registrar and to provide a handover to the midwife in the suite.

16. The trace was removed to transfer her to the birthing suite and re applied at approximately 0942 hours. The trace continued to be abnormal. The birthing suite registrar performed amniotomy and thick meconium liquor was noted. A decision was made at 0958 hours to perform a caesarean section and Mrs Madamshetty was transferred to surgery at 1000 hours. Baby Dishita was born at 1026 hours in poor condition.

17. Paediatric Registrar Dr David Tran reported as to the clinical course after delivery[7].

> *"On delivery she was unresponsive, cyanosed, with no respiratory effort and no movement. She was covered in thick meconium. Her oropharynx was suctioned at less than 1 minute of age under direct vision, with meconium present below the vocal cords. She gasped at 1 minute of age and received intermittent positive pressure ventilation by bag and mask. She was intubated at 8 minutes of age after 3 previous attempts with suctioning between. Irregular breathing was present by 10 minutes of age. Her heart rate was over 100bpm by 1 minute of age and remained adequate throughout the resuscitation. Her Agpars were scored 2 at 1 minute, 2 at 5 minutes, and 4 at 10 minutes of age, by which time there was some improvement in her colour, tone and irregular breathing was*

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[6] Dr Tippet at T193.6

[7] Exhibit 12 Inquest Brief page 13- Statement of Dr David Tran dated 5 March 2009.

noted. PH (cord blood) was 7.25. A capillary blood gas analysis at 11.35 revealed respiratory acidosis with pH 6.983."

18. Dishita was transferred to neonatal ICU at the Royal Children's Hospital with a diagnosis of meconium aspiration. She did not progress and her prognosis for survival was poor. Ventilation and other artificial support measures were ceased on 19 September 2007 and Dishita died on 30 September 2007.

19. An autopsy was undertaken by Forensic Pathologist, Dr Matthew Lynch and an examination of the brain by Neuropathologist Associate Professor, Penny McKelvie[8]. Dr Lynch reported that the cause of death was hypoxic brain injury in the setting of meconium aspiration syndrome and chorioamnionitis/funisitis. The findings on autopsy included profound perinatal anoxic brain injury, focal cortical dysplasia in cerebellum and chorioamnionitis, funisitis and membrane changes of meconium contact and histological changes in lung comprising bronchopneumonia and features in keeping with meconium aspiration syndrome. The final neuropathological diagnosis reported: (1) Profound perinatal asphyxia injury with virtually total loss of neurons, gliosis and cavitation of the cerebral cortex and white matter, very severe (subtotal) neuronal loss and gliosis in the thalamus, basal ganglia hippocampus, basis pontis, colliculi in the midbrain with focal gliosis and neuronal loss in the cerebellum and substantia nigra and (2) Focal cortical dysplasia in the cerebellum.

20. Family contend that the outcome would have been different had the hospital clinicians acceded to the family request for caesarean delivery and that earlier intervention, on 27 and on 30 August 2007 for delivery, would also have been likely to have altered the outcome for Dishita. They also contend that earlier intervention in response to the CTG trace at 0900 hours on the 30 August 2007, would also have been likely to have altered the outcome for Dishita.

21. The scope of this inquest is not broad enough to consider issues in relation to the rights of a pregnant woman to chose or to direct the method and manner of her delivery by caesarean. The extent of that right is a matter for policy makers, and the decision is, within the context of those rights, one for the pregnant woman and attending doctor, to discuss and to determine

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[8] Exhibit 11 – Report of Forensic Pathologist and Forensic Neuropathologist dated 12 March 2008.

between themselves. The issue of caesarean delivery is a matter for consideration in this inquest only in so far as any decision not to undertake a caesarean delivery caused or contributed to the death and was not supportable as a reasonable clinical decision.

22. It is relevant to consider any factors which may have caused or contributed to the death and whether there were interventions of any type which were medically indicated and appropriate on clinical grounds and which if adopted would have likely prevented the death.

23. Dr Christine Tippet, reported[9] upon the clinical course and management. Dr Tippet's view was that a plan should have been made to expedite delivery when the mother presented with pain and bleeding at term, (23 August 2007) particularly in light of the fact that she had conceived with the aid of IVF after prolonged infertility.

24. She was also critical of the decision to book for induction at term plus 10 days. Dr Tippet stated that this would have been appropriate had there been no adverse features, however in view of the bleeding and pain at term a plan for induction should have been instituted earlier.

25. She expressed the opinion that the borderline amniotic fluid at 5.2 and a description of minimal liquor on 27 August 2007 should have resulted in further foetal surveillance or the delivery ought to have been expedited within 48 hours of that attendance[10].

26. Dr Tippet further stated that the delay on 30 August 2007, between when the CTG trace was clearly abnormal, to delivery of the baby which was in the order of 90 minutes was not acceptable. Her evidence was that good practice would have resulted in the CTG being reviewed 10 minutes after commencement. She concluded that[11]:

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[9] Exhibits 9 and 10

[10] T150.19

[11] Exhibit 9

"There is no doubt that the baby was most unwell from the time the mother presented and it is probable that expediting delivery would have resulted in the delivery of an infant in significantly better condition."

27. Dr Tippet reviewed the CTG and commented that the CTG trace was of poor quality and with a lack of accurate dates and times on the traces. She commented:

"The CTG commenced at 0900hours. The first part of the CTG recording is abnormal with the foetal heart recorded as having increased from 120bpm to 160bpm. The trace is consistent with a recovery from a significant deceleration and is smooth and quite abnormal. There was no record of uterine activity on the trace. The trace had persistent abnormalities, with reduced variability, no reactivity and an increasing baseline, and this trace continued from 0920 to 0935 hours."

28. Dr Tippet described the trace as pathological and this characterisation was accepted by other witnesses. She explained her assessment of the trace and her view, in contrast to that of Ms Dibasi, that this was not a trace where any time was required to provide sensible interpretation of the data as follows:

"A normal trace with a well oxygenated baby has a baseline between 110 and 150. The baby will have normal reactivity, which can vary, depending upon whether the baby is in sleep stage or a wake stage and the sleep stage can last up to 40 minutes. But if that baby is stimulated one way or another, or wakes up, it will then show some accelerations. However if you see a pathological trace you do not wait to see whether or not it becomes normal because pathological traces do not recover because this baby is severely hypoxic. Yes you can see brief decelerations, they come back, we often see them in a baby that is a healthy baby, has an isolated deceleration, followed by a normal trace. We see it every day. But, as I say, this trace, if you look at it at 9.10, there's a very smooth recovery with no variability, followed by a very flat trace with no variability. Now that is not a baby that is asleep, that's a baby that's sick."[12]

[12] T193.17

29. Dr Tippet was unable to conclude with certainty as to the specific cause for the baby suffering from severe intrauterine hypoxia. She postulated that placental insufficiency may have been a factor and that the low level of amniotic fluid may have been evidence that this situation was developing[13]. She stated:

> *"I don't believe you were asked to make comment as to when you believe the events may have occurred that saw the harm caused to the baby. Do you have any comment to make on that?---I think that's conjecture, because these things are not usually acute. If they're acute, a mother bleeds or the membrane ruptures and a cord comes down. This was, I would think, without question, a progressive change in placental perfusion, so the baby wasn't adequately oxygenated, and there wouldn't have been an acute episode. One of the reasons we use amniotic fluid index, that reflects how much urine the baby is making, which reflects how much fluid is going to the placenta, and how well the baby is perfused. It is one of the clear indicators we have of reducing placental function. So there was some point in time, probably from then, when the CTG was normal. It doesn't preclude absolutely it having happened before that, but it's unlikely. There was some time over that time where the baby's oxygenation was inadequate. Once the mother started contracting, which she did prior to the morning she came in, that would have been - when a mother is contracting, it's a bit like the baby being dunked in a bucket of water: head goes underwater, comes out again. So there are periods of acute reduction in oxygen supply. If the placenta hasn't got any reserve, when that baby is having that acute reduction in oxygenation, it can't pull on the reserve in the placenta, so it is a time when babies can get an acute - chronic poor placental function becomes manifest."*

30. She noted that the baby was well grown, weighing 3792grams, and at autopsy showed no evidence of foetal abnormality and placental pathology showed no evidence of villitis which would suggest foetal infection. Whilst noting that there was inflammation of the chorion and amnion, and some inflammation of the cord, and Group B strep on culture, Dr Tippet was of the opinion that it was unlikely Group Streptococcus was a major contributing factor to the baby's poor condition at delivery.

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[13] T152.9

Was delivery by caesarean section warranted on 23 or 27 August?

31. Dr Teale's evidence was that there were no clinical indications which would have warranted intervention by caesarean delivery prior to the presentation on 30 August 2007. His evidence was also that there were no clinical indications to suggest that more intensive surveillance or urgent intervention was required. This was because the CTG on 23 and 27 August 2007 was normal, the AFI was within normal range although low, and the reported bleeding had resolved and there were no other clinical indications which would have warranted immediate intervention for delivery. His evidence was that the fact of IVF pregnancy was not a factor which would cause him to be more vigilant or to induce labour at a time earlier than term plus 10 days.

32. Dr Tippet agreed that there was no clinical indication for caesarean delivery on either 23 or 27 August 2007. However Dr Tippet was of the opinion that closer clinical supervision was warranted from the antenatal visit of 27 August 2007 and that there were some indications on that date, including the amniotic fluid index of 5.2 which suggested that things were not travelling well or were about to become problematic.

33. She disagreed with the proposition that an IVF pregnancy did not warrant greater vigilance in the management or surveillance. This was because there is some evidence that associates IVF pregnancy with placental failure. Her evidence was that whether this was related to the fact of IVF or was founded in the initial infertility was a matter of conjecture, however, nonetheless these factors warranted additional vigilance in an IVF pregnancy, particularly one at or beyond term[14]. Dr Tippet stated that IVF is a relevant factor to take into account because it is known that women who have sustained periods of infertility have increased pregnancy complications. She also observed that even well grown babies, like Dishita also have problems with placentation. Her evidence was that this is why the fact that it was an IVF pregnancy was an important matter to be considered when making clinical decisions about the pregnancy, its monitoring and the timing of any interventions including delivery.

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[14] T164.27 – T165.7 and at T187.1

The timing of the hypoxic brain injury?

34. It is not possible to determine with precision, the timing of the hypoxic brain injury which resulted in Baby Dishita's death. I am satisfied that the foetal monitoring on 23 and 27 August 2007 entitles a conclusion that the baby was still well at that time. Dr Tippet agrees with this, although her evidence was that there were some indications at this time that things were not travelling well and that good practice warranted more intense monitoring certainly after 27 August 2007.

35. Dr Teale gave evidence that the insult may have occurred during the period 27 August to 30 August 2007, and that there was a possibility that it occurred by a cord compression event, which resolved itself. However Dr Tippett concluded that an acute cord compression injury was unlikely to have caused the hypoxia, as the CTG continued to be abnormal on 30 August 2007. She stated:

> *"If it had been an acute cord compression injury from which the baby had recovered, that is the event had come and gone, then the baby would have recovered adequate oxygen for its autonomic nervous system to be normal and thus for the trace to be normal and this was not the case."*

36. Having regard to the evidence of Dr Tippet, I am satisfied that it is likely that the pH results at delivery were likely to be indicative of oxygenation of the mother prior to the caesarean section. I do not consider that the pH alone enables me to conclude that a recent and resolved cord compression event had occurred.

37. Dr Tippet's evidence was that at 0900 hours on 30 August 2007, there was an ongoing episode of hypoxia and the baby was struggling. I accept this evidence.

38. The evidence is that the CTG on 30 August, was undertaken as a routine precursor to the induction of labour. It was not being undertaken because there was any abnormal clinical indication or problem with baby or mother and this goes someway to explaining why there was not permanent attendance by a midwife during the course of the monitoring.

39. Ms Dibasi was asked to review the CTG in the course of her evidence. She stated that she regarded the CTG as being normal, '*not perfect but normal*' in the period between 0845 hours and 0900 hours[15]. Both Ms Dibasi and Dr Teale considered that the CTG between 0845 hours as normal, however they conceded that it was not ideal and it was deteriorating.

40. Ms Dibasi's evidence was that she established a foetal heart rate of 132bpm prior to leaving the room. It is not clear whether she reviewed the CTG at commencement and prior to leaving the room. Given the limited information about that observation and the length of time in which it occurred, it is difficult to place much weight upon that evidence, or to conclude with any certainty, that at 0845 hours to 0900 hours there was a normal foetal response or that the critical event first occurred after 0900 hours.

41. Dr Lynch was asked whether he was able to indicate the possible timing of the hypoxic incident. He stated that the neuropathologist was describing an injury to the brain resulting from lack of oxygen and the use of the term 'peri-natal' takes the injury to around the time of birth [16], his evidence was that this could be just before, during or just after. It could not be said whether one day or two days or dated more precisely based upon the pathology alone, although the pathology would support that. Dr Lynch was asked:

> *Q: You have presentation of the membrane changes, you have thick meconium liquor and a CTG which is described as pathological; the CTG commences an hour and a half prior to delivery. Are you able to say in those circumstances whether they, taken together suggest peri-natal asphyxia? Well I think that sort of finding indicates ..an abnormal CTG – that the baby is stressed. The presence of meconium staining of the liquor, evidence that the baby is stressed. If the baby is subsequently born with hypoxic brain injury, one of the possibilities would be – and specially given that she developed meconium aspiration syndrome- that would be one of the explanations. But the reason I used the term in the setting of as opposed to 'due to' is allowing for the possibility that something else caused this insult. So there is lots we know from the autopsy findings. So for example, I know what the placenta was like; that the placenta had developed normally, but it had changes indicating that it had come in contact with meconium. These changes would take some*

[15] T27.24 and T28.7

[16] T205

hours to appear. We know that Dishita's brain showed evidence of injury as a result of lack of oxygen and in broad terms its occurred around the time of delivery, it could have occurred a bit before."

42. He concluded in answer to a question as to whether the findings were consistent with the event having occurred the day before, that is 29 August 2007 as follows:

"The pathology findings are consistent with occurring a day before but they don't allow me to say that it's occurred a day before. So in terms of precisely identifying the time of the insult, the pathology is one piece of information. The interpretation of the CTG, the clinical impression of doctors, midwives, et cetera, of liquor staining and so forth, these are the things that all need to be taken into account. So yes it could have been a day before, but only in terms of whether the pathology would fit with that."

43. The evidence supports the conclusion that the insult, occurred some time after the CTG was undertaken on 27 August 2007 (when the trace was reported as normal) and 0901 hours on 30 August 2007 (when the CTG trace was pathological). The evidence is that it is not possible to entirely exclude that the event occurred before Mrs Madamshetty attended for induction on 30 August 2007 at 0830 hours.

Cause of death, contributing factors and was the death preventable?

44. In considering contributing factors and the question of whether the death was preventable it is necessary to determine whether there were interventions which were reasonable and appropriate in the period from 27 August 2007 and up to and including 30 August 2007 and which may have avoided the hypoxic injury and prevented the death. Dr Tippet considered that there were.

45. I turn first to the 30 August 2007. I am satisfied that the supervision of the CTG trace and the timing of the clinical response to the pathological CTG on 30 August 2007 was inadequate. However it is unclear, whether the extent of the hypoxic injury would have been avoided and the death prevented, had there been earlier intervention on 30 August.

46. In view of the evidence of Dr Tippet that it was likely the hypoxia was a cumulative event, with its genesis in the period after 27 August, I am unable to conclude with any level of certainty that intervention to deliver the baby at 0900 hours in response to the pathological CTG trace, would have resulted in a significantly different outcome for Dishita or that had intervention occurred at 0900hours as opposed to 0935hours that death would have been prevented.

47. I turn now to the period between 27 August and 30 August 2007. Dr Teale's evidence was that the clinical management was reasonable and appropriate and that on the basis of the clinical indications, there was no basis for intervention earlier than 30 August. His evidence however was that in retrospect if Dishita's care been handled differently, the outcome may have been better. Dr Dhupar also expressed this opinion in relation to the monitoring over that period and in particular surveillance after the AFI result of 27 August 2007.

48. Dr Teale's evidence was that because the precise cause of the hypoxia and the precise timing of the event remains unknown, that it is only in retrospect that other interventions might have been regarded as appropriate and reasonable[17].

49. Dr Tippet however did not consider that these interventions were only to be regarded as reasonable and appropriate in retrospect or hindsight. She commented that good obstetric practice is partly putting all of the pieces together and maintaining vigilant surveillance of the clinical course. Her evidence was that there were a number of clinical factors, which would have caused her to conclude that intervention in the form of either increased foetal monitoring or in the absence of such monitoring, proceeding to delivery was appropriate. These were:

> *"That when this mother presented on 23 August, 2007 with pain and bleeding at term in light of the fact that she had conceived with the aid of IVF after prolonged infertility a plan should have been made to expedite delivery. I do not consider discharging a mother with panadeine forte in this clinical situation was appropriate. I do not have access to the ante natal notes. I do not know why and when the mother was booked for induction at term plus 10 days. If there had been no adverse features this would have been most appropriate but when she presented with pain and bleeding at term I am firmly of the view that a plan*

[17] Exhibit 7

should have been made for her induction to be expedited. That when a mother who had a pregnancy conceived through IVF who had previously presented with pain and bleeding was assessed at term plus 7 days and had a borderline amniotic fluid index of 5.2 and a description that the liquor was 'minimal', further foetal surveillance should have occurred within 48 hours or delivery expedited. That the delay from the commencement of the CTG when it was clearly abnormal, to the delivery of the baby, which was in the order of 90 minutes, was not acceptable. There is no doubt that this baby was most unwell from the time the mother presented and it is probable that expediting delivery would have resulted in the delivery of an infant in significantly better condition."

50. Her evidence was that had there been closer monitoring after 27 August 2007 or delivery of the baby within 48 hours of that antenatal appointment, it is likely that the deterioration of the baby's status would have been detected and responded to, with death being prevented. In particular she regarded the AFI result with the notation 'minimal' as of concern and warranting further surveillance.

51. I am satisfied that it is likely had intervention occurred on 27 August 2007 to increase foetal monitoring that the deterioration would have been identified, steps taken to intervene to deliver the baby and death would have likely been prevented.

52. I find that the failure to increase foetal monitoring from the attendance on 27 August 2007 or to proceed to delivery within 48 hours contributed to the death of Dishita Madamshetty.

COMMENTS

Pursuant to section 67(3) of the **Coroners Act 2008**, I make the following comment(s) connected with the death:

1. The evidence of the midwife was that she was unaware of any clinical practice guidelines in relation to the CTG monitoring or the timing of that monitoring and whilst it appears that the hospital had adopted the RCOGS guidelines, there is unclear when and how they are published to the staff.

2. There were no specific requirements relating to time frames for reviewing or monitoring a CTG trace in the MDSU. It appears that there are still no such requirements. Dr Tippet's

evidence is that good practice would see monitoring of the CTG within 10 minutes of it being commenced. The hospital submitted that the college guidelines do not identify any time frames, however that may be because it is anticipated that there will be some initial oversight or more regular supervision of the trace. It would be appropriate in view of this case for the hospital to ensure that pre-induction CTG traces are monitored in a manner which ensures a more timely response to any abnormality which may arise.

3. The family submitted that there was a deficiency in the conveying of clinical information to those responsible for Mrs Madamshetty's care. In particular there was no information available to the midwife performing the CTG monitoring, as to the recent attendance on account of bleeding and pain on 23 August, either on 27 August attendance or on 30 August. The midwife stated that this would not have been likely to have altered her approach to the care because she assumed that the doctors had reviewed this matter and attended to it at the clinic, and that her role was confined to undertaking the CTG and providing its results.

4. I agree with the family submission that it would be helpful for all of the midwifery and obstetric clinicians caring for the patient to have an overall picture available to them. This may have resulted in a different approach to the oversight of the patient after 23 August and greater vigilance in the approach to the CTG on 30 August.

5. The fact that the CTG monitoring pre induction was regarded as routine surveillance may have influenced the timing of the review of the CTG and its priority as against other patients in the unit and may have contributed to the failure to return to review the trace earlier than 35 minutes after fixing. A practice requirement that the trace be reviewed at least after 10 minutes would resolve that issue.

6. The family submitted that they did not consider their concerns were being paid any regard by the clinicians planning the care. Whilst this largely related to the issue of their desire for a planned caesarean delivery, nevertheless there did not appear to be a detailed or comprehensive explanation of the clinical findings provided to the parents or an engagement of the parents, in particular the patient, in the decision making relating to the clinical course. Greater engagement with the patient may result in greater clarity of issues relating to the pregnancy and better information to the clinicians.

RECOMMENDATIONS

Pursuant to section 72(2) of the **Coroners Act 2008**, I make the following recommendation(s) connected with the death:

1. That the hospital adopt and publish formal procedures in relation to CTG monitoring, supervision and oversight and that these procedures be published to all relevant clinicians, including a requirement for regular monitoring of pre induction CTG trace.

I direct that a copy of this finding be provided to the following:

The family of Dishita Madamshetty;
Western Health;
Interested parties.
Dr Christine Tippet

Signature:

K. M. W. PARKINSON
CORONER
Date: 16 July 2012