QUEENSLAND COURTS

CORONERS COURT OF QUEENSLAND

FINDINGS OF INVESTIGATION

CITATION:	**Non-inquest findings into the death of Baby L**
TITLE OF COURT:	Coroners Court
JURISDICTION:	Brisbane
DATE:	9 June 2017
FILE NO(s):	2016/984
FINDINGS OF:	Ainslie Kirkegaard, Coronial Registrar
CATCHWORDS:	CORONERS: Neonatal death; Group B Streptococcus (GBS) disease; early onset vs late onset GBS; management of maternal fever and foetal tachycardia during labour; management of febrile infant at birth; Queensland Clinical Guidelines *Early onset Group B Streptococcal disease*

Contents

Background

Baby L was an 18 day old baby girl who died at the Lady Cilento Children's Hospital on 7 March 2016.

Baby L's death was reported to the coroner because of potential concerns about aspects of the management of her delivery and postnatal care at a regional private hospital.

These findings have been informed by review of Baby L and her mother's medical records (treating obstetrician, treating paediatrician, the regional private hospital, Queensland Ambulance Service, two regional public hospitals, Lady Cilento Children's Hospital), preliminary independent clinical advice, formal statements provided by the treating obstetrician, paediatrician and midwives involved in the labour, delivery and post-natal care, the regional private hospital clinical review outcomes and independent neonatology opinion.

Baby L died from Group B Streptococcus (GBS) sepsis. This potentially fatal condition can present early (during the first week of life) or late (after the first week up to three months of life). Early onset GBS is preventable; late onset GBS is not. Consequently, the focus of this investigation has been which manifestation Baby L died from and whether her risk of developing this infection was managed appropriately.

The mother's antenatal management

This was the mother's first pregnancy. Her general practitioner referred her to consultant obstetrician, Dr M.

The mother saw Dr M regularly for antenatal care and her pregnancy was uncomplicated. She returned a normal antibody screen and normal routine antenatal investigations. Her estimated due date based on early ultrasound was 1 March 2016. She was booked to deliver at a regional private hospital.

The mother was not screened for Group B Streptococcal (GBS) carriage. Dr M's notes contain an entry dated 4 January 2016 about having discussed GBS with the mother. Although Dr M cannot recall the detail of this discussion she says her usual advice reflects the following information:

- GBS is a type of bacteria found in 30% of women at any given time and it does not usually cause harm;
- there is a 30% chance that babies will contract it during labour and if they do there is a 3% chance they will become unwell;
- there are two approaches to identifying pregnant women for intrapartum antibiotic prophylaxis to prevent GBS infection – the first, to screen for GBS by reference to maternal risk factors and then treat with prophylactic antibiotics; and the second, to swab for GCS bacteria at 36 weeks gestation and if positive, assume the bacteria will be present at birth and treat with prophylactic antibiotics.

Dr M says she 'usually remarks' that the current Queensland Clinical Guidelines for Early Onset GBS disease (https://www.health.qld.gov.au/qcg/publications#maternity)

recommend the approach of identifying maternal risk factors before antibiotic treatment, and she usually advises patients if they are at risk.

She says she did not recommend or document swabbing the mother as she did not consider her to be at high risk for developing a GBS infection.

At the 4 January appointment the mother signed a consent form for a water birth acknowledging the associated risks including neonatal and maternal sepsis.

Management of the labour and delivery

On 17 February 2016, the mother noted reduced foetal movements and began to develop contractions. She phoned Dr M that morning advising she had recently presented to hospital for a CTG given concerns about the baby's reduced movements. She had previously presented to the regional private hospital on 15 February at which time CTG monitoring and vaginal examination were reassuring. She was advised to return to hospital if she remained concerned about reduced foetal movements.

Dr M arranged for the mother to come in for review that afternoon.

The mother told Dr M she had noticed decreased and less prominent foetal movements and had experienced on and off contractions over the previous two days. She reported having lost the mucous plug. On examination the cervix was very thin and was 2cm fully effaced. Dr M did not notice any meconium liquor. A bedside ultrasound scan was reassuring. She says she advised the mother to have a formal scan the following day if she had not gone into labour that night.

Dr M recalls they again discussed the mother's preference for a water birth. She says she advised her it was likely she was in early labour and it was also likely she would present to hospital in advanced labour that evening.

The mother presented to the regional private hospital at around 8:30am the next day, 18 February, reporting that her waters had broken at around 6:30am. Dr M saw her shortly after she was admitted, noting she had some mild contractions but otherwise her observations were normal. On admission, the midwife noted a finding of a possible slight meconium stain on the pad but on examination of the stain, Dr M noted it was clear and considered it was most likely mucous. CTG monitoring was reassuring.

Dr M says she discussed the options of conservative management with premature rupture of membranes (PROM) and active management with intravenous syntocinon and continuous CTG monitoring. She says she advised The mother the conservative management option would involve waiting for events to unfold at home while monitoring her temperatures, with a requirement to re-present to hospital if her temperature was over 37.5 degrees Celsius, if she noticed a change in the colour of the liquor, had an increase in pain, or if she had any other concerns. The mother indicated her preference for conservative management and she decided to go home and wait. She left the hospital at around 9:15am.

Dr M says she did not order a specimen collection for GBS detection as there were no maternal risk factors for antenatal screening.

At 12:40pm, the mother phoned the Maternity Unit advising she was having contractions lasting approximately 45-60 seconds. She was advised to return to hospital. She arrived at 1:20pm in well-established labour. At 2:40pm her contractions became more intense.

The midwife notified Dr M who asked to be notified if the mother's temperature rose above 37.5 degrees Celsius.

The mother maintained her preference for a water birth. She laboured using the shower, birth ball and bath. She entered the bath at around 3:00pm. The water temperature was 37 degrees Celsius. The mother's temperature was recorded as 36 degrees Celsius. The foetal heart rate was recorded as 150 beats per minute.

At 3:50pm the mother's temperature was recorded as 36.7 degrees Celsius. She exhausted quickly with the pain of contractions and slow dilation and left the bath at 4:10pm. The midwife left a voicemail message for Dr M at this time advising the mother was 3cm dilated and had requested an epidural.

A blood test was taken at 4:30pm.

Dr M gave a telephone order for an epidural after 4:30pm. It was in place by 5:00pm. Post epidural insertion there was an increase in the baseline foetal heart rate and reduced variability noted on CTG. This was initially thought to be related to the insertion of the epidural. The baseline remained elevated prompting review by the Clinical Nurse Manager who recommended that Dr M be notified. At 6:15pm, the mother was tachycardic (100 beats per minute) and her temperature was noted to be 37.5 degrees Celsius. Dr M describes this as normal post-epidural.

Dr M reviewed the mother between 6:15pm – 6:30pm. She noted the baseline of 160 beats per minute with variable accelerations, nil decelerations and reported uncomplicated tachycardia. The mother was now 9cm dilated. Dr M's plan was to reassess her in one hour and to commence active pushing if she was still feeling pressure.

Over the following hour the mother was feeling rectal pressure with contractions, the foetal heart rate remained at 160 beats per minute with an improvement in variability. At 7:00pm she was given a fluid bolus as the CTG was noted to again be having decreased variability and increased baseline to 170 beats per minute. The mother was noted to be febrile with a temperature of 37.6 degrees Celsius at 7:00pm and was given ice chips and Panadol at 7:13pm. Both the Team Leader and Dr M reviewed the CTG again at this time.

The mother was fully dilated at 7:30pm and commenced active pushing at 7:35pm. The midwife phoned Dr M at 8:15pm to advise the mother had been pushing for 50 minutes. Dr M arrived at the birthing suite at around 8:50pm. After discussion of delivery options, the mother asked for a vacuum extraction due to maternal exhaustion. The vacuum was applied at 9:20pm and after two pulls, Baby L was born at 9:22pm.

Baby L was born 'stunned' but picked up rapidly within a minute and did not require any resuscitation. Her APGAR scores were 9/10 at one minute and 9/10 at five minutes. She weighed 3870g, with head circumference of 34.5cm and length of 54.5cm. Her growth had been normal in utero. A neonatal examination conducted by the midwife was otherwise normal and she received vitamin K and a hepatitis B vaccination.

The mother's temperature immediately post-delivery was recorded as 37.4 degrees Celsius at 9:27pm and peaked at 38.6 degrees Celsius at 9:35pm. Dr M was informed and asked to be notified of any further temperatures over 38 degrees Celsius.

Baby L was noted as being 'hot to touch at birth' and her temperature was recorded as 39.5 degrees Celsius at 9:35pm. This finding was not escalated to a paediatrician for review at that time. When rechecked by a midwife at 10:10pm, Baby L's temperature was recorded as 37.6 degrees Celsius. It appears no paediatrician was informed that evening of Baby L's delivery or of her elevated temperatures.

The mother's temperature was recorded as 37.6 degrees Celsius at around 10:30pm.

She and Baby L were handed over to the night staff at 11:00pm.

When checked again at 11:45pm, the mother's temperature was 36.5 degrees Celsius. She and Baby L remained under observation and as at 1:00am, it was noted they were both afebrile.

The mother's blood test results returned that evening showing a high white cell count (26,000) with a significant neutrophilia (21,000).

Baby L's post-natal management and discharge home

Baby L was examined by the on call paediatrician, Dr S, at around 10:00am the following morning. Dr S subsequently explained that the medical record contained a form titled 'Uniting Hospital Paediatric Checklist', which is a form partially completed by the midwives looking after the mother and baby. There are boxes at the bottom of the form to be completed by the reviewing paediatrician at the initial post-natal examination and again prior to discharge. Her entries on this checklist were the only entries she made in Baby L's medical record.

Dr S noted there was maternal and baby temperature at birth and that the mother felt unwell. She also noted '*?no paediatrician notified at time of birth'.*

Dr S noted Baby L's observations since birth were all normal and ordered continued three-hourly observations for the following 24 hours. Dr S subsequently explained she ordered this purely as a precautionary measure given the raised maternal and neonatal temperatures immediately after birth.

Dr S says that had she been made aware of the maternal and neonatal temperatures immediately after birth, she would have reviewed Baby L and at least ordered a full blood count and would likely also have taken a blood culture and commenced antibiotics, as recommended by then current Queensland Clinical Guidelines (https://www.health.qld.gov.au/qcg/publications#maternity). Dr S advised that

maternal temperature can be a sign of chorioamnionitis, but there are other causes including epidural anaesthesia which the mother had during labour.

However, at the time Dr S reviewed Baby L on 19 February, Baby L was 12 hours old and other than the elevated temperature immediately after birth, she had remained well. On this basis, and in the absence of any further temperatures or abnormal physiological parameters, she decided sepsis was unlikely.

The progress notes contain entries indicating that Baby L's observations remained within normal limits during her admission. While the hospital used a combined a Neonatal Feeding and Observation Chart at the time of Baby L's admission, this chart was missing from the medical records and has since been unable to be located.

Dr S says her intention was to investigate further if there were any ongoing concerns, but all of Baby L's subsequent observations remained normal.

The mother and Baby L remained in hospital for three days. The nursing notes and statements provided by the midwives involved in Baby L's care showed some slowness in establishing feeds on her first day of life which was managed by the midwives in consultation with Dr M. By the time of discharge home on 22 February, Baby L had begun to regain weight after an initial loss. Her discharge weight was 3670g.

During her admission the mother was charted and given Panadol and Voltaren for pain relief as follows:

- Panadol 1g twice a day on 19, 20 & 22 February and once daily on 21 February.
- Voltaren 50mg twice a day on 19 February and daily in the morning on 20, 21 and 22 February.

Apart from one temperature of 38.6 degrees Celsius charted at 9:35pm post- delivery, the mother did not have any further temperatures.

Dr M reviewed the mother before her discharge home at 12:30pm on 22 February 2016. A post-natal check-up was planned for six weeks' time.

Baby L was reviewed by another paediatrician, Dr Sc, at 11:15am and was noted to be well and alert, and feeding well. There were no documented concerns.

Events following Baby L's discharge home

A hospital midwife contacted the mother on 29 February for a routine postnatal discharge check. The mother reported Baby L was feeding well every three hours and had a very settled sleep pattern.

Baby L continued to feed well and behave normally until 2 March 2016, day 13 of life, when she developed loose stools and her feeding pattern began to change. She had previously been on an approximately two-hourly breast feeding cycle but on 2 March became irritable and was fed more frequently, up to hourly.

Baby L stopped feeding during the night of 2-3 March. She was given colic drops overnight to alleviate her irritability. She developed a high pitched cry. She presented

to a small regional public hospital emergency department early on 3 March. There were no obvious signs of viral infection and no recent history of fever. Her last wet nappy was approximately 11 hours prior to presenting to hospital.

She was immediately recognised as being unwell and probably septic. She was noted to be irritable, unsettled and markedly tachycardic (195 beats per minute). Her oxygen saturation was good. There was some suspicion of abdominal tenderness leading the treating team to query gastroenteritis. She was noted to be responsive with no marked neurological changes. Blood gas measurement showed no hypoxia or any respiratory or metabolic acidosis. Arrangements were made to transfer her to a large regional public hospital for further management. Before transfer she was appropriately commenced on intravenous fluids and intravenous antibiotics (cefotaxime and ampicillin) after blood cultures were taken.

On arrival at the large regional public hospital Baby L was noted to be unwell, pale, mottled and very irritable, with a high pitched cry and 'grunting' respiration. Her capillary refill time was mildly increased, her peripheries cool and her heart rate was 200 beats per minute. She was responsive to stimuli, her grasp and suck reflexes were intact and her pupils were reactive. Her fontanelle was noted to be full. She was floppy and had reduced spontaneous movements.

CT imaging of the head and abdomen were normal. She was intubated and ventilated and required inotrope therapy to manage her cardiovascular instability. At about this time she was noted to have a purpuric eruption in the nappy area and on her upper thighs, consistent with severe sepsis. Lumbar puncture was not performed at this time due to concerns about Baby L's cardiovascular instability and her intracranial pressure. Her intravenous therapy continued with fluids and the addition of gentamicin (antibiotic), acyclovir (anti-viral) and hydrocortisone.

Arrangements were made to transfer Baby L to the Lady Cilento Children's Hospital for tertiary management. There was some initial delay due to ambulance availability. However, she had already been commenced on appropriate antibiotic therapy and her condition remained relatively stable during transfer.

Blood cultures taken at the small regional public hospital grew a gram positive coccus, subsequently identified as Group B Streptococcus (Strep Agalactiae) – this was evident within 8-9 hours, suggesting a significant bacteraemia. Culture of maternal breast milk did not show any evidence of Group B Streptococcus.

Baby L was admitted to the Lady Cilento Hospital where it was established she had developed sepsis with Group B Streptococcal infection. MRI scan of the brain performed on 5 March showed severe cerebritis, cerebellitis and meningitis with significant cerebral oedema due to aqueduct stenosis (blockage of a portal of cerebrospinal fluid within the brain causing backpressure and increased brain swelling). She was reviewed by a paediatric neurologist that evening who considered Baby L was unlikely to survive and even if she did, there was a very high likelihood of severe and permanent brain injury. Following extensive discussions with Baby L's parents, it was decided to transition her from active treatment to palliative comfort cares. She was extubated late in the evening of 6 March and died early on the morning of 7 March 2016.

Preliminary independent clinical review

Preliminary independent clinical review undertaken by an independent doctor from the Department of Health Clinical Forensic Medicine Unit queried whether the maternal and neonatal fevers were appropriately investigated and managed at the regional private hospital.

The reviewing doctor had no concerns about Baby L's management at the small regional public hospital, the large regional public hospital and the Lady Cilento Children's Hospital, describing her assessment and treatment at the regional hospitals as 'swift and appropriate'.

The reviewing doctor was not concerned about the delay in ambulance transfer to Lady Cilento Children's Hospital as Baby L had already been commenced on appropriate antibiotic treatment and intravenous fluids.

Having considered these concerns, I determined that Baby L's death may be health care related and required further coronial investigation, including post-mortem examination.

Autopsy findings

An external examination and review of the medical records was performed at the John Tonge Centre on 10 March 2016. Having regard to the clinical history and post-mortem radiological findings, the pathologist considered the cause of death to be Group B Streptococcus meningitis and sepsis.

Regional private hospital clinical review outcomes

The regional private hospital appropriately commissioned a comprehensive investigation, including a Root Cause Analysis (RCA), following Baby L's death. RCA is a systemic analysis of what happened and why and is designed to make recommendations to prevent adverse health outcomes from happening again, rather than to apportion blame or determine liability or investigate an individual clinician's professional competence. It is conducted by a review team who had no involvement in the patient's care. I received the final RCA report on 30 June 2016.

The regional private hospital also commissioned expert opinions from an independent obstetrician (Dr Robert Ford), paediatrician (Dr David Slaughter) and paediatric infectious diseases consultant (Dr Michael Nissen).

Having regard to these expert opinions, the RCA Team concluded that Baby L died from late onset GBS, an outcome considered unable to have been predicted or managed during her admission at the regional private hospital. This was on the basis of clinical literature which overwhelmingly confirms late onset GBS is not preventable or predictable.

The collective opinion of Drs Ford, Slaughter and Nissen was that the mother and Baby L were managed appropriately and consistent with the Queensland Maternity and Neonatal Clinical Guideline for Early Onset Group B Streptococcal disease and related Royal Australian and New Zealand College of Obstetricians and Gynaecologists guidelines for Maternal Group B Streptococcus in Pregnancy:

Screening and management**.**

While the RCA team was satisfied Baby L's death from late onset GBS could not have been prevented, it identified a not inconsiderable nor insignificant range of opportunities to improve maternity systems and processes at the regional private hospital.

Most significantly, the regional private hospital acknowledged that Baby L should have been escalated for paediatric review sooner even though it considers this would not have changed the outcome for her. As a result, the regional private hospital has revised its Newborn Care policy to require:

- all neonates to be examined by an accredited paediatrician with the first 24 hours of life; and
- babies with any signs of respiratory distress, hypoglycaemia or who are febrile at birth to be referred for immediate paediatrician review, with the referral to be documented in the progress notes contemporaneously.

I consider the RCA findings and recommendations are worth summarising given their potential significance in contributing to adverse patient outcomes, particularly had Baby L died from early onset GBS, as opposed to late onset GBS (as the team concluded).

The RCA team identified the following issues arising from the management of the mother's labour and delivery and Baby L's post-natal care:

1. Lack of full appreciation that a febrile mother and foetal tachycardia in labour is a variance from 'well' normal pathway.

The RCA team identified a constellation of factors which it considered may have contributed to the treating team's failure to recognise maternal fever and consistent foetal tachycardia as a variance to normal care, including:

- lack of prescribed standard operating procedures to drive prescribing of antibiotics for a low grade fever and foetal tachycardia in labour;
- lack of clarity around requirements to complete a full patient assessment and document that assessment and actions;
- no hospital requirement for obstetricians to test all obstetric patients and report GBS results to the patient and hospital;
- the overall assessment process while working in a 'wellness model';
- infrequent exposure of midwives to an unwell mother in birth suite.

To address these issues, the RCA team recommended a standardised system of assessment, escalation and management of variances from 'well' normal care that requires assessment by a midwife/ obstetrician and appropriate documentation; provides information to patients and carers about the process of normal labour and what is not normal; requires clinical staff involved to ensure timely escalation of concerns to the relevant Visiting Medical Practitioner; a new electronic CTG partogram system with trigger alerts for maternal temperature and foetal tachycardia and introduction of Medical Early Warning Trigger System (MEWTS) documentation for all obstetric patients.

This recommendation required review of then current clinical policies regarding the management of a woman in labour, warm water immersion during labour and birth, intrapartum foetal surveillance and newborn care.

2. Lack of prescribed requirements for admitting obstetricians to have fulfilled a GBS assessment antenatally prior to admission.

The RCA team identified that the absence of standard operating procedures had led to an ad hoc system in which:

- individual Visiting Medical Practitioners were independently managing GBS assessments antenatally;
- maternity ward staff did not have specific guidelines to follow when no antenatal screening has been conducted in the admitting obstetrician's rooms;
- there was no formal requirement for obstetricians to perform antenatal screening on all pregnant women.

This 'system' was identified as potentially creating an inability to identify if a patient was GBS positive and consequently may have contributed to a missed opportunity for appropriate management.

To address this the RCA team recommended:

- developing a standardised system of GBS assessment and management, with reference to 'obstetric guidelines' not only for the regional private hospital but also for consideration across affiliated hospitals providing maternity services;
- Craft Group consideration of implementing gastric aspirates on all neonates who are febrile at birth and where there is a history of maternal fever during labour and foetal tachycardia during labour;
- considering standard operating procedures for an initial dose of antibiotics for the newborn pending gastric aspirate results;
- ensuring infections are reported and monitored by the Infection Control Clinical Nurse Consultant.

3. Disjointed process of escalation and handover between and within nursing and medical teams.

The RCA team identified there was:

- no formal process of referral and escalation to paediatricians when high risk babies are born;
- a need to develop specific management guidelines for neonates who are febrile at birth and have had foetal tachycardia during labour;
- the need for a tool to determine escalation of unwell babies for paediatric review.

It considered this situation may have increased the likelihood of fragmented and disjointed midwifery and medical handovers which may have contributed to the events of the labour and post-delivery not being handed over sufficiently to all staff caring for the mother and Baby L on subsequent shifts and days.

To address this, the RCA team recommended:

- reviewing the then current clinical handover model using the SHARED

framework for midwifery staff, incorporating consideration of implementing a Medical Early Warning Trigger System for both mother and baby; implementing a system to empower midwives to notify paediatricians of all babies whose observations are not within normal limits and requiring Team Leaders to have face to face bedside handover to discuss all women in labour and high risk/deviation from normal cases; and

- reviewing the then current structure of clinical handover model for medical staff to reduce fragmentation of multiple medical staff being involved with particular patients.

4. Lack of appropriate documentation in response to known adverse event.

The RCA team identified a constellation of factors which it considered led to a failure by oncoming doctors and nursing staff to recognise the clinical picture given the adverse events during labour and clinical manifestations including that Baby L was febrile post-delivery, including:

- a lack of standardised operating procedures for management of a febrile baby;
- a lack of knowledge about Medical Emergency Team criteria;
- a general acceptance by clinical staff that a MET call does not need to be enacted when the patient is being reviewed by an attending doctor or to escalate to a MET call in the presence of the attending doctor when the patient continues to be symptomatic and looks unwell;
- lack of evidence in the medical record on review of neonatal observations and feeding while an inpatient;
- a lack of appreciation of the benefits of integrated notes.

This was considered to increase the likelihood of a lack of action to address a clinically deteriorating patient in an appropriate and timely manner which may have contributed to the overall assessment, management and documentation of the febrile neonate.

To address this, the RCA team recommended:

- implementing an Early Warning System tool in the maternity services of the regional private hospital and its affiliates;
- including neonates on the MET criteria list;
- implementing objective decision support aids to reinforce the need to activate MET;
- reviewing the then current feeding and observation chart and implementing the feeding chart as a separate document;
- developing a take home booklet with feeding guidelines and log for parents to continue to document feeding and baby output;
- reinforcing the importance of integrated notes to create a clinical picture of events occurring on previous shifts

The regional private hospital has since implemented strategies to reduce the risk of observation charts being misplaced and a subsequent audit of baby feeding charts demonstrated 100% compliance. The loss of Baby L's feeding and observation chart was escalated across the maternity services of the regional private hospital and its affiliates to raise awareness of this risk. These discussions identified the need to develop a separate feeding and observation document in the longer term.

As at 30 June 2016, the regional private hospital was intending to trial the NSW

Standard Newborn Observation Chart and this was under discussion at it major affiliate hospital in Brisbane. I am advised a new Neonatal Feeding Chart and Neonatal Observation Form (with clinical escalation triggers) were being trialed at the regional private hospital as from 31 October 2016.

5. Lack of recognition to the potential problems associated with breast feeding post-breast feeding augmentation.

This issue, though not relevant to the issues examined by my investigation, led the RCA team to recommend a standardised system for follow up with mothers who are discharged breast feeding post-breast feeding augmentation.

The regional private hospital advised that as at 4 November 2016, it was implementing the RCA recommendations in full.

Independent neonatology review and opinion

I arranged for Dr Andrew Watkins, consultant neonatologist at Mercy Hospital for Women in Melbourne, to provide an opinion about the nature of Baby L's illness and the extent to which it may have been preventable. Dr Watkins is recognised by his peers as having expertise in neonatal intensive care, neonatal transport and perinatal palliative care and counselling. As such he has extensive experience in the prevention and management of infection in neonates.

Dr Watkins concluded that Baby L died from late onset Group B Streptococcal meningitis. He considered her presentation was typical of late onset meningitis against a background of having been well in the period following discharge home from hospital. There is no doubt Baby L had developed a Group B Streptococcal septicaemia. The MRI findings were wholly characteristic of severe meningitis with encephalitis and cerebellitis.

He advised that the early signs of septicaemia and meningitis in infants are very non-specific, most commonly reduced responsiveness and a change in feeding behaviour. He noted that the time from first symptoms to presentation was approximately 12 hours with Baby L showing predominantly irritability and changed feeding behaviour. He explained this duration of symptoms is commonly seen and there was no undue delay in Baby L being presented to hospital as it does take some time for a pattern to emerge and to distinguish itself from normal behavioural variability.

Dr Watkins confirmed that late onset Group B Streptococcal disease is not affected by perinatal management and antibiotic treatment, whether intrapartum or immediately post-partum, would not have prevented Baby L's death. As such he was satisfied that the management of the labour and immediately after birth did not contribute to Baby L's death.

However, Dr Watkins described this aspect of the care provided to the mother and Baby L as suboptimal in that there was evidence strongly suggestive of chorioamnionitis, namely maternal fever and tachycardia, maternal malaise, foetal tachycardia and early fever in Baby L, all of which were noted at the time.

Dr Watkins considered the fever and tachycardia emerged so late in labour there was

little opportunity for intrapartum treatment, which would not in any case have changed the outcome for Baby L. The maternal chorioamnionitis appeared to have resolved with delivery of the baby and placenta and membranes, and to have received no treatment.

He advised that Baby L was nonetheless at risk and should have been assessed by a paediatrician at birth. He considered it would have been appropriate to have undertaken basic screening investigations and to have considered blood cultures. He acknowledged that some paediatricians would have treated with antibiotics, but close observation would also have been acceptable. He considered this to have been a departure from the Queensland Maternity and Neonatal Clinical Guideline for Early Onset Group B Streptococcal disease as maternal fever, whether intrapartum or postpartum is an indication for assessment of the baby, as is infant fever. However, he was very clear this was not relevant to Baby L's death given she died from late onset GBS which cannot be eliminated by intrapartum or postpartum treatment with antibiotics such as penicillin.

Dr Watkins provided a very helpful explanation of the reason for the ineffectiveness of intrapartum or postpartum antibiotic treatment in preventing last onset GBS:

- In early onset GBS, the baby receives a large inoculum of organisms from the maternal genital tract during birth causing a fulminant septicaemic illness which characteristically progresses from babies being initially well but then collapsing with severe illness, often over only 2-6 hours.

- Intrapartum treatment of the mother, if administered appropriately, causes the baby to have an antibiotic level in the bloodstream at the time of initial inoculum, either preventing or ameliorating the disease. It has been shown to significantly reduce mortality from early onset sepsis.

- In contrast, late onset GBS is acquired by colonisation at birth, by infection from maternal flora or breast milk, by nosocomial spread or sometimes by contact from others. The essential difference is that the sequence is of colonisation of one individual, these organisms then colonising the baby and ultimately causing infection in a percentage of those colonised.

Between 10-30% of pregnant mothers are colonised with GBS, it being principally carried in the bowel, this being reflected in lower genital tract colonisation. Colonisation is not infection - the reason why a colonising organism may change to becoming an infecting organism are not fully understood.

Antibiotic treatment has been shown to be generally ineffective in eliminating colonisation; the most it may achieve being the overgrowth of resistant organisms. Consequently it is not possible to eliminate passage of GBS from maternal flora to the baby, as this passage is a normal event and is generally of benefit to the baby ensuring the baby becomes colonised with a relatively benign flora. This passage is reinforced by ongoing exposure to the mother in both general contact and through breast milk.

The fact of maternal Group B Streptococcal carriage also makes it very likely there will be ongoing contact with GBS from the mother in other ways. For this reason, the baby

remains susceptible to infection and little can be done in our current state of clinical knowledge, beyond attention to detection of early signs, to avoid late onset GBS mortality.

Dr Watkins considered the question of whether Baby L's infection may have occurred at birth and progressed indolently. He noted a small number of case reports of infants presenting late with GBS ventriculitis or meningoencephalitis in which it is clear the child had been unwell for some weeks. In these cases, there is a history of a child who has been unwell, though not massively so, since birth. Dr Watkins was satisfied Baby L did not behave this way, feeding and gaining weight well, with normal responsiveness and behaviour until the 12 or so hours before she presented to the small regional public hospital. Further, the central nervous system changes seen in Baby L were not suggestive of chronic changes, in that she had no evidence of ventriculitis, no hydrocephalus and no cystic changes which take some time to develop, but rather the changes seen on MRI scan were characteristic of recent severe insult. On this basis, Dr Watkins considered it highly unlikely that Baby L had developed infection from the time of birth.

Dr Watkins observed that the likely course of GBS infection was from the mother who was very likely to have been colonised with GBS at the time of birth. Other sources cannot be excluded but are very unlikely.

Dr Watkins noted that the mother was not swabbed for GBS during pregnancy and reinforced his view that Baby L's death was unrelated to issues of antenatal GBS screening or risk factor assessment. On this issue, Dr Watkins commented that the Queensland Maternity and Neonatal Clinical Guidelines for Early Onset Group B Streptococcal disease are a good statement of the clinical evidence, representing a credible and responsible approach to the prevention of early onset GBS. He considered the quality of the work undertaken in the development of the Queensland guidelines and the quality of the clinical taskforce behind them give strong grounds for confidence that any future evolutionary change will be evidence based and will reflect good practice, whatever disagreement any individual may have with particular aspects of them.

Dr Watkins was satisfied that although Baby L's diagnosis was not made immediately on initial presentation to the small regional public hospital, the clinical story having been muddied by a history of some diarrhoeal bowel actions, she was recognised as unwell and managed appropriately, with little delay in commencing antibiotic therapy. Her management at the large regional public hospital, during transfer by ambulance to Brisbane and then at Lady Cilento Children's Hospital was all appropriate, as was the ultimate decision to cease active treatment.

Dr Watkins considered the only way to reduce morbidity and mortality with late onset GBS is a focus on early detection of the signs of illness in the infant. This can be difficult, as signs are subtle and overlap some normal infant behaviour. A consistent change in feeding performance, alertness or responsiveness are important danger signs. While this may have been helped by a knowledge of maternal GBS status, Dr Watkins identified the importance of parents being equipped to detect early signs of infection, coupled with the confidence to bring their concerns to medical attention without the fear of being stigmatised as a 'panicky mum'. As such, he considered the

essential factors to be education and awareness, and suggested standardised scoring systems such as the Baby Check system may be a useful adjunct to parent education and may lead to earlier presentation and better outcomes.

Findings required by s. 45 *Coroners Act 2003*

Identity of the deceased: Baby L

How she died: Baby L died from natural causes. Although aspects of the management of the mother's labour and delivery and Baby L's post-natal care were suboptimal, I am satisfied that these deficiencies did not cause or contribute to Baby L's death from late onset Group B Streptococcus infection. The regional private hospital has identified the need for some significant changes to its maternity systems and processes and has demonstrated equally significant efforts to improve those issues. There is nothing Baby L's parents could have done to have changed the outcome for her as she died from a condition that carries a high mortality rate and which is currently unpreventable.

Place of death: Lady Cilento Children's Hospital, South Brisbane

Date of death: 7 March 2016

Cause of death:

1(a) Main disease in neonate: Group B Streptococcus meningitis and sepsis
1(b) Other diseases in neonate: Nil identified
1(c) Main maternal disease affecting neonate: Nil identified
1(d) Other maternal diseases affecting neonate: Nil identified

2 Other relevant circumstances: Nil

Underlying cause of death: Group B Streptococcus meningitis and sepsis

Ainslie Kirkegaard
Coronial Registrar
Brisbane
9 June 2017