OFFICE OF THE STATE CORONER

FINDINGS OF INQUEST

CITATION:	**Inquest into the death of Oliver Steven McVey**
TITLE OF COURT:	Coroners Court
JURISDICTION:	Brisbane
FILE NO(s):	COR 1172/06(9)
DELIVERED ON:	28 November 2008
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HEARING DATE(s):	21 – 25 July, 24 & 25 September 2008
FINDINGS OF:	Ms Christine Clements, Deputy State Coroner
CATCHWORDS:	CORONERS: Inquest – premature second twin, patent ductus arteriosus, carbon dioxide build up when ventilated, appropriate ventilation modes and machine, problems with re-intubation

REPRESENTATION:

Ms Julie Wilson – appearing to assist the Coroner

Mrs Shannon McVey – representing the family

Mr DK Boddice SC with Mr R Ashton – representing Mater Misericordiae Health Service instructed by Minter Ellison Lawyers

Ms J Rosengren – representing Dr Andrew Clarke instructed by Thynne and Macartney Lawyers

CORONERS FINDINGS AND DECISION

The *Coroners Act 2003* provides in s45 that when an inquest is held into a death, the coroner's written findings must be given to the family of the person who died and to each of the persons or organisations granted leave to appear at the inquest. These are my findings in relation to the death of Oliver Steven McVey. They will be distributed in accordance with the requirements of the Act and placed on the website of the Office of the State Coroner.

The Coroner's jurisdiction

Before turning to the evidence, I will say something about the nature of the coronial jurisdiction.

The scope of the Coroner's inquiry and findings

A coroner has jurisdiction to inquire into the cause and the circumstances of a reportable death. If possible he/she is required to find:-

- whether a death in fact happened;
- the identity of the deceased;
- when, where and how the death occurred; and
- what caused the person to die.

There has been considerable litigation concerning the extent of a coroner's jurisdiction to inquire into the circumstances of a death. The authorities clearly establish that the scope of an inquest goes beyond merely establishing the medical cause of death.

An inquest is not a trial between opposing parties but an inquiry into the death. In a leading English case it was described in this way:-

> *"It is an inquisitorial process, a process of investigation quite unlike a criminal trial where the prosecutor accuses and the accused defends… The function of an inquest is to seek out and record as many of the facts concerning the death as the public interest requires."* [1]

The focus is on discovering what happened, not on ascribing guilt, attributing blame or apportioning liability. The purpose is to inform the family and the public of how the death occurred with a view to reducing the likelihood of similar deaths. As a result, the Act authorises a coroner to make preventive recommendations concerning public health or safety, the administration of justice or ways to prevent deaths from happening in similar circumstances in the future.[2] However, a coroner must not include in the findings or any comments or recommendations, statements that a person is or maybe guilty of an offence or is or maybe civilly liable for something.[3]

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[1] *R v South London Coroner; ex parte Thompson* (1982) 126 S.J. 625
[2] s46
[3] s45(5) and 46(3)

The admissibility of evidence and the standard of proof

Proceedings in a coroner's court are not bound by the rules of evidence because section 37 of the Act provides that the court "*may inform itself in any way it considers appropriate."* That doesn't mean that any and every piece of information however unreliable will be admitted into evidence and acted upon. However, it does give a coroner greater scope to receive information that may not be admissible in other proceedings and to have regard to its provenance when determining what weight should be given to the information.

This flexibility has been explained as a consequence of an inquest being a fact-finding exercise rather than a means of apportioning guilt: an inquiry rather than a trial.[4]

A coroner should apply the civil standard of proof, namely the balance of probabilities but the approach referred to as the *Briginshaw* sliding scale is applicable.[5] This means that the more significant the issue to be determined, the more serious an allegation or the more inherently unlikely an occurrence, the clearer and more persuasive the evidence needed for the trier of fact to be sufficiently satisfied that it has been proven to the civil standard.[6]

It is also clear that a Coroner is obliged to comply with the rules of natural justice and to act judicially.[7] This means that no findings adverse to the interest of any party may be made without that party first being given a right to be heard in opposition to that finding. As *Annetts v McCann*[8] makes clear that includes being given an opportunity to make submissions against findings that might be damaging to the reputation of any individual or organisation.

Background

Oliver Steven McVey (Oliver) died at the Mater Mothers' Hospital on 15 April 2006. Oliver had been hospitalised since birth. He was the second twin, born prematurely by caesarean section, to Shannon and Steven McVey on 7 February 2006. He and his brother Hamish were born at 31 weeks and 1 day gestation. Oliver weighed 1036 grams at birth.

A rotation of teams of doctors and nurses, each working with a consultant were involved in Oliver's care in the hospital.[9] Consultants were also on

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[4] *R v South London Coroner; ex parte Thompson* per Lord Lane CJ, (1982) 126 S.J. 625
[5] *Anderson v Blashki* [1993] 2 VR 89 at 96 per Gobbo J
[6] *Briginshaw v Briginshaw* (1938) 60 CLR 336 at 361 per Sir Owen Dixon J
[7] *Harmsworth v State Coroner* [1989] VR 989 at 994 and see a useful discussion of the issue in Freckelton I., "Inquest Law" in *The inquest handbook*, Selby H., Federation Press, 1998 at 13
[8] (1990) 65 ALJR 167 at 168
[9] Consultant neonatologists caring for Oliver

- A/ Prof Peter Gray 1- 19 February 2006
- Prof David Tudehope 1-19 February 2006
- Dr Paul Woodgate 7 February- 12 February 2006
- Dr Helen Liley 20 February – 3 March 2006
- Prof David Tudehope 6-19 March 2006
- Dr Paul Woodgate 20 March – 2 April 2006
- Dr Helen Liley 3 April- 15 April 2006

Registrars involved in caring for Oliver

- Dr Swaminathan, Dr McGill, Dr Zankl, Dr Arreza, Dr Deo

rotational call during the night. After Oliver's death, Dr David Cartwright, Director of Neonatology at the Royal Brisbane and Women's Hospital, reviewed the medical records and provided his expert opinion.[10] Dr Richard Chard, Deputy Director of the Adolph Brasser Cardiac Institute at the Children's Hospital Westmead, similarly provided his expert opinion.[11]

On 18 January 2006 a cardiologist, Dr Cameron Ward reviewed Shannon McVey. Mrs McVey's obstetrician in the United Kingdom had expressed concern there may be a problem with the left side of the heart (a coarctation) of one of the twins. An echocardiogram was performed and Dr Ward considered there was no sign of a cardiac abnormality. He therefore did not consider a further echocardiogram during the pregnancy or after birth was necessary.

Oliver weighed 1036 grams at birth. He was well below the 5th percentile for gestation, meaning he was much smaller than most babies of his gestation.[12] It was due to Oliver's intrauterine growth restriction that the twins were delivered at this time. The main concern after birth was prematurity.

Both babies were initially cared for in the intensive care unit. Oliver was on constant positive airway pressure ("CPAP") for a couple of days before being placed in special care nursery 2 ("SCN2") and then progressing to special care nursery 1 ("SCN1"). Baby Hamish was on CPAP for about 10 days before being transferred to SCN2 and then SCN1. Babies in special care nurseries were reviewed daily on a ward round, although this did not necessarily include a physical examination.[13]

Because the possibility of a diagnosis of coarctation of the aorta was raised antenatally,[14]the McVeys' initial concerns for Oliver were about the condition of his heart His pulses were assessed regularly in SCN2. The condition of mild hypospadias[15] was diagnosed and was to be treated surgically sometime later. A sacral dimple was noted but was not a cause for concern.

Oliver did not require any further respiratory support until the surgery was performed. He was fed through a nasogastric tube as well as a period of about one week when he received intravenous nutrition. Caffeine was administered from day 7 to stimulate his breathing after he exhibited apnoea. Apnoea is an expected condition in a premature baby born before 33 weeks. By day 30 the caffeine levels were increased due to further episodes of desaturation. His mother had commenced some breast feeding.

[10] Exhibit E1
[11] Exhibit E2
[12] Dr Liley states this weight was well below the 5th centile but Professor Tudehope's evidence was Oliver's birth weight was 300 gms below the 3rd centile. See T2, p.53, Ll34-36
[13] T2, p.45, L25 (Professor Tudehope)
[14] Coarctation or stenosis of the aorta is a narrowing of the lumen of the aorta due to congenital maldevelopment. The site is usually at the isthmus and just above the ductus arteriosus.
[15] Hypospadiasis a congenital defect of the wall of the male urethra so that instead of the normal external orifice there is an opening for a greater or lesser distance on the under side of the penis.

The first notation recording a suspected heart murmur was on 15 March 2006 when Oliver was about 35 days old. This observation was accompanied by "*very good femoral and brachial pulses*".[16] This observation was recorded in the context of previously suspected coarctation. However, a strong pulse is not expected in such a condition, whereas it is in the condition of patent ductus arteriosus.[17] The respiratory rate of 60 breaths per minute was high. Professor Tudehope considered the possible explanation of it being a haemodynamic flow murmur associated with anaemia. He directed monitoring of the haematocrit levels.

On the following day Oliver's weight was noted to have increased by 300 grams during the week. Professor Tudehope considered this may be fluid retention or overload and ordered Lasix (diuretic medication) to increase the rate of fluid excretion in conjunction with restricted fluid. Professor Tudehope concurred on review with Dr Cartwright's opinion that weight gain in these circumstances can also be a sign of cardiac failure. Initially the fluid restriction improved Oliver's condition and on 17 March the notation was made "*no echo for now*".[18]

On 18 March, Oliver was noted as having a right inguinal hernia and arrangements for its surgical repair were to be made.

By 19 March there was a notation indicating the need for a chest x ray. Professor Tudehope considered it appropriate to rule out common problems first before considering the possibility of a patent ductus arteriosus ("PDA"). Conditions such as viral pathogens, or mild chronic neonatal lung disease were expected in a premature baby and were far more likely than a PDA becoming symptomatic for the first time.

Diagnosis of Patent Ductus Arteriosus

On 21 March a neonatal registrar (Dr Zankl) was examining Oliver prior to planned surgery for the repair of his hernia. She noted tachypnoea, audible bilateral crackle sounds in the lungs and a heart murmur. This was suggestive of a heart problem and she called upon the consultant Dr Woodgate to review Oliver.

A request was then made for a review by the cardiologist (Dr Ward) around 21 or 22 March as a patent ductus arteriosus (PDA) was suspected.

Dr Ward described a PDA as:

> *"A normal cardiac structure in a foetus essential for normal development but which ordinarily closes in the first few days after birth. While the PDA is a normal structure, persistence beyond the early neo-natal period is abnormal. A ductus arteriosus which persists after birth may require surgical ligation. The problem with a PDA is that it allows additional, potentially excessive, blood flow*

[16] Exhibit C1, p.68, entry 15 March 2006
[17] T2, p.14, Ll41- 43.
[18] Exhibit C1, p.70, entry 17/3/06.

through it into the lungs and may result in rapid or laboured breathing and an inability to feed properly."[19]

Professor Tudehope explained the condition:

"In the foetal circulation the ductus is widely patent to shunt blood away from the lungs because the placenta is acting as the lung in utero. After birth in a full term infant when the baby starts to breathe after birth and the circulation changes, the duct rapidly goes into spasm and then closes anatomically about 4-7 days after birth in a healthy term infant. In a preterm infant, although there may be vasoconstriction of the duct after birth, it frequently opens up again particularly if that preterm infant is exposed to any lack of oxygen or any respiratory difficulties. The likelihood of the duct opening up again is very much related to birth weight and gestational age. The more premature the baby the lower the birth weight the greater the likelihood of that duct opening up again and becoming a so called patent ductus arteriosus, and that duct is a small channel between the aorta and the pulmonary artery. In utero, it's shunting blood from the pulmonary artery to the aorta, but usually when it opens up after birth it shunts blood from the pulmonary artery so that too much blood is flowing to the lungs. And if that duct is allowed to persist for any length of time the baby may well go into cardiac failure and become symptomatic from the ductus."[20]

How long it will take the ductus to become symptomatic depends on how much blood is flowing from left to right. In the majority of premature babies, the ductus will vasoconstrict but will not anatomically close until after 14 days. Less commonly, a duct will close and then reopen. There is no clear evidence whether Oliver's ductus was open from birth or whether it closed and then reopened. Once the duct closes it forms scar tissue.

Initial treatment of a PDA can be non-surgical, through the use of medication known as Indomethacin (or Indocid). Indomethacin is a prostaglandin synthesised inhibitor that causes constriction of the duct if the condition is detected in the first few weeks after birth. Medication is not suitable for term babies, is more effective in very young babies (not beyond 34 weeks gestational age) and so surgical ligation is then required to close the ductus. Echocardiography can assist in the early detection of a PDA in susceptible babies. Without this, in order to diagnose a PDA, reliance must be placed on the following triad of clinical signs:

1. an active precordium (feeling the heart beating through the chest);
2. full or bounding pulses; and
3. an audible murmur.[21]

[19] Exhibit B13, paragraph 8,9,10
[20] T2, p.39, Ll21- 41.
[21] T2, p.41, Ll53 - 58.

These symptoms were present and first recorded at about day 35, but interpreted as insignificant and indeed reassuring because they were contrary to the existence of the previously suspected condition of coarctation. In hindsight, Professor Tudehope acknowledged the chest x ray should have been done 2 or 3 days earlier, and indeed prior to fluid restriction occurring. The cardiac consultation could have been ordered a couple of days earlier.[22] He also agreed in retrospect with Dr Cartwright that Oliver was suffering cardiac failure from mid March which was only just under control by 5 April.[23]

On 23 March, Dr Ward assessed Oliver including performance of an echocardiogram and confirmed there was a moderate to large PDA requiring surgical ligation. After the review, he discussed the findings of the echocardiogram and the recommendation for surgery with Dr Andrew Clarke (a cardiac surgeon at the Prince Charles Hospital). In 2006 the Mater Children's Hospital staff did not perform cardiac surgery on babies. These procedures were usually performed by cardiac surgeons from the Prince Charles Hospital. The surgery was initially scheduled for 29 March but was not performed until 13 April 2006.

Issue of delay before PDA surgery

There was a great deal of detail provided to the inquest about the efforts to arrange surgery from the perspectives of the doctors at the Mater Hospital and the surgeon at the Prince Charles Hospital. Dr Liley actively involved herself in trying to assist the process of co-ordinating a date for surgery. In hindsight the mounting anxiety of the parents would have been greatly relieved had they been fully informed of the level of communication occurring to organise the surgery.

There was no problem with the availability of the surgeon, Dr Andrew Clarke and his team. As was the usual practice, Dr Clarke provided numerous dates of his surgical availability. The co-ordination of the surgery (e.g. allocation of the operating theatre and an anaesthetist) occurred at the Mater Hospital end. Dr Clarke and his cardiac surgical team (a surgical registrar and 2 experienced cardiac operating room nurses) would then attend for the surgery.

The surgery was initially booked for 29 March 2006 at the Mater Children's Hospital. The surgery did not go ahead on this date. It seems the difficulty was in organising the attendance of a neonatal anaesthetist and the competing claims of more acutely ill babies.[24]

Dr Clarke was given an alternative of undertaking the surgery that evening as part of the emergency list. Dr Clarke considered but rejected this proposal. This was quite reasonable as there was no set time for the surgery to occur and may have resulted in short staffing at the Prince Charles Hospital due to the requirement for specialist theatre nurses from that hospital to be in attendance for an unknown period of time. Dr Clarke was also concerned the

[22] T2 p.65, L 44 ;T2, p.67 Ll 4-5
[23] T2 p.73, Ll 22-23; p.74 L25
[24] T2, p.85, Ll 30-40.

proposed night surgery was less than ideal timing for the performance of semi-elective major surgery on a neonate. Also, being the evening, there would be fewer staff available in the event of an emergency.

The differing views of doctors from the respective hospitals about where the surgery should occur also affected arrangements being quickly resolved. The Mater Hospital neonatologists, Drs Tudehope and Liley expressed the view it was preferable for Oliver to remain at the Mater with existing support in place. They considered it best to avoid the need to transfer a baby. Evidence from Dr Clarke and Dr Cartwright indicated there was a well established practice of surgery and post surgical care of more acute and smaller babies than Oliver at the Prince Charles Hospital.

The evidence was clear that despite the parents' anxiety and desire for the surgery to proceed as soon as possible, Oliver was in a stable but unwell condition on room air. He required the ligation procedure, but was not in an emergency situation. He was classified as a category 1 patient (requiring surgery within thirty days), which is what occurred.[25]

Dr Cartwright thought from reviewing the record that Oliver had "*deteriorated over that time, becoming tachypnoeic and ceasing to be able to suck feeds, despite fluid restriction".*[26] Nevertheless Oliver was not intubated or ventilated between the 23 March and the 13 April.

On review, Dr Chard[27] was clear that the delay before surgery had not caused or contributed to Oliver's death. The symptomatic effects of the ductus had been sufficiently managed in the interim period by fluid restriction. Oliver could not feed independently but with the assistance of nasogastric feeds he was putting on weight. At no time prior to surgery did he require ventilation.[28] Dr Cartwright was of a similar view, as was Professor Tudehope.[29]

The surgery

Ligation of the PDA occurred at the Mater Children's Hospital on 13 April 2006. Dr Clarke considered it was a relatively straightforward and uneventful procedure with no surgical complications. His operation notes record:

> *"A left lateral thoracotomy was performed. Entry into the chest was via the fourth intercostal space. The anatomy was well defined before proceeding. The aortic arch, descending aorta, patent ductus arteriosus and recurrent laryngeal nerve were identified. Subsequently a silk tie was passed around the patent ductus. A small breach in the pericardial sac was made with a small leak of pericardial fluid only. Subsequently the duct was tied down with a single silk ligature. The area was inspected and there was no bleeding identified. The ribs were subsequently reapproximated*

[25] Statement of Dr Ward, Exhibit B13 paragraph 18
[26] Exhibit E1, paragraph 2.
[27] Exhibit E2 p.2
[28] T2, p.53, Ll35-45.
[29] T2, p.89 Ll44-55

with Vicryl sutures and superficial tissues closed in layers. No chest drain was placed as the site was haemostatically dry and there was no injury to the left lung made. The patient returned to the intensive care in a satisfactory condition."[30]

Oliver was transferred to the intensive care unit at approximately 3.00pm on 13 April.

The independent review of Oliver's management concurred that the surgery was competently performed.[31] Surgery was uneventful and the subsequent autopsy findings confirmed the ductus arteriosus was correctly and completely ligated. There was no evidence of bleeding in the chest, pleural space collection or any other surgical complication. Professor Tudehope also agreed the surgery had nothing to do with the post surgical ventilation difficulties.[32]

Size of endotracheal tube

On return from surgery, Oliver was managed on endotracheal ventilation.

Dr Shirley Cheung was the anaesthetist during Oliver's surgery. She saw Oliver two days prior to surgery to assess him for anaesthetic purposes. She did not anticipate any problems and it was routine to request an intensive care bed in the immediate post surgery period. Dr Cheung was assisted by her anaesthetic registrar, Dr Amanda Smith. Her recollection was Dr Smith performed the intubation prior to surgery without any difficulty. Had there been difficulties, Dr Cheung would have taken over from the registrar.

Dr Cheung explained there is a balance to be reached in selecting the size of an endotracheal tube (ETT). There must be a degree of leakage around the tube to ensure appropriate ventilation pressure and gas exchange is maintained. There must also be sufficient clearance to avoid tissue damage which could cause stenosis in the trachea over the longer term. She relied on her clinical experience and judgment to select a 3.0mm size tube.

There was some discussion at inquest about this choice of size. Dr Cheung stated there were no national guidelines to determine ETT sizes in neonates.[33] She relied on the Royal Children's Hospital in Victoria which recommended a size 3.0mm ETT for infants weighing 2.5 kg to 3.0 kg. Oliver weighed 2.66 kg at this time. These guidelines also recommended a size 3.5mm ETT for full term babies who weigh more than 3.5kg. Dr Cheung explained these are guidelines to be considered but clinical experience might require adjustment. Had there been a significant leak around the tube after intubation then she would have upgraded the tube to a 3.5mm size. Ventilation observations were good throughout surgery and so there was no indication to consider the tube was of inadequate size.

[30] Exhibit B4, p.8
[31] Exhibit E2, p.2
[32] T2, p.90, Ll10-15
[33] Exhibit B3, paragraph 14

The neonatologist, Dr Helen Liley has a particular interest and experience in intubation of infants. She was responsible for running in-house resuscitation training at the Mater Mothers' Hospital and other hospitals in the southern zone. Dr Liley referred to the Neonatal Resuscitation Program of the American Academy of Paediatrics and the Australian Resuscitation Council guidelines. These recommended the use of a size 3.5mm ETT where a baby was over 2 kilograms.

Subsequent comment by an independent cardiac surgeon, Dr Chard, noted it is the prerogative of the anaesthetist to choose the size of the ETT. He confirmed a correctly sized tube permits a small air leak around it and thus avoids tissue damage. The evidence was the 3.0mm tube selected was satisfactory during surgery and for 24 hours after surgery.

Upon review, Dr Cartwright noted in his experience it was common that anaesthetists used a 3.0mm ETT for a baby of Oliver's size in routine anaesthesia. He did comment it might be expected that a child with a large left to right shunt would have stiffer lungs than normal, given the high blood flow that had been going through them.

Post surgical period and ventilation

At about 1500hrs on 13 April a neonatology registrar (Dr Arreza) accompanied Oliver from theatre back to neonatal intensive care. Oliver was intubated and ventilated at this time. A chest x ray during the day confirmed correct positioning of the ETT and nasogastric tube. There was no sign of a collapsed lung during the day.

Oliver was ventilated satisfactorily in the immediate post surgical period, as evidenced by the first post operative blood gas results at 1600 hrs on 13 April. His pCO2 level of 49.8mmHg was achieved on modest ventilation pressure of 20/5 cmH2O at a rate of 60/minute, inspiratory time 0.3secs. The ventilator used was a Draeger Babylog 8000 in synchronised intermittent positive pressure ventilation (SIPPV) mode. This is where the ventilator senses the baby's attempts to breathe and supports every attempted breath (although there would be few spontaneous breaths by a baby heavily sedated after such an operation), with the ventilator set rate providing 'back-up' breaths at the set number of breaths per minute rate if the baby does not attempt to breathe as quickly as the ventilator set rate.

A senior registrar (Dr Swaminathan) and registrar (Dr McGill) were on duty over the first post surgical night. Both doctors had also been involved earlier in Oliver's care. From their evidence it was apparent that Dr Swaminathan was the more experienced and confident decision maker.

During that first post operative night, Oliver's carbon dioxide (CO2) levels were significantly raised and indicating a problem with ventilation.[34] Pressure and rate of ventilation were increased. At or about 0300hrs a chest x-ray was ordered. The doctors interpreted these results as a collapsed left lung, although subsequent expert review considered it may simply have been oedema fluid due to insufficient ventilation.[35] Carbon dioxide levels remained high. Dr Swaminathan decided to change the mode of ventilation on the Draeger Babylog 8000 ventilator from SIPPV to high frequency oscillation ventilation (HFOV). This mode held the lungs open at a constant pressure with very fast oscillations to promote gas exchange in order to achieve ventilation.

Use of Draeger Babylog 8000 ventilator in HFOV

The Draeger Babylog 8000 is known to be inefficient in HFOV mode with babies of more than 2000 grams in weight because it is inadequately powered for this task.[36] Dr Liley's evidence stated this ventilator has a weight limit of a two kilogram baby when used in HFOV mode, but there was "*nothing wrong with the conventional ventilation on a Draeger ventilator for a baby even quite a bit bigger than Oliver".*[37]

Dr Swaminathan was aware of the limitations of the Draeger Babylog ventilator.[38] Not all registrars appeared to understand the ventilators and their appropriate application. Doctors Arreza and McGill acknowledged the limitations of their understanding of the ventilation machines and appropriate applications.[39]

Dr Swaminathan considered whether Oliver required the more powerful "Stephanie" ventilator but thought Oliver was not expected to require ventilation for more than a couple of days. He gave evidence he handed over the information at the morning ward round to Dr Liley and indicated his view that if there was no improvement then the Draeger Babylog ventilator should be changed to the "Stephanie" ventilator. Dr Swaminathan said Dr Liley considered Oliver should be reviewed after physiotherapy and a chest x ray.

In reviewing Oliver's care Professor Tudehope acknowledged Oliver should have been ventilated by the "Stephanie" ventilator rather than the Draeger Babylog machine. By the time of his operation he was equivalent to 41 weeks

[34] Exhibit C1, p.285 extract from "Ventilation, Blood gas flow sheet".

Time 13 April	**Mode**	**Pressure**	**pCO2**	**Ventilator rate**
2303hrs	SIPPV	18/5	97mmHg	60
0132 hrs **14 April**	SIPPV	20/5	96.2	60
0257	SIPPV	24/5	99.9	70
0412	SIPPV	24/5	71.4	70
0500	HFOV	100	63.0	10

[35] Exhibit B11, para25; T8, p.19, Ll20-30;
[36] Exhibit B7, attachment PL7 "High Frequency Oscillatory Ventillation (HFOV) by the Draeger Babylog 8000"
[37] T3, p.34, Ll30-40 and p.35, Ll40-45
[38] T5, p.31-32
[39] Not all registrars were aware of the capability and appropriate use of different ventilators- see Dr Arreza, T2, p.109, Ll34-38, Dr McGill T4, p.67, Ll21-26, and p.69

gestation and weighed 2.66 kilograms. The "Stephanie" is a more powerful oscillator. Professor Tudehope noted that a baby who has a PDA often has "stiff, wet" lungs requiring quite high pressures. He also considered, in retrospect, it was premature to have decreased inspiratory pressure in the early post operative stage. During the operation a right radial artery line was in situ which gave easy access for blood gas samples, but unfortunately this was "lost" at some undetermined time. A capillary line was inserted to obtain blood gas samples at about 4.00am on 14 April. Professor Tudehope considered on reviewing the information after Oliver's death that there should have been higher pressure and a longer inspiratory rate. Oliver was an unusual baby in that most babies requiring the ligation procedure are ventilated prior to surgery whereas Oliver was managing on room air. The signs were there on the first night post operatively that although oxygenation was satisfactory, ventilation and respiratory acidosis was problematic.[40]

There were signs of a problem with carbon dioxide elimination on the first post operative night but, on review, Professor Tudehope agreed this was a reasonable decision to then consider the results of the change to oscillation mode at the next morning ward round with the consultant.

As Professor Tudehope explained:

> "*I think that was a decision that should have been made at 8 o'clock in the morning. It is quite a big deal to change a baby from a Babylog to a Stephanie. It's not a decision to be made lightly. It takes a lot of nursing staff, you can't provide the oscillation right through the time that you're going to change, so you want to have full staffing if you possibly can, and not do it acutely."*[41]

Although Professor Tudehope confirmed there should have been discussion with the consultant before the registrar, Dr Swaminathan, made the decision to change to high frequency oscillation, he confirmed it was the right decision with positive improvement within the hour. The on call consultant, Dr Gray was contacted within a couple of hours.

The CO_2 levels were rising again by 4.00pm of 14 April. Professor Tudehope considered the oscillation was inadequate:

> *"one had almost pushed the oscillator as far as you could. The baby was on 90% amplitude……and a mean airway pressure of 14, so you didn't have much room to manoeuvre…..It wasn't the oxygen that was causing the problem, so you probably need to go to something that was a more powerful oscillator that would shake the lungs more strongly, if you were going to stay in that mode of ventilation."*[42]

[40] T2, p.81, Ll10-60
[41] T2, p.95, Ll48-60
[42] T2, pp.82- 83

Professor Tudehope considered the elevated carbon dioxide readings at 4.00pm and 11.00pm on 14 April should have been followed up with another blood gas sample within two hours or sooner. Consideration should also have been given to change to the more powerful Stephanie oscillator or to changing the tube to address the significant leak at an earlier time. If the existing oscillator mode of ventilation was to continue then close observation of the child and a follow up gas was necessary to see how well the baby was progressing.[43]

Professor Tudehope also considered the 7 hours between 4.00pm and 11.pm on 14 April was too long a gap between blood gas testing where there was now an arterial line in place. He also agreed that the next blood gas at 3.30am was too long an interval although it was perhaps explicable due to the emergency delivery of premature twins after 2.00am on that morning.

The 14 and 15 April 2006

Oliver continued to be ventilated on the Draeger Babylog ventilator in HFOV mode.

Dr Liley was in the neonatal unit for most of the day of 14 April. She reviewed Oliver on the morning ward round. She recalled the two overnight registrars were present as well as Dr Arreza.

Dr Arreza recorded the notes which indicate Oliver was assessed to be fluid overloaded and his fluids were adjusted accordingly. Ventilator pressure was increased and a blood transfusion was arranged due to mild anaemia. His morphine level was adjusted upward. Physiotherapy to assist in clearing the lungs was provided during the day. Dr Liley could not say at what time she went home on 14 April but thought it was either late afternoon or early evening. Her statement indicated she considered Oliver was stable when she left the ward.[44] The repeat chest x ray at 11.43am showed improvement in the inflation of the left lung.

Dr Arreza finished her shift at 5pm. Blood gas results received at 1604hrs indicated Oliver's carbon dioxide level had risen from 43.8 (at 1054hrs) to 64.3, an unacceptable level. The pressure had been reduced in the interim to avoid any injury to the lungs. Dr Arreza's evidence gave the impression there was little understanding of the significance of higher carbon dioxide. She could not recall to whom she handed over Oliver's care at the end of her shift, but she had indicated he was stable, which did not address his increased carbon dioxide levels. It was another registrar (Dr Deo) who worked until 10.30pm when Dr McGill commenced the overnight shift. I note Dr McGill was solely responsible for the unit until he contacted the on call consultant, Dr Liley.

Dr Cartwright commented some action should have been considered when the carbon dioxide levels commenced to rise again. Consideration of

[43] T2, p.83
[44] Exhibit B8 paragraph 37

swapping to a higher powered ventilator would have provided more scope. Indeed he considered the Draeger Babylog 8000 was simply inappropriate in HFOV mode for a baby of Oliver's size.

Oliver's oxygen requirement had increased to 30-35% and a blood gas test at 2305hrs showed a pH of 7.17 and a PCO2 of 71.1. The mean air way pressure, which had earlier been decreased, was increased again from 13 to 14 and a follow up blood gas test was ordered for 3 - 4 hours time. A review of the evidence by Dr Cartwright indicated that at 11.00pm on 14 April Oliver's condition should have been a matter of concern. He considered the response by Dr McGill to increase the pressure to 14 was "modest".

Dr Liley had been called back to the hospital at about 2.00am on 15 April. This was not due to Oliver's condition but due to the emergency delivery of premature twins, one of whom was in a serious condition. Dr McGill and Dr Liley attended the delivery.

It was at 3.31am on 15 April, when the next blood gas result was received for Oliver, that his critical condition was realised. The pH level of 6.94 and the PCO2 of 145mmHg were seriously abnormal. A PCO2 reading of 145 is extremely high, but at the time the oxygenation was quite good (the PO2 was 113 and the oxygen on the ventilator was 40%) which meant there was a problem with ventilation.

Neither Dr McGill nor Nurse Morris (who was caring for Oliver) realised the seriousness of his condition prior to these readings being available. Nurse Morris was aware of a leak and she had repositioned Oliver to try to remedy this, but without success. Otherwise, Oliver's appearance had been stable and not concerning. The nurse's last recorded observation at 2.30am indicated a triple plus rating for chest wall movement. There was also a notation at 3.30am of "3C" suctioning, which indicated a moderate amount of creamy secretion had been suctioned.[45]

As Dr Cartwright interpreted the record, there was a sudden unexpected deterioration in Oliver's blood gas results, "*something tipped the balance*".[46] In his subsequent review he considered this could have possibly been caused by a pneumothorax (but this would have caused a corresponding dramatic increase in oxygen needs which did not occur) or a partial blockage in the trachea or a kink in the tube. If there was a partial blockage, gas could get into the lungs sufficient to extend them but can not get out well, meaning carbon dioxide was not getting out. If there had been a complete blockage oxygenation would have been terrible as well, but this was not the case.

Although unable to say with any certainty, it is reasonable to suggest that a mucus accumulation could cause a partial obstruction of the airway. Mucus secretions are expected, and the medical record shows regular suctionings to remove them. The partial blockage by mucus could be overcome (unblocked) by more vigorous ventilation.

[45] T8, p.37, Ll38-40
[46] T8, p.49, Ll35-40

Dr Liley and Dr McGill immediately assessed Oliver. Dr McGill's recollection was there was no problem with cardiac output. Oliver's lactate was normal which indicated sufficient oxygen supply to body organs. Dr Liley interpreted the high carbon dioxide level together with low pH and normal lactate with acceptable oxygen levels indicated there was a problem with eliminating carbon dioxide. She thought Oliver's chest wall vibrations were inadequate and she commenced manual ventilation with an anaesthetic bag with the existing ETT in place. Dr Liley achieved good chest wall movement by this mechanism and so she recommenced Oliver on conventional ventilation (tidal breaths in and out) instead of the previously set HFOV mode. On return to the ventilator there again appeared to be adequate chest wall motion and the ventilator recorded expired tidal volumes of about 17mL. A leak from the ETT of 25 - 35 % was noticeable.[47]

Dr Cartwright's evidence was that at this point the ventilation appeared to be going adequately; the tube was no longer blocked as the chest was moving very well.

Decision to re-intubate

The cause of Oliver's deterioration was not clear but Dr Liley was concerned with the substantial leak around the ETT. She considered this was reducing the efficiency of ventilation and decided to replace Oliver's ETT with a larger size (3.50mm) in an attempt to rectify the ventilation crisis. Dr Liley's decision was justifiable on the evidence. Although she had successfully ventilated Oliver by hand ventilation with the anaesthetic bag she remained concerned about his ventilation. The possibility of a pneumothorax had been discounted and there was no evidence to indicate a kinked tube. Her success with hand ventilation ruled out a total blockage of the tube. In her view it was only the persistent leak around the tube that remained the likely explanation for Oliver's difficulty.

The decision to re-intubate Oliver with a larger ETT (3.5mm) was made at around 4.00am.

In his report, Dr Cartwright stated: "*a baby of 2.5 kg weight would be expected to require tidal volumes of 10 - 12.5 mL, so the measured 17mL would be expected to be quite adequate. While a leak of 25 - 35% is of some significance, it is not so severe as to be dramatically inhibitory to ventilation. In the presence of some difficulty in ventilating, particularly with stiff lungs, it would be wise to be rid of it, but it is not at all uncommon to have a leak of 40 - 50 % and still be able to ventilate a baby very adequately*."[48]

[47] Exhibit E1, p.5. "This 'leak' is assessed by the ventilator as the difference between the volume of gas going into the lungs through the ETT and that coming out. It is assumed that some of the gas going in will pass into the airway and then out again between the ETT and the wall of the airway when there is a gap between them, but, because the airways are a little narrower during breathing out, very little of the gas coming out of the lungs will escape outside the ETT, and most will go through it, so the measured expired tidal volume is considered to closely approximate the volume of gas that has truly ventilated the lungs."

[48] Exhibit E1, pp.5 - 6.

His evidence at the inquest was that the 'leak' did not come on suddenly, it had been there, and "*it did not cause the carbon dioxide to go to 145, something else happened. Something and whatever that was in the clinical description seems to me to have been largely overcome".*[49]

Hindsight, without the pressure of another seriously ill baby and with the luxury of the opportunity to review all the material would suggest the crisis may have passed.

As Dr Cartwright said in evidence it was not an unreasonable decision[50], "*only the people there at the time can really say yeah I think changing this tube is going to make a difference. Clearly the tube wasn't blocked. The evidence is that the chest wall was moving very well, more so than we would normally expect to be ventilating - the need to ventilate with tidal volumes to ventilate a baby like this and ah - we have no more evidence than that. If we had another blood gas it might have shown us what the response had been in the baby to the manoeuvres that had already been undertaken. It was assumed that the changing of the tube would be straightforward and simple procedure, as it had been at the initial operation, apparently, described by the anaesthetist. They didn't have any difficulty intubating and then difficulties were encountered."*[51]

Professor Tudehope considered the first response to address the leak from the ETT was to reposition the baby to trial whether this reduced the leak. Nurse Morris had done so during her shift. Alternatively, the ventilator could be changed or consideration given to muscle paralysis. Overall he considered the decision by Dr Liley to change the tube was a reasonable one and a matter of her clinical judgment at the time. [52]

Dr Liley had returned to care for the sicker of the two twins who had been delivered and directed Dr McGill to replace the existing ETT. Dr Liley described Dr McGill as an “experienced advanced training paediatric registrar”.[53] Dr McGill told the court he had significant expertise with intubation. His evidence was he had performed 150-200 previous intubations.

Although the intubation prior to surgery occurred without any difficulty there was always the possibility of difficultly in re-intubation. Twice Nurse Morris suctioned Oliver’s airway before re-intubation was attempted. There was no mucus plug. Dr McGill encountered difficulty when he tried to insert the larger size 3.5mm tube. He did not express any problem with the light source in the laryngoscope. He described difficulty in identifying the usual anatomical landmarks due to what he described as a large amount of swelling. He could not see the larynx. He withdrew the tube and used the bag to ventilate Oliver before trying again. He used suction as the first attempt had caused some

[49] T8, p.34, Ll10-15,.
[50] T8, p.50, L3.
[51] T8, p.35, Ll 1-15.
[52] T2 p.96
[53] Exhibit B8, paragraph 43

trauma but he still could not insert the tube at which time he stopped and asked for assistance. He continued to ventilate Oliver with bag and mask.

The description at autopsy was "the trachea and upper airways showed some ulceration and inflammatory changes in keeping with intubation."[54] The pathologist Dr Williams explained the swelling was consistent with a reaction from the *first* intubation performed for surgery rather than the attempts to re-intubate on the morning Oliver died. It is also noteworthy that at autopsy there were no signs of collapsed lung following surgery.

Dr Liley was called upon to assist in attempting re-intubation. She could not visualise the larynx but she expressed difficulty with the light source. She was not convinced there was any swelling.[55] A replacement laryngoscope was obtained and she made three attempts to re-intubate but without success. The anaesthetic registrar was called but also encountered difficulty with re-intubation. Oliver's ventilation was managed in the interim with hand pumping and initially this maintained good chest wall expansion but this decreased with time. It was Dr Liley who finally achieved re-insertion of the larger tube but by this time Oliver's initial good levels of oxygenation saturation had declined from the 80's to the 40's. His heart rate had slowed and he arrested. Efforts were made to resuscitate Oliver including chest compressions and intravenous adrenalin but he was declared deceased at 5.05am. According to Dr McGill this was 35 minutes from when there was output. Dr Liley considered it was over a fifteen minute period that Oliver did not receive sufficient oxygen.

The possible alternative means of ventilation by a laryngeal mask airway was not available at the hospital. Even at the time of inquest laryngeal masks remained on "back order". This device may have been of assistance because it does not require the identification of anatomical landmarks to utilise the mask. It blocks the oesophagus and pushes the epiglottis out of the way to enable some oxygen to be administered for a temporary period. The device may not have worked in this situation where Oliver had previously required high pressure ventilation.

Dr Liley was acutely aware of the distress Oliver's unexpected death would cause his parents. She met with Oliver's parents and explained to the best of her ability what had occurred although she was uncertain what had caused Oliver's initial decline leading up to the decision to re-intubate him.

Autopsy

The autopsy examination was conducted by a forensic pathologist (Dr Rebecca Williams) on 19 April 2006. On the basis of her examination and chest pathology results reported by Dr Edwina Duhig, Dr Williams concluded that the cause of Oliver's death was:

1(a) Complicated endotracheal tube insertion, due to or as a consequence of
1(b) Recent closure of patent ductus arteriosus

[54] Exhibit A3, p.12
[55] T3, p.60, Ll 5-10

Other significant conditions were premature birth and pulmonary hypertension.

In particular, the autopsy confirmed there were no surgical complications. The surgery was successful in closing the ductus with an appropriately placed suture. There were no signs of either bleeding or infection and there were no signs of a previously collapsed lung, which led the pathologist to infer this was not relevant to Oliver's death.

Ulceration of the vocal cord mucosa and inflammation of the trachea were interpreted as being related to the timing of the initial intubation for surgery. Dr Williams explained it takes some hours for inflammation to develop. Dr Duhig's histological examination noted mild submucosal oedema (swelling) of the epiglottis.

The pathologist could not comment upon the positioning of the ETT as it had been removed prior to examination.

There were signs of heart failure detected at autopsy including small pleural effusion, a pericardial effusion and ascites but the pathologist concluded there were no other signs to support this diagnosis. There was no other observable condition discovered at autopsy to explain Oliver's death. Dr Williams commented in her conclusion (reached from reading the records and what she observed at autopsy) that Oliver was already deteriorating prior to changing the endotracheal tube. She postulated the baby may have died had the tube not been changed.

However, the pathologist concluded it was the extended time of twenty five minutes during which Oliver did not receive sufficient oxygen to maintain his heartbeat that ultimately led to his death. His heart rate slowed to a point where there was no cardiac output and this could not be restored.

I note Dr Liley's evidence clarified it was perhaps over a period of fifteen minutes that Oliver was not receiving sufficient oxygen during the course of the attempted resuscitation.

After consideration of the reviewing experts' opinions, Dr Williams withdrew the condition of pulmonary hypertension as a contributing factor in Oliver's death.

Explanation for final decline prior to re-intubation

There is no clear explanation to account for Oliver's decline prior to the alarming blood gas results of 3.30am on 15 April. Autopsy rules out the most commonly expected conditions of pneumothorax or infection or lung consolidation. The evidence that Dr Liley was able to re-establish chest wall movement with hand bag ventilation is inconsistent with a total blockage of the tube. There was no evidence of kinking of the tube but there was a not unexpected history of mucus secretions requiring suctioning. As Dr Cartwright opined, the suctioning recorded at 3.30am and Dr Liley's

successful hand ventilation may have resolved a partially blocked tube. It remains a matter of some conjecture.

The significance of Pulmonary hypertension stage 1

Dr Liley raised the possibility of pulmonary hypertension as a condition which had adversely impacted on efforts to re-intubate and resuscitate Oliver immediately before his death. She was referring to the physiological condition in life as distinct from the anatomical state that could be observed at autopsy. She explained the phenomenon of smooth muscle reactivity where there is a predilection to pulmonary hypertension. Such muscle can vasoconstrict, making it more difficult to resuscitate. She distinguished this from the anatomical state of pulmonary hypertension which had been observed at autopsy at the lowest grade. Dr Liley was perplexed at the difficulties encountered and conscientiously sought to understand and explain what had occurred.

The autopsy report included a reference in the histology tests to pulmonary hypertension but noted it was evident only at the lowest grade.

Dr Chard, Dr Ward and Dr Cartwright's evidence was helpful on this issue. The PDA caused Oliver's lower pulmonary artery pressure. The excessive pulmonary flow was manifested by tachypnoea (rapid breathing) and subsequent impediment to breast feeding. If Oliver was suffering from pulmonary hypertension he would have been better from a cardiac perspective and there would have been no impediment to feeding.

This situation was to be distinguished from pulmonary hypertension, or as Dr Cartwright expressed it, pulmonary hypertension would have led to reversal of the large left to right shunt and persisting cyanosis and poor saturations in Oliver.[56] Dr Cartwright noted that although there had been deterioration in Oliver's condition after 23 March there were no clinically significant signs of cyanosis or desaturations requiring intervention.

Drs Chard and Ward interpreted Dr Williams' histology findings at autopsy of pulmonary hypertension as resolving neonatal pulmonary muscularisation with no clinical significance.[57] Dr Ward was emphatic Oliver did not have pulmonary hypertension, but he did concede the possibility of secondary pulmonary hypertension as a response to high carbon dioxide, low oxygen, constricting the pulmonary artery.[58]

On reflection Dr Williams withdrew her opinion that the anatomical condition had contributed to the cause of death.

The cardiac surgeon (Dr Clarke) considered the proposition but did not consider the issue of pulmonary hypertension postulated by Dr Liley to be

[56] Exhibit E1 paragraph 1
[57] Exhibit E2
[58] T5, p.58, Ll40-60 and p.59 Ll40-50

relevant to problems in resuscitation. He considered it was a ventilation problem.[59]

Given the state of the evidence I find it remains no more than a "possibility" that pulmonary hypertension may have contributed to Oliver's death.

Additional observations and issues raised by Mr and Mrs McVey:[60]

Although some of this information repeats the previous background summary, it is important to document the parents' perspective in this inquest.

The parents, and in particular Shannon McVey (Shannon), were very involved and informed about the care of their babies while they were in hospital. I accept Shannon's evidence that she attended daily for most of the day while both children were in hospital. After Hamish was discharged home on 18 March 2006 Shannon was not always able to be present during the doctors' ward rounds but still usually attended daily. Otherwise, her husband Steven would attend.

Shannon recalled both children developed "colds". This was just prior to Oliver moving from SCN2 to SCN1. Her recollection was that Hamish had improved over a week and was well when he came home, whereas Oliver remained "chesty" sounding, up to the date of his operation on 13 April 2006.

Shannon had started to introduce breast feeding to Oliver. It was shortly before Oliver was moved to SCN1 that Shannon recalled being aware of episodes of desaturation of oxygen levels after nasogastric tube feeds. On being moved to SCN1 he was placed back into an isolete because his oxygen saturation levels were not as good as they had been.

Oliver did not establish successful breastfeeding; he would detach and appear too tired to continue. His 'snuffy' symptoms interfered with his ability to suck and breathe. He remained reliant on nasogastric tube feeding. His mother also noted a decline in saturation levels when he was held. He was given saline nasal drops and suction was used to assist him. Caffeine was also ordered to stimulate his breathing and reduce the episodes of desaturations and apnoeas.

Shannon recalls Oliver was increasingly desaturating from early March 2006 with high respiratory and heart rates. She observed her child appearing more tired and having less interest in feeding. He was demonstrating "head bobbing" as he detached from the breast to breathe. Shannon expressed her concerns to Dr Tudehope and Dr McGill. She recalled there was consideration of anaemia as the explanation for these symptoms, or perhaps it was simply that Oliver was demonstrating behaviours of a typical premature baby. He and his brother were started on iron supplements but she considered there was no improvement in his condition which was worrying.

[59] T4, p.31, Ll1-10
[60] Exhibits B19 and Exhibit D1

By 11 March Shannon raised her concern about the standard of nursing care in SCN1[61] and her baby's condition with the head nurse. She was worried there was a progression of agency nurses whom she considered demonstrated a lower standard of care from what she had observed in SCN2. A doctor reviewed Oliver due to his mother's concerns about laboured breathing, congestion, retraction from breast feeding, working hard to breathe and desaturations. Shannon was concerned he had a heart problem and wanted an echocardiogram.

During the period 17-19 March Shannon McVey raised her concerns with the senior consultant, Professor Tudehope.[62] Oliver was treated with fluid restrictions. A slight heart murmur had been heard but his pulses were strong. This was explained to Mrs McVey in the context of the previous possibility of coarctation. A strong pulse was not consistent with this condition. Professor Tudehope decided an echocardiogram was not necessary at the time. An unexpected weight gain of 300gm in a week raised concern of fluid overload and diuretics were ordered. His oxygen desaturations improved on 17 March but then reverted.

Shannon recalled a nurse known to her as "Maree" was concerned Oliver remained chesty sounding despite continual suctioning with nothing much extracted. He remained tired and was "head bobbing" indicating more effort required to breathe.

A chest x ray was ordered after Mrs McVey's continued expression of concern about Oliver's continual congestion. The chest x ray was performed on 19 March and Dr McGill rather than Professor Tudehope reviewed it with Mrs McVey.[63] He considered the only variation from the normal was a slightly enlarged heart. He expected an echocardiogram would be arranged before Oliver went home.

A change in the team caring for Oliver brought a fresh perspective. The consultant, Dr Woodgate with the registrar, Dr Zankl took over from 20 March. Mrs McVey remained concerned for Oliver and expressed her concerns to the registrar, Dr Zankl. She was reviewing Oliver for his fitness for surgical repair of inguinal hernia. She considered his lungs to be "crackly" sounding. She expressed the view Oliver was not fit for surgery. On hearing the mother's concerns she arranged for the consultant (Dr Woodgate) to review Oliver. The heart murmur, which had been considered benign, was reconsidered and it was decided a review by a cardiologist was required. A patent ductus arteriosus was suspected. The echocardiogram was performed by Dr Cameron Ward on 23 March 2006 and confirmed a patent ductus arteriosus (PDA).[64] Surgery was required to close the defect and the McVeys were initially informed this would occur on 29 March.

[61] Exhibit B19, pp.4, 5, 6 paragraphs 32-42
[62] T2, p.12
[63] T2, p.14, L55
[64] Patent ductus arteriosus; persistence of the normal foetal patency of the ductus arteriosus beyond the first weeks of life, it is associated with a shunt of blood from the aorta to the pulmonary artery with the production of a characteristic continuous murmur.

On 28 March Oliver had a blood transfusion in preparation for surgery. The family did not become aware that surgery was delayed until the day before or the day of surgery. Another booking was made but again did not eventuate. The parents were told there was another baby who was more seriously ill than Oliver. Oliver's parents were increasingly concerned as their observations indicated to them that Oliver was deteriorating. Shannon observed he was refusing to feed, was tired and unsettled. His oxygen saturations became more labile when the baby was held. He was sleeping more.

5-10 April 2006

The consultants changed again and Oliver came into the care of Dr Helen Liley. Shannon was actively petitioning the scheduling of surgery and her anxiety increased. Dr Liley became involved in trying to facilitate arrangements between Prince Charles Hospital and the Mater Hospital. The preference of the treating team at the Mater was for surgery to occur on site. They considered that optimal care would be provided if transport could be avoided and the baby be cared for post surgery by the existing team. The surgical team from Prince Charles considered they also had the required expertise, equipment and personnel. Dr Liley reassured Shannon that although Oliver was stably unwell he was managing to maintain his oxygen levels on room air.

Shannon recalled 10 April when she became quite concerned for Oliver. His heart rate went as high as 200-220 and his saturation levels dropped to 75-85 over a two hour period. She requested review by a doctor. Dr Swaminathan listened to Oliver's chest and did not consider there was a significant problem.[65]

In retrospect Shannon considered Oliver was exhibiting signs of cardiac failure. Shannon's concerns continued particularly due to the variability in recording of the nurses' observations in the chart and the delay in responding when monitors sounded indicating he was desaturating. Oliver's parents were distressed observing their son during the wait for surgery as he worked hard to breathe and was easily tired. It was little comfort to be told that Oliver was categorised as "stably unwell".[66]

14 April 2006

In the post surgical period Oliver's parents were told he had a collapsed left lung which was shown on x ray. The parents were dismayed they were not told during the night how unwell their son had been. This was explained as a condition to be expected due to the need to partially deflate the lung to access the heart for surgery. He was changed onto high frequency oscillation ventilation mode. It was a nurse who told the parents and they were distressed they were not informed by Dr McGill during the early hours of 14 April of Oliver's deterioration. Shannon observed secretions from Oliver's nose but when she informed Dr "Sally " (as she knew Dr Arreza) she indicated she would get a nurse to suction. Shannon persisted and the suctioning was

[65] T2, p.20, Ll20-50
[66] T2, p.22

performed. Shannon's observation was that the secretions were milky. It was explained that this mucus was normal in the context of being intubated.

The parents were told the x ray on 14 April showed the lung was reinflating. Shannon was aware that adjustment of ventilator settings and adjustment of the child's position could improve his ventilation.

During the day of 14 April Shannon recalled periods when Oliver's saturation levels dropped to as low as 50.

The McVeys left the hospital in the late afternoon believing their son was in a stable condition. Shannon phoned the hospital at 11pm and was told Oliver was on room air and doing well. This must be an error either in Shannon's recollection or in her statement or an error in the information that was given. Oliver was intubated and being ventilated since his operation up until the final decision was made to change his ETT to a larger size.

The parents phoned again at 2am. They were told Oliver's oxygen level had increased to 30% and he was stable and still fully ventilated. This is consistent with other information that he was being ventilated. There was no mention of increasing carbon dioxide levels.

Between 4 and 5am the McVeys received a phone call from the hospital. They were told Oliver's blood gases were not good and they should come to the hospital. Dr Liley and the nurse team leader, Mary, met the parents and Shannon's father at the night entrance to the hospital. Janice, the nurse who had been looking after Oliver, was also present.

Dr Liley told Mr and Mrs McVey they needed to re-intubate Oliver because an air leak had developed. They had been unable to re-intubate in a timely fashion. Oliver suffered a cardiac arrest and died despite efforts to resuscitate him. Dr Liley explained the blood gas result showed 145% carbon dioxide in his blood at 3 am. Dr Liley told them she examined Oliver and noted a significant air leak past the tube. She explained there was difficulty in replacing the tube. Eventually, Dr Liley had achieved re-intubation. By this time Oliver's heart had slowed and he arrested. They could not resuscitate him. Dr Liley was uncertain what had caused this and told the McVeys an autopsy could occur to investigate the cause of death.

Oliver was brought to them and the parents took time with their child and bathed him.

Subsequently a meeting was arranged in July 2006 with the parents, Dr Liley and Dr Lucy Cooke. Dr Liley discussed the size of the endotracheal tube. The McVeys recalled Dr Liley explaining a full term 2.5 kg baby would use a 3.5 mm tube. The anaesthetist had inserted a 3.0mm tube which Dr Liley considered was too small for Oliver's size and weight. They did not change the tube over during the first day after surgery because Oliver was coping once they changed him to high frequency oscillation ventilation. Dr Liley's opinion of best practice indicated a tube of size 3.5mm was appropriate. The

smaller size tube allowed oxygen to escape up the side of the tube as Oliver's lungs began to "stiffen" and greater pressure was used for ventilation.

The parents asked why it had taken so long to re-intubate Oliver. Dr Liley explained that the first attempts caused some bleeding which obscured vision and there was also a problem with the laryngoscope light source. There were 8 attempts made by the three doctors over approximately 25-30 minutes during which time Oliver was not sufficiently oxygenated.

It was explained blood gas testing is usually performed four hourly. This was in response to the family's questions raising concern that Oliver's carbon dioxide levels had gone from about 40% at 7pm to 71% at 11pm on 14 April.

There were discussions about whether the delay in surgery might have contributed to Oliver being less capable of withstanding the surgery. His parents believed Dr Liley agreed this might be so, although Dr Liley did not concede this in her evidence. Dr Liley explained that although Oliver needed the surgery he was not in urgent need and other babies with greater need might be ahead in the waiting period.

There were further discussions about the parents' other concerns about the standard of nursing care, particularly after Oliver was moved to SCN1.

Findings pursuant to section 45

Oliver McVey was born at the Mater Mothers' Hospital on 7 February 2006 and died there on 15 April 2006. He was born prematurely at 31 weeks and 1 day gestation. On 23 March a diagnosis of patent ductus arteriosus was confirmed by echocardiograph. Oliver was 2.66 kilograms at the time of surgery. Surgical ligation occurred successfully on 13 April 2006 and he was returned to the intensive care nursery. He was ventilated with a 3.0mm endotracheal tube connected to a Draeger Babylog 8000 ventilator. The ventilator had sufficient capacity for Oliver's weight when used in the conventional synchronised intermittent positive pressure ventilation (SIPPV) mode. When used in the high frequency oscillation (HFOV) mode the ventilator was not recommended for babies over 2 kilograms.

Problems with retention of carbon dioxide rather than oxygenation developed during the first post surgical night of 13-14 April. A suspected partial collapse of a lung was revealed with a chest x ray. Subsequent expert review considered this may have been an oedematous lung, consistent with under ventilation. The attending doctors increased the pressure and then changed to high frequency oscillation ventilation mode. Oliver's condition improved in the early hours of 14 April. Consideration of a changeover of ventilator to the higher capacity "Stephanie" ventilator was to be undertaken during the following day.

On 14 April the consultant, Dr Liley reviewed Oliver's condition. She directed monitoring of his condition and ordered physiotherapy to assist Oliver's breathing and to assist clearance of any mucus secretions. Ventilation in the high frequency oscillation mode continued as it appeared that Oliver's

condition was stable although carbon dioxide levels were rising by the end of the day.

At about 11.00pm on the second post surgical night Oliver's ventilation required an increase in pressure to address rising carbon dioxide levels. Again, subsequent expert review considered the adjustment to be "modest". Around 2.00am on 15 April Dr Liley was called into the hospital to assist in the delivery of premature twins.

Nursing staff monitored Oliver's condition during this period and although there was a persistent leak from the endotracheal tube there were no apparent signs of a serious decline in his condition. He was suctioned at 3.30am due to moderate levels of creamy secretion and at 3.31am blood gas results were received. These results revealed Oliver's ventilation was critically awry with a carbon dioxide level of 145 as well as increasing respiratory acidosis. Dr Liley reviewed Oliver with Dr McGill. She hand ventilated Oliver through the existing tube with an anaesthetic bag and this achieved an increase in chest wall movement. Oliver was returned to the conventional SIPPV mode of ventilation on the Draeger Babylog ventilator.

It is unknown what had triggered this critical decline in Oliver's ventilation. There was no evidence of complete blockage of the endotracheal tube, or of a kink in the tube. A partial blockage was a possibility or some unknown underlying lung pathology. What was apparent was a significant leak around the endotracheal tube. Dr Liley considered this may be adding to the difficulties in ventilating Oliver and she decided to upgrade the endotracheal tube to a size of 3.5mm. Independent expert review considered this decision was a reasonable clinical judgment call. In retrospect, Oliver could have remained on conventional ventilation under close observation as conventional ventilation had increased chest wall movement.

Dr Liley directed Dr McGill to re-intubate Oliver with the larger size tube. Initial intubation prior to surgery was uneventful but the re-intubation proved problematic to the registrar, the consultant and the anaesthetic registrar who all made attempts to introduce the larger tube. The consultant Dr Liley was finally successful but she acknowledged there was a period estimated at 15 minutes of the overall 25-35 minute period during which Oliver was not sufficiently oxygenated. He arrested and all efforts to resuscitate him were unsuccessful. The treating team were uncertain of Oliver's cause of death.

Autopsy examination excluded any anatomical reason or disease or infective process to account for Oliver's death. The examination of the surgical site confirmed the procedure was effective in closing the patent ductus arteriosus. There was histological evidence of the lowest grade of pulmonary hypertension. Expert comment considered this was merely resolving muscularisation in a premature baby and the pathologist resiled from her previous view that the condition contributed to Oliver's death.

Dr Liley, who also had expert experience in research involving lung development in infants and the physiology of pulmonary vascular smooth

muscle, postulated this reaction as an explanation why the team experienced such unexpected difficulty in resuscitating Oliver. I consider there was insufficient evidence to reach a conclusion on this proposition.

The reason for Oliver's critical levels of carbon dioxide and respiratory acidosis remain unknown. A partial blockage of the endotracheal tube appears the most plausible explanation.

I conclude Oliver died due to lack of sufficient oxygenation in the course of a complicated endotracheal tube re-insertion. The context of the need for re-intubation was to remove an air leak by re-inserting a larger endotracheal tube (3.5mm) to assist in ventilation. The need for ventilation was due to recent successful surgery to close a patent ductus arteriosus. Oliver's prematurity was a contributing factor in his death.

I conclude anatomical pulmonary hypertension did not contribute to Oliver's death, but I am unable to determine whether physiological pulmonary hypertension contributed to his death.

Comments pursuant to Section 46

These comments are directed towards improving public health and safety and with the aim of helping to prevent deaths from occurring in similar circumstances in the future.

Standards of nursing care

Oliver's mother, Shannon McVey, presented as an intelligent and articulate witness with good recall of the management of her baby in hospital. The unexpected death of her son Oliver must have heightened her recollections but I consider her a reliable and honest witness. Her concerns included nursing standards as detailed in her statement in this inquest.[67]

I acknowledge the unavoidable necessity of bringing in "agency" nurses who are less familiar with individual ward practices. I hasten to add I am not suggesting an inferior standard necessarily applies to a nurse sourced from an agency, but rather the importance of continuity of care, good handover procedures and an insistence on essential practices and procedures in each unit.

I consider it would be beneficial for the Director of Nursing to carefully read Mrs McVey's statement as well as the transcript of her oral evidence[68] and consider what issues are fairly raised regarding the standard of care. How can these issues be addressed with the aim of maximising the safety and optimal care of babies at the hospital? I note there was evidence that observations were sometimes only recorded where they fell outside the accepted parameters, but in this case, there was also evidence of instances where monitors were turned off or ignored and entries were not made in the record.[69] Oliver was waiting for surgery and although he did not require

[67] Exhibit B19
[68] T2, pp.3-37
[69] T2, p.8, Ll 10 - 20 (see also nursing note in chart for 14 /3/06)

ventilation his condition curtailed his ability to breastfeed.[70] Frequently recorded observations are the mainstay of information relied upon by the treating doctors to inform their conclusions and decision-making regarding treatment.[71] The importance of sufficient accurate recording of observations cannot be overemphasized.[72] This leads to the next observation.

General record keeping

There were days without any notation in the medical record.[73] I accept the evidence that Oliver was observed, cared for, examined and treated during such periods, but the consultants' and reviewing experts' opinion was clear; registrars are required to adequately and regularly update the medical record with entries detailing examinations and treatment.[74] Hospitals need to consider how they monitor compliance with this requirement.

An example of an oversight in record keeping was revealed in Professor Tudehope's evidence. He indicated he had stated a day or two earlier than 19 March a chest x ray was required. The notation was only entered on 19 March by the registrar. The chest x ray was reviewed by the registrar, Dr McGill. He considered the x ray to be "normal" and an echocardiogram was not ordered at the time.

It remains a moot point whether an earlier diagnosis of patent ductus arteriosus might have been reached with greater record keeping.

Continuity of care and communication

There is always a difficulty in achieving continuity of care with different nursing and medical personnel. Mrs McVey raised a valid observation that it is the parents who provide a continuum of contact with the baby or child through the progression of shifts with changing nursing and medical staff. Professor Tudehope acknowledged the importance of both nursing and parental observations in informing the consultant about the condition of a baby.[75] Mrs McVey made the practical suggestion that hospitals could consider formalising a process for parents to record their concerns or questions. This could facilitate communication with medical personnel. I consider it is worthy of consideration.[76]

Echocardiographs

Professor Tudehope and others noted there are some neonatal units around the world that routinely perform echocardiography in the first days after birth, but they are very few. The echocardiogram is the mechanism by which a definitive diagnosis of patent ductus arteriosus is reached. In the alternative, clinical signs are interpreted to indicate whether a susceptible baby requires this diagnostic examination. Professor Tudehope considered there was a need for research to drive proper consideration of more widespread use of

[70] T2, p.47
[71] T2, p.45
[72] T2, p.46
[73] T2, p.52, Ll25-60, T2, p.56, Ll10-20, T3, p.41
[74] T2, p.56,Ll20-30
[75] T2, p.46, Ll4-8
[76] T2, p.17

echocardiographs of neonates. Consideration should be given to funding randomised controlled clinical trials to assess the benefit of echocardiographs in early diagnosis. The circumstances of Oliver McVey's diagnosis demonstrate the decisive role of this diagnostic tool.

Ventilators

The evidence indicated variable levels of understanding of the workings, limitations and appropriate application of the Draeger Babylog 8000 and Stephanie ventilators amongst the neonatology and paediatric registrars who treated Oliver. This is of concern as some of those registrars were solely responsible for numerous babies when rostered alone at night. The hospital needs to address how it will properly teach its medical staff about the appropriate and safe use of ventilators.

Laryngeal Mask Airways

The evidence indicated a laryngeal mask airway might have been of assistance as a temporary device when medical officers encountered difficulty in re-intubating Oliver. The Mater Mother's Hospital did not have these items available at the time. Consideration of their utility in similar life threatening circumstances should be undertaken.

Coronial Protocols

An unexpected death, particularly of a baby, causes enormous distress to the family but also to the treating team involved in the baby's care. It is important that hospitals maintain accurate information about coronial reporting requirements, which is accessible to staff immediately after the death occurs. There is a difficulty ensuring that all staff members are up to date with information when staff are constantly changing. The hospital must accept responsibility to provide an accurate and accessible information resource known to staff. When unexpected deaths occur in hospitals the on call coroner or on call forensic pathologist must be contacted to discuss whether medical lines and other apparatus are to be removed from the deceased person prior to autopsy.[77]

Concluding remarks

Oliver McVey lived for only a short time. He was the much loved son of Shannon and Steven McVey and twin brother to Hamish. He faced medical difficulties due to his premature birth and the discovery of a patent ductus arteriosus. This condition was successfully dealt with in surgery, but he died due to ventilation problems. His unexpected death was a tragedy and his parents were committed to finding out how their son died and how similar deaths might be prevented. Likewise, medical and nursing personnel demonstrated their conscientious efforts to assist the inquest in reviewing the circumstances of Oliver's treatment.

I acknowledge the assistance of all who have been involved in giving evidence and in the preparation of this inquest, in particular the independent

[77] State Coroner's Guideline re Preserving evidence when a "reportable death" occurs in a health setting

reviewing doctors who have made themselves available to assist the parents and the court to understand and interpret the medical information.

Finally I commend Mr and Mrs McVey for their fortitude, resilience and positive response after their baby son's death, particularly in their continued support of the Mater Children's Hospital Foundation in fund raising efforts for the enhancement of care for all children.

The inquest is closed.

Chris Clements
Deputy State Coroner
28 November 2008