CORONERS ACT, 1975 AS AMENDED

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 4th, 5th, 6th and 28th days of February 2002, before Wayne Cromwell Chivell, a Coroner for the said State, concerning the death of Lesley Anne Galea.

I, the said Coroner, find that, Lesley Anne Galea aged 31 years, late of 11 Kernick Avenue, Willunga, South Australia died at the Flinders Medical Centre, Bedford Park, South Australia on the 30th day of April 1999 as a result of respiratory failure related to bronchopneumonia following cerebral anoxia due to amniotic fluid embolism following uterine rupture during labour

1. Introduction

1.1. Lesley Anne Galea was 30 years old when she was admitted to Flinders Medical Centre (FMC) on 29 December 1998 for the birth of her second child.

1.2. Mrs Galea's previous child had been born by lower segment caesarean section ('LSCS') on 5 June 1997. Nevertheless, the plan was to conduct a 'trial of scar'. This means that a vaginal delivery would be attempted, with care being taken not to rupture the uterus at the side of the previous LSCS.

1.3. Mrs Galea's ante-natal progress had been unremarkable. Her membranes ruptured at about 5:00pm on 29 December 1998, and her contractions started at about 8:00pm.

1.4. Mrs Galea was admitted to FMC at about 8:30pm on 29 December 1998.

1.5. At about 6:45am on 30 December 1998 an infusion of the hormone Syntocinon, to augment labour, was commenced, and at about 7:35am an epidural anaesthetic was commenced for pain relief.

1.6. Mrs Galea complained of a 'slight headache' at 9:15am, for which she was given Panadol.

1.7. The first sign of a problem developing was at 9:25am when the FMC casenotes (Exhibit C10) record that Mrs Galea was feeling 'funny'.

1.8. Mrs Galea collapsed and appeared to be fitting at around 9:30am. The Obstetric Registrar, Dr Colin Wetherill, was called. The Director of Anaesthesia, Dr Scott Germann, who had coincidentally been walking past the room, was present within a few seconds, and began emergency treatment. Dr Germann's statement reads:

> 'Initially I administered 100% oxygen via a black bag and mask. Mrs Galea's airway was clear, there was no sign of vomit in the mouth and her breathing was shallow but adequate. The pulse oximeter arrived within two minutes and I applied the probe to her earlobe – it showed a saturation of 98%.
>
> With the arrival of the pulse oximeter, nurse Val Follows (Charge Nurse, Recovery) and nurse Joan Jericho (Charge Nurse, Anaesthesia) also attended. They were closely followed by Dr Alisteir Norton, another Consultant in Anaesthesia.
>
> Mrs Galea's respirations and oxygenation were adequate, there was a thinly palpable carotid arterial pulse but no discernible peripheral circulation. At about this time I was asked by the obstetricians whether they should proceed with the delivery.
>
> I indicated to them that the situation was stable and unlikely to improve in the short term and they should deliver the baby as soon as possible. This was duly done and there was little blood present when the episiotomy incision was made. By that stage I had intubated Mrs Galea's trachea and had commenced to assist her ventilation in order to relieve her acidosis.'
>
> (Exhibit C7a, p1-2)

1.9. Dr Verco was paged since he was the duty Obstetrics Consultant. He attended at 9:42am. On vaginal examination, he noted that Mrs Galea's cervix was fully dilated. The baby was delivered vaginally at 9:49am with the aid of forceps, after Mrs Galea had been intubated and ventilated by the anaesthetist. Mrs Galea was unconscious by this stage.

1.10. Mrs Galea suffered a cardiac arrest at around the time of delivery. Cardio-pulmonary resuscitation was commenced along with inotropic medication and defibrillation, and circulation was restored.

1.11. When circulation was restored, Mrs Galea began bleeding heavily, and a coagulopathy (failure of the blood to clot) had developed. Intramyometrial prostaglandin and manual compression of the uterus were tried without success.

1.12. In the Intensive Care Unit, extensive transfusions of red cells, platelets and fresh frozen plasma were undertaken, but the bleeding could not be controlled.

1.13. At about 1:00pm, a sub-total abdominal hysterectomy was performed by Dr Verco, at which time a small rupture, which he thought was only about 2-3 centimetres in length, was found in the lower uterus (Exhibit C5a, p4).

1.14. Following this operation, Mrs Galea's condition slowly stabilised with further transfusions, but it became apparent that she had suffered extensive hypoxic brain injury.

1.15. Mrs Galea was transferred to a ward at FMC on 15 January 1999, but her condition deteriorated, and she eventually died from respiratory failure on 30 April 1999.

1.16. One can only imagine the suffering these events must have inflicted upon the Galea family. The fact that an apparently healthy, 30 year old woman, who has had a normal pregnancy, would suffer such a catastrophic event in the course of an apparently routine labour in a major teaching hospital calls for close analysis.

2. Cause of death

2.1. A post-mortem examination of the body of the deceased was performed by Associate Professor A C Thomas, Senior Specialist Histopathologist, on 2 May 1999. Dr Thomas' significant findings were:

- the presence of severe congestion and exudate in the trachea and bronchi;
- widespread bronchopneumonia consolidation in both lungs;
- early scarring of the heart, consistent with hypoperfusion (lack of blood supply) was noted microscopically.

2.2. Dr Thomas' conclusions were that Mrs Galea's death was due to respiratory failure related to bronchopneumonia subsequent upon cerebral anoxia following uterine rupture during labour.

2.3. A neuropathological examination of the brain was performed by Dr Grace Scott on 18 May 1999. Her conclusions were:

> 'Histological examination of the brain confirms the presence of severe hypoxic injury in all arterial territories of the cerebral hemispheres and in the right superior cerebellar artery territory, with milder changes elsewhere in the cerebellum. The histological features are consistent with a severe hypoxic insult many weeks to months old. In the brain stem, there is evidence of hypoxic injury to the colliculi of the midbrain and also of secondary degeneration of the left cortical spinal tracts secondary to hypoxic injury at the level of the left motor cortex.'
>
> (Exhibit C4b, p4)

2.4. Because of the lapse of time between the initial cerebral event and Mrs Galea's death, it was no longer possible to determine the underlying cause of that event.

2.5. The materials were referred to Professor Roger Pepperell, formerly Chairman of the Department of Obstetrics and Gynaecology at the Royal Women's Hospital in Melbourne. Professor Pepperell has wide experience in obstetrics, and has provided me with an extensive and very clear analysis of these events.

2.6. On the basis of the clinical events, Professor Pepperell said that in view of Mrs Galea's profound collapse, and subsequent coagulopathy, the 'only probable cause' was an amniotic fluid embolism in a cerebral blood vessel leading to cerebral anoxia.

2.7. An embolism is something which occludes a blood vessel, and can be a gas, forming a bubble, or a liquid or a solid. Professor Pepperell explained:

> 'Amniotic fluid embolism classically occurs when the amniotic fluid from within the uterus passes into an open vein within the uterine wall, or within the placental bed.
>
> It has certainly been described in association with a uterine rupture, and anyone who performs a caesarean section is well aware of the size of the uterine sinuses within the area of the wound, into which liquor could pass.
>
> Amniotic fluid embolism is less common when the membranes have been ruptured previously, unless the head is acting as a plug within the pelvic cavity, and some of the amniotic fluid therefore remains above the head.
>
> I would presume that what happened to Mrs Galea is that there was some amniotic fluid held above the head, and when the scar of the uterus gave way, at about 0925 hours (presumably), a moderate amount of amniotic fluid passed into an open sinus at the site

> of the rupture, and then into the general circulation. Because it ultimately got to the cerebral circulation, having passed through the lungs, she became unconscious, and the amount of it disturbed the whole circulatory system, ultimately causing a cardiac arrest in addition, and producing the coagulation failure.'
>
> (Exhibit C19a, p8)

2.8. Professor Pepperell thought that the uterine rupture would not have occurred earlier than about 9:25am, having regard to the absence of bleeding in the genital tract until after the baby was delivered, and the absence of clotted blood in the peritoneal cavity. (Exhibit C19, p9).

2.9. The materials were also referred to Professor Jack Cade, the Director of Intensive Care at Royal Melbourne Hospital, for an assessment of the care given to Mrs Galea after her collapse.

2.10. Professor Cade agreed with Professor Pepperell's analysis. He said:

> 'I too agree that this diagnosis best explains the circumstances of the event. Indeed, it is hard to think of a plausible alternative explanation, as the several other causes of collapse in such circumstances do not fit properly with the clinical features described in her case.
>
> However, it should be remembered that the diagnosis of amniotic fluid embolism is a clinical one and is often presumptive, as laboratory evidence of circulating foetal material is non-specific. Amniotic fluid embolism is uncommon (1 in 8,000-80,000 live births) and can be subclinical, but it can also be fulminating. It is one of the few remaining obstetric causes of maternal death in our society and is a feared complication in those cases sick enough to require urgent admission to an Intensive Care Unit.'
>
> (Exhibit C9a, p1-2)

2.11. Taking all this information into account, I find that the cause of Mrs Galea's death was respiratory failure related to bronchopneumonia following cerebral anoxia due to amniotic fluid embolism following uterine rupture during labour.

3. <u>The progress of Mrs Galea's labour</u>

3.1. Mrs Galea was seen by Dr Olivia Magno, the Intern, at 10:00pm on 29 December 1998. Dr Magno took a brief history, noting the previous LSCS.

3.2. Dr Magno performed a vaginal examination at this time, and confirmed that the amniotic membrane had ruptured. She also noted that the cervix was still 'long and closed'.

3.3. Dr Magno saw Mrs Galea again at 1:00am on 30 December 1998. Her contractions had increased in frequency and intensity, according to the note made by the midwife, Mary James, at that time. They were now at the rate of 2-3 in ten minutes, and 'moderate'. Mrs Galea was requesting an epidural anaesthetic, but Dr Magno thought it was too soon since another vaginal examination revealed that the cervix was only 1cm dilated. This was the last time Mrs Galea was examined vaginally until after she collapsed at around 9:30am.

3.4. Dr Magno prescribed Pethidine for Mrs Galea's pain, and this was given at 1:05am and 4:35am.

3.5. By 4:00am Mrs Galea's contractions had increased in intensity, and her observations were taken more frequently. In particular, the foetal heart rate, which had been taken hourly by Ms James, was taken half-hourly from 4:00am. Except for one deceleration at around 8:00am, it remained normal. The casenotes record that continuous foetal monitoring, using a cardio-tocograph ('CTG') was commenced at 8:10am as a result of this one deceleration but the trace (Exhibit C16) remained normal.

3.6. Syntocinon

The following entry, made by Ms James, appears in the casenotes at 6:00am:

> 'Comfortable, contractions continue – mild 4:10 (4 contractions in 10 minutes). Discussion with Reg (Registrar) – for Synto (Syntocinon)'
>
> *The words in brackets are mine.*
>
> (Exhibit C10)

3.7. Syntocinon (chemical name 'Oxytocin') is a synthetic hormone which stimulates the muscles of the uterus thereby producing, or augmenting, contractions. It is administered intravenously, in a quantity of 10 International Units contained in a 1ml ampoule, mixed with 1 litre of saline, at the rate of 2mls per minute. This infusion commenced at 6:45am.

3.8. It is noteworthy that no vaginal examination was performed prior to the decision being made to infuse Syntocinon. There is therefore no way to assess the extent to which Mrs Galea's labour had progressed to that point, in order to determine whether augmentation was even necessary. Only 2¾ hours later, at around 9:30am, her cervix was completely dilated.

3.9. The significance of this, is that if Mrs Galea's baby had been delivered earlier, the uterine rupture and consequent amniotic fluid embolism may not have occurred. I will deal with this issue in more detail shortly.

3.10. Ms James finished her shift at 7:15am. She noted that Mrs Galea's contractions had increased to 3:10 minutes, which is in fact a decrease. In any event, the casenotes indicate that Mrs Galea's progress was entirely unremarkable to that point.

3.11. At 7:15am the replacement midwife, Ms Wyllie, noted that Mrs Galea was requesting an epidural again. The anaesthetist, Dr Wangel, commenced this procedure at 7:35am, and completed it by 7:50am. Marcaine, an anaesthetic agent, was administered. This was renewed, with a 'top up' of Pethidine, at 9:10am. At this time the foetal heart rate was normal, at 140 beats per minute, and Mrs Galea's blood pressure was normal, at $^{120}/_{75}$. These were the last observations taken prior to her collapse at 9:30am.

4. Issues arising at inquest

4.1. Continuous foetal monitoring

Professor Pepperell was critical of the fact that a CTG was not used during Mrs Galea's labour, in view of the fact that she was having a 'trial of scar'. He said:

> 'In virtually all tertiary referral hospitals in this country, it would be normal to use a continuous CTG monitor of the fetal heart during the whole of labour when someone is having a trial of scar. This does not need to be done with a scalp electrode, if an adequate fetal heart rate recording can be determined using an external monitor. The monitoring of the fetal heart is required because a fetal heart abnormality is often the first sign that a uterine rupture is occurring. The risk of uterine rupture is approximately 1% in patients who are having a trial of scar.
>
> It was therefore necessary for her to have a continuous CTG monitor applied to the abdomen through the whole of labour just on the grounds of the fact she was having a trial of scar. I can find no evidence in the documentation sent to me that a continuous CTG monitor was used, and have certainly not been sent any CTG monitoring record to peruse. I have therefore assumed that the monitor was not used until the fetal heart was first detected to be abnormal on clinical grounds at about 0800 hours.'
>
> (Exhibit C19a, p6)

Professor Pepperell's criticisms were even stronger since there was still no CTG monitoring after Mrs Galea had the Syntocinon infusion, and after she had the epidural anaesthetic. He said:

> 'It is also routine in tertiary referral hospitals to use CTG monitoring in patients who are having epidural anaesthetics, and in those who are having Syntocinon infusions to stimulate or augment labour. The reason for this is because fetal heart abnormalities are more common in the presence of epidural anaesthesia, and also more likely when Syntocinon is being used to stimulate the labour. It is impossible to know exactly what dose of Syntocinon will be required, and if an excessive dose is administered, the contractions tend to become prolonged, the resting pressure between contractions increased, and fetal hypoxia results. This is often recognized first on the CTG, and when the CTG is found to be abnormal the Syntocinon drip is then ceased.
>
> It is also well known that the use of Syntocinon, even in someone who has not had a previous caesarean section, is associated with an increased risk of uterine rupture.'
>
> (Exhibit C19a, p6)

4.2. The CTG trace was not produced by FMC when Mrs Galea's documentation was warranted, despite several requests by the investigating officer, Senior Constable Paul Gross, for <u>all</u> documentation. This is a matter for grave concern. I rely on the professionalism and integrity of hospitals to produce <u>all</u> documentation when a warrant under Section 13(1)(c) of the Coroner's Act is executed. The alternative is for the officer to use the search and seizure powers granted by that section of the Act. This is the second time this has occurred (both involving a CTG trace), the previous occasion involved the Women's and Children's Hospital (see Trimboli, Inquest 11/01). On each occasion the hospital's lawyers have been able to produce the document at, or just prior to the inquest, when the coercive powers in the Coroner's Act have failed to produce that effect. I will draw this matter to the attention of the Chief Executive Officer of the Department of Human Services.

4.3. In any event, once Professor Pepperell was able to see the CTG trace, he was able to discern that there was no abnormality therein which could have alerted the clinicians to the impending disaster, so it cannot be said that the failure to use the CTG earlier has any causal relevance to Mrs Galea's death, except as evidence of a generally substandard approach to obstetric care.

4.4. I note that Exhibit C14, the Handbook for Registrars and Resident Medical Officers, current to 1997, required auscultation (listening to) of the foetal heart rate every 15

minutes, or electronic monitoring 'if indicated', after the membranes have been ruptured. Even on this standard, Mrs Galea's monitoring was inadequate.

4.5. Since these policies were revised, effective January 2002, much clearer guidelines have been put in place. The 'Temporary Policy Documents' (Exhibit C16) now provide that a woman receiving a Syntocinon infusion should have CEFHRM (Continuous Electronic Foetal Heart Monitoring) as a matter of course. I will discuss this issue again later.

4.6. Decision to use Syntocinon/assessment of progress of labour

As I stated earlier, Professor Pepperell was critical of the fact that Syntocinon was infused without checking first whether Mrs Galea's labour had progressed, and to what extent, by examining the cervix. He said:

> '***Assessment of progress in labour***. I am most critical that there was no assessment of progress in labour performed between the time a pelvic examination was done at 0100 hours, and the time of collapse at about 0930 hours. The usual rules in Obstetric practice are that pelvic examination should be performed approximately 4 hourly in patients who are having a trial of scar, to ensure adequate progress is being achieved and the trial of scar is then allowed to continue, and certainly it should also have been performed prior to the use of the epidural anaesthetic at 0750 hours, and again when this was topped up at 0910 hours. It is just not possible to know what is going on with the cervix without the performance of a pelvic examination, and had the cervix already been fully dilated when the epidural was inserted, it may well have been that delivery could have been effected at that stage without much difficulty. It will never be known whether the cervix was fully dilated at that time, and whether delivery was possible, but certainly failure to assess progress of labour during an 8 hour period in someone with a previous caesarean section, who is having labour stimulated, and who has an epidural anaesthesia, is not adequate care.'
>
> (Exhibit C19a, p7)

4.7. Professor Pepperell expanded upon this in oral evidence, given via video-link with Melbourne, as follows:

> 'Q: Are you able to say in Mrs Galea's case what might have been detected if pelvic examination had been done either at the four hourly interview intervals suggested by you or alternatively at the time of the administration of the epidural and/or the Syntocinon whether the outcome would have been any different in this case.
>
> A: I can't say because we don't know what those findings were. If that indicated that the cervix was still only minimally dilated then what was done was appropriate. If however they had shown that the cervix was eight or nine centimetres dilated then Syntocinon might not have been necessary at all and that action may well have been

> taken to the earlier stage prior to the uterine rupture which was presumably the cause of the amniotic fluid embolism' (T146-147)

4.8. Dr Jodie Dodd is now a Consultant, but at the time was the Obstetric Registrar on duty, and was the Registrar with whom Midwife James conferred at 6:00am on 30 December 1998. Dr Dodd acknowledged that it was standard procedure to perform a vaginal examination before deciding to augment labour with Syntocinon (T164).

4.9. Dr Dodd was unable to recall the details of the conversation with Ms James, which is not surprising given the lapse of time since then. She said that she would normally ensure that a vaginal examination had been done, either by the midwife, the Intern, or personally (T164). She was sure that if it had been brought to her attention she would have done so, but could not say that it was, or was not (T173). It seems that the most likely explanation of her failure to arrange for a vaginal examination was that she overlooked it, or assumed that the midwife had done it (T168).

4.10. Ms James, on the other hand, asserted that she had no trouble recalling the incident. She said that she would not perform a vaginal examination unless directed to do so by a Senior Midwife or Medical Officer (T100). She also said that she was sure that she drew the fact that Mrs Galea had not had a vaginal examination to Dr Dodd's attention, although she could not specifically recall the conversation (T113).

4.11. I have serious doubts about Ms James' veracity on this issue. She has been a Registered Midwife since 1973, having trained in the United Kingdom, and had been at FMC since 1996.

4.12. It is my firm impression, after hearing both witnesses, that if the matter had been drawn to her attention, Dr Dodd would have either performed a vaginal examination herself, or asked Ms James or Dr Magno to do it. I do not believe Ms James when she alleges that she drew the matter to Dr Dodd's attention. I find that the topic was not raised by either person, each perhaps assuming that the other had attended to it.

4.13. Whatever was the case, this demonstrated a very serious breakdown in communication between doctor and nurse which must be avoided if such tragedies are to be prevented in the future.

4.14. Hospital protocols

Mr Mills tendered a number of documents touching upon these issues. For the sake of clarity, I will set out a number of matters referred to:

- In the 'Labour and Delivery Suite Handbook', 1997 (Exhibit C12), the role of the midwife is set out:
  - 'The midwife provides one to one care for a woman in labour. This places the midwife in the ideal place to liaise between a woman and the other medical and midwifery personnel.
  - The midwife's prime responsibility is to ensure safety for the woman and her baby, and also provide for her physical comfort and psychological needs.
  - It is the midwife's responsibility to initiate changes in the patterns of care as the clinical circumstances change. This responsibility includes keeping the medical team informed of deviations from normal.
  - The midwife providing care is legally responsible for the consequences of that care and any treatments initiated.'

  The whole tenor of this section is at odds with Ms James' assertions about her responsibilities in the labour ward. The most recent revision of this protocol, in 2000, repeats these provisions verbatim (Exhibit C13);

- The procedure for conducting vaginal examination's is set out at page 18:
  - '4-hourly, if adequate contractions have been present; earlier, if decision needs to be made about analgesia, or augmentation of labour.'
- The procedures for foetal monitoring in labour are set out at page 23:
  - 'The FH is auscultated 15-minutely during established labour, and after every contraction during active second stage.

    Continuous electronic fetal monitoring is not routinely performed in labour. Use is determined by clinical circumstances.

    REASONS FOR ELECTRONIC MONITORING

    - Meconium-stained liquor
    - Reduced liquor volume
    - Antepartum haemorrhage/intrapartum haemorrhage
    - Suspected IUGR
    - Preterm labour
    - Twin pregnancy
    - Pre-eclampsia
    - Prolonged labour/second stage
    - Abnormal FHR

- Breech presentation
- Insulin-dependent diabetes

Scalp electrode applied as indicated'

(This clearly is not in line with Professor Pepperell's practices).

- The procedures when the mother had had a previous LSCS are at page 24:

  - 'Monitor maternal condition including hourly BP, pulse
  - Auscultate FH 15-minutely. Electronic monitoring if suspicious of scar dehiscence or more than 1 previous Caesarean
  - IVT, HB, G&S only if suspicious of scar dehiscence or more than 1 previous Caesarean
  - Use caution with Syntocinon

  SIGNS OF SCAR DEHISCENCE

  - May be 'silent'
  - Bright blood loss
  - Pain over scar
  - Fetal distress
  - Maternal tachycardia, hypotension
  - Decrease in the strength and/or frequency of contractions

  (It is noted that Mrs Galea's contractions decreased in frequency after the Syntocinon infusion).

- In the Manual of Obstetrics, Midwifery and Gynaecology Procedures (Exhibit C15), a number of these procedures are also dealt with. The conditions for CTG monitoring are set out at Section 1.5:

  - The section on Intra-partum care (2.3) requires a vaginal examination every 4 hours, once in established labour, by either doctor or midwife.

  - The section on Induction of Labour (2.13) requires continuous foetal and uterine contraction monitoring for the high risk mother and foetus.

  - The section on Continuous Electronic Foetal Heart Monitoring (2.14) provides that monitoring is not routine, but is performed when indicated. Indications include a combination of more than one of prostaglandin, Syntocinon, epidural and previous LSCS.

  (Mrs Galea had the last three of these indications).

- In a 'Temporary Policy Document' dated January 2002 (Exhibit C16), to form part of Exhibit C15 from that month 'until confirmed or amended by Consultants' meeting', the following changes were introduced:

    - 'Women having 'trial of scar' are to have Continuous Electronic Foetal Heart Monitoring if they are having a Syntocinon infusion;
    - Vaginal examination's 4-hourly are mandated, "to ensure progress with augmentation of labour …"
    - Syntocinon is only to be used with the permission of a consultant'

5. **Conclusions**

Taking all these matters into account, I consider that the following conclusions may be drawn from the evidence:

1. The cause of Mrs Galea's death consisted of complications arising from an amniotic fluid aneurism, which occurred when her uterus ruptured during labour.
2. The fact that Mrs Galea had a previous LSCS did not preclude her from having a 'trial of scar'.
3. Mrs Galea's ante-natal care was quite standard and adequate.
4. Mrs Galea's contractions were initially irregular until about 1:30am. They were sufficiently strong to require pain relief in the form of Pethidine at 1:05am. Mrs Galea's cervix was 1cm dilated at 1:00am. The conclusion can be drawn that she was in established labour by at least 2:30am.
5. Monitoring of the foetal heart was conducted half-hourly until 4:00am and then 15-minutely thereafter. Continuous Electronic Foetal Heart Monitoring was not performed until after 8:00am when a deceleration was noted.
6. No vaginal examination was performed after 1:00am until the emergency occurred at around 9:30am.
7. In particular, there was no vaginal examination prior to a Syntocinon infusion commencing at 6:45am, and prior to an epidural anaesthetic commenced at 7:50am.
8. Because no vaginal examination was performed, it will never be known whether Mrs Galea's baby could have been born earlier than 9:30am, and the uterine rupture thereby avoided.

9. This failure to assess the progress of Mrs Galea's labour before deciding to augment it with a Syntocinon infusion was, in Professor Pepperell's words, which I adopt, 'not adequate care'.

10. Even if it was necessary to introduce Syntocinon, the fact that Mrs Galea was also having 'trial of scar', and that she was also having an epidural anaesthetic, dictated that she should have received continuous electronic foetal heart rate monitoring throughout labour..

11. When Continuous Electronic Foetal Heart Monitoring was introduced at 8:00am, the foetal heart rate was normal, so it cannot be said that the failure to monitor it earlier was relevant to Mrs Galea's death.

12. Dr Verco's decision to expedite the birth by forceps delivery was appropriate, and did not cause the uterine rupture. LSCS at that stage was not an option, since it would have made the chances of a lethal haemorrhage even greater (T159).

13. Dr Germann's efforts, and those of his colleagues, to resuscitate Mrs Galea after her collapse were appropriate and timely. She was in the care of the Director of Anaesthesia of the Hospital, and expert Intensive Specialists within minutes. Professor Cade was 'unable to find any matters which would warrant any criticism in their conduct of the resuscitation' (Exhibit C9a, p1).

14. Similarly, Professor Cade was 'unable to find any matter for criticism in the detailed and manifestly expert care which she received following the events of the morning of 30 December' (Exhibit C9a, p2).

15. Professor Pepperell and Professor Cade agree that, in the words of Professor Pepperell:

> 'Amniotic fluid embolism is one of the most difficult Obstetric complications to diagnose and treat, and had she not been at Flinders Medical Centre, or another tertiary institution, I believe she would have been unlikely to have survived beyond about 09:45 hours.'
>
> (Exhibit C19, p9)

6. Recommendations

6.1. I accept Mr Mills' submission that the changes to the protocols for foetal heart rate monitoring, vaginal examinations and Syntocinon infusions which have already been made (some as recently as last month), render it unnecessary to recommend that further provisions be made to avoid such a tragic event.

6.2. However, I remain concerned about the lack of communication between the midwife and medical staff that night, and Ms James' attitude to the extent of her responsibilities.

6.3. Accordingly, pursuant to Section 25(2) of the Coroner's Act 1975, I recommend that the Head of Obstetrics and Gynaecological Department and the Director of Nursing at Flinders Medical Centre consider whether further guidelines need to be issued, or training needs to be given, so that such misunderstandings might be avoided in future.

Key Words: *Hospital Treatment; Birth Accident; Midwifery; Foetal Monitoring; Amniotic Fluid Embolism*

In witness whereof the said Coroner has hereunto set and subscribed his hand and Seal the 28th day of February, 2002.

Inq No 4/02 (1048/1999)

Coroner