2014FAI 14

SHERIFFDOM OF GLASGOW AND STRATHKELVIN AT GLASGOW

FATAL ACCIDENTS AND SUDDEN DEATHS INQUIRY (SCOTLAND) ACT 1976

DETERMINATION

by

LINDA MARGARET RUXTON
SHERIFF OF GLASGOW AND STRATHKELVIN

IN RESPECT OF THE INQUIRY INTO THE DEATH OF

KATHRYN ROSE BEATTIE

GLASGOW, 4 July 2014

The Sheriff, having resumed consideration of all the evidence adduced and submissions thereon, **FINDS AND DETERMINES** in terms of Section 6(1) of the Fatal Accidents and Sudden Deaths Inquiry (Scotland) Act 1976 that:

(a) time and place of death

Kathryn Rose Beattie, born 23 August 1990, formerly of 80 Hamilton Avenue, Glasgow, G41 4HD, died on 21 June 2004 at 1700 hours at the Southern General Hospital, Glasgow.

(b) the cause or causes of death

The cause of her death was Intra-Cerebral Haemorrhage due to Acute Promyelocytic Leukaemia.

(c) the reasonable precautions whereby the death might have been prevented

There were no reasonable precautions whereby Kathryn's death might have been avoided.

(d) the defects, if any, in the system of working which contributed to the death

There were no defects in any system of working which contributed to Kathryn's death.

(e) other facts which are relevant to the circumstances of the death

(i) There was a lack of a clear system to ensure that urgent blood films were instructed out of hours when the standard operating procedures were not fully applied. There was confusion as to whose responsibility it was to give the necessary instruction to prepare the film and no clear pathway to ensure that contact was made with the on-call haematologist. The absence of clear instructions created ambiguity and uncertainty and was an unsatisfactory feature of the voluntary on-call arrangements which operated in 2004.

It is noted that these arrangements are no longer in place having been replaced by a full shift system in the haematology laboratory incorporating full compliance with the standard operating procedures in respect of the preparation and examination of blood films at all times of day and night.

(ii) It was an unsatisfactory feature of the on-call arrangements for the haematology department on 20-21 June 2004 that it was not clear to medical staff seeking specialist haematology advice that the doctor on call was not a haematologist. This led to others concerned in Kathryn's care proceeding under the misapprehension that appropriate

specialist advice from a qualified haematologist had been obtained in respect of the management of her coagulopathy.

(iii) The failure by the on-call haematologist to contact the on-call haematology consultant was a serious omission. Had the consultant been made aware of Kathryn's circumstances, an urgent blood film would have been prepared and the provisional diagnosis of leukaemia made at an earlier stage. A referral to the on-call consultant would thereafter have ensured a multi-disciplinary approach to the management of Kathryn's condition at a senior level, which would have included urgent investigation into the underlying causes of her coagulopathy and haemorrhage and allowed appropriate communication with the neurosurgeons involved in her care.

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NOTE

Introduction

[1] This is an Inquiry under section 1(1)(b) of the Fatal Accidents and Sudden Deaths Inquiry (Scotland) Act 1976 into the circumstances of a death in respect of which it appeared to the Lord Advocate to be expedient that an Inquiry should be held on the ground that Kathryn's death occurred in circumstances such as to give rise to serious public concern.

[2] The Inquiry heard evidence over 45 days over an extended period between March 2012 and September 2013. Legal submissions were heard on 6 May 2014 and the Inquiry concluded.

Representation

[3] Miss Laura Miller, Procurator Fiscal Depute, appeared for the Crown in the public interest. Half way through the Inquiry, as a result of illness, Miss Miller's role was taken over by Miss Gail Adair, Procurator Fiscal Depute.

[4] During the early stages of the Inquiry Mr Malcolm McGregor, Advocate, represented Ms Jean Crawford (Kathryn's mother). Thereafter, Mr Gordon Lamont, Advocate appeared on her behalf.

[5] Mr James Kelly, Solicitor, initially represented Dr Gerald Beattie (Kathryn's father). However, in the course of the Inquiry, Dr Beattie dispensed with the services of a solicitor and elected to represent himself for the remainder of the Inquiry.

[6] Miss Laura Irvine, Solicitor, represented Dr James Weir.

[7] During the early part of the proceedings, Miss McKerral, Advocate, appeared on behalf of Dr Jan Bunemann. Thereafter, he was represented by Miss Lucy Keane, Advocate.

[7] Mr Vinit Khurana, Advocate, appeared on behalf of Greater Glasgow Health Board.

The witnesses to the Inquiry

[8] The Inquiry heard evidence from 55 witnesses, including 8 experts. A list of these witnesses is attached as an annex to this determination.

Delay

[9] The Inquiry into the circumstances of Kathryn's death commenced some eight years after she died. The delay in holding this inquiry is clearly a matter of public interest, as was demonstrated early on in the inquiry when it was the subject of a letter in the *Herald* newspaper from a concerned member of the public.

[10] At the conclusion of the Inquiry, at the request of the court, the procurator fiscal produced details of the timings involved. In particular, reference was made to the fact that the circumstances of Kathryn's death were not brought to the attention of the procurator fiscal until some three years after it had occurred. The investigations that followed were made more difficult as a result. On several occasions over the months that followed, Dr Beattie produced additional information which raised further matters that the Crown considered merited investigation. Thereafter some considerable time was spent identifying and instructing suitable expert witnesses and assessing the content of

their reports. Nevertheless there was an unacceptable delay in bringing the matter to court.

[11] The death which is the subject of this Inquiry happened over ten years ago. It is never satisfactory to have an inquiry into the circumstances of a death after such a long time. The pernicious effects of such a delay are obvious: all aspects of the proceedings are adversely affected and potentially undermined. It is intolerable that the relatives of the deceased person have to wait for so long for the Inquiry to be held. For those members of the medical profession involved in the case and whose actions might be subject to public scrutiny, such a lengthy delay is likewise unacceptable. All those called to give evidence are placed in the invidious position of being asked to recall matters which occurred so long ago. Some against whom criticism has been directed cannot properly respond to that because they now have no direct recollection.

[12] The passage of such a long period between the death and the associated fatal accident inquiry is likewise a matter of concern where, as here, the inquiry proceeds under section 1(1)(b) where the circumstances appear to give rise to matters of serious public concern. It is axiomatic that these matters should be addressed promptly lest they remain unresolved. Future public safety and future patient safety may be at risk and lengthy delay may exacerbate that risk. Equally, such delay may have allowed matters to resolve, for whatever reason, so that the issues of public concern no longer arise by the time the Inquiry is heard. In the intervening period changes will inevitably have occurred. Systems of working, equipment, personnel, scientific and medical knowledge will all have moved on. Thus some of the concerns which prompted the holding of the

Inquiry may no longer hold relevance or may have resolved by the time the proceedings are commenced. Thus, lengthy delay in holding an FAI has the potential to undermine the fundamental aims of the legislation and dilute the outcome of any particular inquiry.

[13] Delay of this magnitude inevitably affects the quality of the evidence available to the inquiry. Stated simply, memories fade and direct recollection may be lost. However, the position is more complex. The passage of time allows genuine memory to become corrupted and there is the risk that it becomes affected by external influences, thus rendering an otherwise credible witness unreliable. This was a live issue throughout the Inquiry. Despite constant efforts to clarify the position during the course of their evidence, it was difficult to separate what was the direct recall of witnesses, unaffected by extraneous influence, and what was "recall" constructed from what had since been read, discussed or gleaned from elsewhere. Often it was a mixture of both. This was exacerbated by the fact that most, if not all, of the medical and nursing witnesses were unaware that any inquiries were taking place until several years after Kathryn's death. Some only became aware a matter of weeks before they were called to give evidence, some eight years or so later. In these circumstances, when the witnesses had otherwise no reason to have remembered events in the intervening period, it was not surprising that they had no direct recall and had to reconstruct events by reference to external sources, principally the medical records. Accordingly, a great deal of the evidence before the inquiry was had the character of second-hand "reconstructed" evidence. Inevitably, this affected the weight that I could attach to it.

[14] Paradoxically, this potential for the corruption of memory applies with equal force to those witnesses who, in the intervening years, have constantly revisited and rehearsed the events of 20 and 21 June 2004. The quality of their evidence can also be significantly affected. In this case Kathryn's parents clearly blame the hospital for their daughter's death. They harbour great feelings of anger and resentment which have festered over the years and doubtless magnified. They have been unable to come to terms with her death and as Dr Beattie explained, there are issues of unresolved grief. Dr Beattie has devoted the intervening years to meticulous examination of all aspects of his daughter's death and has carried out extensive research into leukaemia in general and acute promyelocytic leukaemia (APL) in particular. He has built up an impressive knowledge. This, in effect, has been a full time occupation for him – he has not felt able to return to his work as a general practitioner since. In such exceptional circumstances, I have to be alert to the potential for aspects of his evidence to have been affected by external influence as a result of his own extensive investigations. Moreover, with reference to the evidence of the close family members, their accounts of events that occurred in situations of great emotional stress and shock require to be considered with care.

[15] Where, as here, the Inquiry takes place a very long time after the death, the medical records take on a central importance as a source of reference. It was, therefore, most unfortunate that at two critical stages in the progression of Kathryn's disease the medical records were either silent or incomplete in respect of these important events. The case notes also contained a number of inaccuracies. While it is recognised that in situations of critical emergency attention will be fully focused on the patient, the importance of including a retrospective note of such pivotal clinical events goes without saying. It was

unfortunate, too, that the anaesthetic notes of Kathryn's transfer to the Southern General were missing. The events relating to those three areas of evidence were fundamentally disputed by the family and the lack or absence of adequate records served to fuel the family's suspicions. Accurate and full notes of these important events would therefore have been of considerable assistance to the Inquiry.

The legislative framework

[16] Fatal accident inquiries are statutory proceedings set up and governed by the Fatal Accidents and Sudden Deaths Inquiry (Scotland) Act 1976. The sheriff's duty is set out under five separate headings in section 6. After hearing evidence and the submissions of the procurator fiscal in the public interest and from represented parties, the sheriff must produce a determination in terms of these headings.

[17] At the conclusion of the evidence and having heard submissions thereon, in terms of subsection 6(1) the sheriff must determine

(a) where and when the death and any accident resulting in the death took place;
(b) the cause or causes of such death or any accident resulting in the death;
(c) the reasonable precautions, if any, whereby the death an any accident resulting in the death might have been avoided;
(d) the defects, if any, in any system of working which contributed to the death or any accident resulting in the death; and
(e) any other facts which are relevant to the circumstances of the death.

[18] In addition to determining the circumstances of the death under inquiry, the focus of the sheriff's determination should also be forward-looking having regard to future public safety. This is achieved by examining whether there are any lessons to be learned

from the circumstances of the death whereby future accidents or deaths may be avoided. That is the principal ethos of a public inquiry of this nature. This forward-looking focus becomes evident when it is appreciated that the purpose of an FAI is not to establish fault – at least not in the legal sense involving such concepts as duty of care and reasonable foreseeability. Foresight is irrelevant in an FAI. However, hindsight is a legitimate and necessary perspective if the inquiry is to fulfill its purpose.

[19] Significantly, section 6(3) sets out that the determination of the sheriff shall not be admissible in evidence or be founded on in any judicial proceedings, of whatever nature, arising out of the death or out of any accident resulting in the death. Such a prohibition is designed, in part, to encourage a full and open exploration of the circumstances of a death where witness should feel free to give frank evidence in the spirit of the Inquiry, untroubled by concerns about it being used in any other proceedings. However, a more fundamental reason for such a prohibition lies in the fact that an FAI is not a forum designed to establish fault. Nor does the sheriff have any power to make such a finding.

[20] The position was authoritatively stated in the opinion of Lord President Hope in *Black* v *Scott Lithgow Limited* 1990 SC 322, at p 327 in the following terms:

> *There is no power in this section to make a finding as to fault or to apportion blame between any persons who might have contributed to the accident. … It is plain that the function of the sheriff at a fatal accident inquiry is different from that which he is required to perform at a proof in a civil action to recover damages. His examination and analysis of the evidence is conducted with a view only to setting out in his determination the circumstances to which the subsection refers, insofar as this can be done to his satisfaction. He has before him no record or other written pleading, there is no claim for damages by anyone and there are no grounds of fault upon which his decision is required. The inquiry is normally held within a relatively short time after the accident …It provides the first opportunity to canvass matters relating to precautions which might have avoided the death or any defects in any system of working which contributed to it, at a stage when*

these issues have not been clearly focused by the parties to any future litigation which may arise.

[21] The position was more recently confirmed by Lord President Hamilton in *Global Santa Fe Drilling* v *Lord Advocate* 2009 SLT 597 where at p 604 paragraph [28] he observed under reference to *Black* that although the sheriff presiding at a fatal accident inquiry has judicial duties …he does not sit to determine the rights or obligations of parties. Thus a fatal accident inquiry is a fact-finding, rather than a fault-finding, procedure.

[22] However, that is not to say that evidence tending to demonstrate fault may not properly be led before the Inquiry. Nor does it mean that the sheriff is precluded from reaching findings which may infer fault where it is proper to do so. This is important when having regard to future safety and the prevention of a recurrence of the accident or the death. In the words of the learned author I H B Carmichael: *[W]here evidence is sufficiently compelling, the responsibility of exposing and finding fault should be accepted. The whole object of a public inquiry is to get at the truth, to expose fault where fault is proven to exist, and in all case to see to it so far as is humanly possible that the same mistake, when it arises through fault or any other reason, is not made in the future. The public interest, in whose name inquiries are held, requires and deserves no less.* (Sudden Deaths and Fatal Accident Inquiries 3rd Ed, paragraphs 5-63 and 5-76.)

[23] Such a position was recognised by Lord Cullen in the course of his review of fatal accident inquiries: *It is true that the investigation into the circumstances of a death in an FAI may disclose grounds of criticism from which a basis for alleging fault may be inferred. That may be unavoidable if the FAI is to fulfill its function of investigating the circumstances of a death.*

(Report of the Review of Fatal Accident Inquiry Legislation (2009) at paragraph 3.23.) In any event, it is legitimate for a sheriff to reach conclusions indicative of fault in the generally accepted meaning of the word as opposed to the notion of fault as a basis of legal liability.

[24] An important purpose of a fatal accident inquiry is to provide a public airing for the circumstances of a death so that those with an interest may be informed. It allows the relatives of the deceased person the opportunity to hear directly from those involved what happened. It also informs those with an interest in and the means of preventing a recurrence of the circumstances of the death with a view to identifying any lessons that may be learned and any reasonable means by which a recurrence of the circumstances may be avoided in future. Thus although the evidence led at the inquiry may be detailed and wide-reaching in an effort fully to inform relevant parties, it does not follow that every matter explored should be included in the sheriff's determination. Like the Inquiry itself, the sheriff's determination must be finite. Only those matters which properly fall under the provisions of section 6(1) of the Act are relevant.

[25] It follows from all that has been said that proceedings under the 1976 Act are inquisitorial in nature. They are not and should not be adversarial. In the proceedings before me, Kathryn's parents and family remained highly critical of the care which Kathryn received and throughout the Inquiry adopted an antagonistic, sometimes hostile, attitude towards many of the medical and nursing staff who gave evidence. There was often an adversarial atmosphere in the courtroom, despite repeated warnings from the bench. The atmosphere was highly charged and tense. At times that tension spilled over into the well

of the court, particularly between Dr Beattie and the procurator fiscal as a result of further inquiries that had been instructed, entirely properly, by the Crown during the Inquiry. This negative atmosphere was counter-productive and at times made the proceedings unnecessarily stressful for all involved.

[26] The Inquiry itself took place over a protracted period – in total the evidence was heard over a period of 19 months. With the best will in the world, timescales were impossible to adhere to. Initial estimates of court time required for such complex inquiries are invariably optimistic. In this case, it was hoped six weeks of court time should have sufficed for the leading of evidence. That proved to be over-optimistic. It soon became clear that the initial dates (three two-week tranches of court time) would not suffice. Once timetables began to slip, the availability of witnesses and counsel became a problem which continued throughout the length of the Inquiry. No amount of management could resolve these difficulties. As it was, there was a significant change in personnel during the proceedings. Furthermore, the Inquiry was held up for several months due to illness of the procurator fiscal depute and a new fiscal had to be brought into the proceedings. Some time was required to allow the new fiscal to bring herself up to speed, not only with a complex investigation, but to familiarise herself with the evidence already led. Additional enquiries took place on at least three occasions as a result of circumstances which could not have been foreseen and as a result the Inquiry took a number of unexpected turns. Additional material came to light and further witnesses required to be led. Half way through the Inquiry, Dr Beattie dispensed with the services of his legal representative and required some time to consider alternative representation. Various motions were made in the course of the proceedings, largely on

the part of Dr Beattie, most of which were opposed and required some debate. These included motions for specification of documents lodged on behalf of Ms Crawford and Dr Beattie which provoked lengthy debate. Illness and injury to those involved also added to the overall timescale. All these combined to create an Inquiry which proceeded over an inordinately lengthy period.

KATHRYN

[27] Kathryn Beattie was a pretty, bright and bubbly girl when she died at the age of thirteen years and ten months. At the age of nine she was taken out of school by her parents and together they went travelling to various places on the family boat. They spent some time in France. They came back to Scotland intermittently but were largely away on their travels until early in 2004 when they returned on a more permanent basis for Kathryn's continued education. Kathryn was educated by her parents during her time abroad but at the age of fourteen she was set to return to school in Glasgow to complete her education. She had just passed the entrance exams for St Aloysius' College and was excited at the prospect of starting school there with her friends. Kathryn was a talented musician – a promising guitarist – and by the time she retuned to Glasgow was described as bi-lingual, being fluent in French as a result of prolonged visits to France. She was described by all who knew her as a lovely girl, gentle and caring and looking forward to a life of great potential and achievement: at nearly fourteen, it was her dual ambition to be a rock guitarist and a translator. It was a poignant moment in court when a recording of Kathryn's last message to her grandfather was played in which she told him that she was feeling much better. No-one could possibly have anticipated that within a day Kathryn would have died from a bleed in her brain as a complication of a very rare form of Leukaemia.

PART 1 PRE-ADMISSION EVENTS

Events in the week preceding Kathryn's admission to hospital (evidence from Kathryn's parents and aunt)

[28] Kathryn's parents gave evidence that for six days prior to her admission to hospital on Sunday, 20 June 2004 she had been unwell. She had been suffering from an apparently minor flu-type virus which had confined her to the house although not to bed. Her mother said that she was a bit unwell as if she had caught a chill: she had a bit of a cough and had vomited once. On the previous Wednesday, Ms Crawford had commenced her on antibiotics (amoxicillin) which she had brought back from Spain where they are available over the counter. Dr Beattie described her illness as an unspecific, intermittent, mild flu-type viral illness. Dr Beattie did not know that his wife had started Kathryn on a course of antibiotics but, while he would not have done so himself, was satisfied that they would not have done her any harm. However, by the weekend of her death, Dr Beattie had become concerned that Kathryn might be anaemic. Although she had a naturally pale complexion, her father considered that she was *un*naturally pale and was showing clinical signs of anaemia. It had been his intention to have this investigated on the Monday.

Events on the evening of Sunday 20th June

[29] Both parents described how Kathryn had been feeling better on Sunday, 20th. Kathryn had asked if she could go to visit her cousin who lived nearby so that they might play a new computer game. Despite the apparent improvement in her condition, Kathryn's parents thought it prudent to keep her inside for one more day, just to be on the safe side. Accordingly, Kathryn did not go out but stayed in the house. At about

5:30pm Kathryn phoned her grandfather to tell him that she was feeling much better but just had a bad cough.

[30] Dr Beattie was so concerned about Kathryn's apparent anaemia that he contacted his sister, Dr Rosaleen Beattie, a consultant physician in palliative care, and asked her to come round to the house "to give Kathryn the once over". He explained that he had also wanted some advice as to how best to proceed to have Kathryn checked out.

[31] Dr Rosaleen Beattie lived a few minutes away. She had been in Orkney on business and had just arrived back from the airport. Her brother collected her from her house and drove her to his house between 8.30 pm and 9.00 pm on Sunday, 20th. She understood that Kathryn had been off- colour and that her father thought that she was anaemic. Dr Rosaleen Beattie examined Kathryn – what she described as a brief but thorough examination. She noted that Kathryn had a bit of a cough and agreed that she was a bit off-colour. She confirmed that it would be sensible to take her to her GP on the following morning to have her bloods checked. (In the course of her evidence, she made no specific mention as to whether she, too, thought that Kathryn was anaemic.)

[32] Dr Beattie drove his sister home. They had been gone from the house a matter of minutes when, according to Ms Crawford, Kathryn came into the kitchen and told her that she had had difficulty formulating her words. She said something like "Mum, something really strange happened. I wanted to say something to you but I couldn't think of the words". She had wanted to say something "but it just didn't come out right". There was no further specification as to what exactly had happened.

[33] Ms Crawford was immediately alarmed although by then Kathryn was speaking clearly and making sense. Ms Crawford's evidence was that she herself had not witnessed this episode but became very alarmed by what Kathryn had said. She had a terrible feeling inside and was convinced that something was seriously wrong. She phoned her husband at once. He, too, was seriously alarmed, as was his sister. Although the description of the event was somewhat vague, they were alarmed that it might represent some sort of neurological event which should be investigated without delay. Dr Rosaleen Beattie returned to the house and examined Kathryn again. At that time she noted nothing different and the episode, whatever it had been, seemed to have resolved. Neither she nor Dr Beattie gave any further detail of anything Kathryn might have said to them in describing what had happened shortly before.

[34] Dr Beattie then contacted a local GP who was also his (now former) brother-in-law, Dr Douglas Bruce. Dr Bruce was Kathryn's doctor although he had rarely had occasion to see her as a patient. He explained the position to Dr Bruce. According to Dr Beattie, Dr Bruce told him to look up the phone book, find the surgery number, phone it and listen to the recorded message. Dr Bruce's recollection of the conversation was somewhat different. He recalled that Dr Beattie had phoned him at home sometime in the evening. His recollection was that Dr Beattie had explained that Kathryn had had cold symptoms but that over the last couple of hours she had slurring of speech and confusion. He said to Dr Beattie that he (Dr Beattie) would be in a better position to judge whether she needed to go to casualty and if he was so concerned, that was where she should go. Dr Bruce explained that the big worry in such circumstances would be meningitis, although

he did not recall that actually being discussed. He did not mention NHS 24 as with Dr Beattie's experience, if Kathryn needed something it would be hospital. His recollection was that it had been a fairly sudden event. Although he did not recall it, he conceded that Dr Beattie may have described dysphasia.

[35] In the event, Kathryn's parents and aunt decided to call an ambulance via the 999 emergency network.

Paramedic assessment

[36] The 999 call was received at 21:31 hours. An ambulance was immediately dispatched arriving at the house at 2140 hours. It was noted that the nature of the call was recorded as a "collapse".

[37] Two paramedics arrived and examined Kathryn. They found her to be conscious and talking but noted that she was a bit pale. After examination, with the exception of her pulse being somewhat fast at 120 beats per minute, all other tests were normal. Kathryn appeared well when the paramedics were with her in the house.

[38] At that time, Kathryn's mother did most of the talking to the paramedics while her father and aunt, although present, did not immediately take part in the conversation. Evidence was heard from Mr James Fleming who was one of the paramedics who attended. He described Kathryn's condition and their examination. From the history that was given, his colleague completed the patient referral form. Under the section dealing with "History and Additional Information" the following was noted:-

Unwell since last Tues. vomiting, headaches, high temperature. Epistaxis, sores in mouth. Not ate anything earlier. Has been drinking fluids. Commenced on Antibiotics yesterday. To-nite (sic) speech slurred, confused. Pyrexial. Speaking o/e Speech and confusion improved. Complaint of Muscular pain in neck.

[39] Initially, the advice given to Kathryn's mother was to refer Kathryn back to NHS 24 and to take her to see her GP if they were still concerned. It was clear that the paramedics did not consider that Kathryn's condition required that she be conveyed to hospital. At this stage, as it was evident that the paramedics did not intend to take Kathryn to hospital, Dr Beattie and Dr Rosaleen Beattie intervened and explained that they were medically qualified and that they had serious concerns about a specific event. They were strongly of the opinion that Kathryn should at least be taken to hospital. In these circumstances, the paramedics were readily persuaded that Kathryn should be taken in. Because of the geographical location of Kathryn's address, she was taken to the Victoria Infirmary rather than to the Royal Hospital for Sick Children at Yorkhill, Glasgow.

[40] Throughout all this, Kathryn was embarrassed by all the fuss and attention. She appeared well and, indeed, followed the paramedic out to the ambulance when he went to get a chair for her and, according to her parents, "jumped in". On arrival at the Victoria Infirmary, she was apparently laughing and had to be persuaded to allow the paramedics to put her in a chair to take her from the ambulance inside. The atmosphere was described as "light". The ambulance left the house at 21:54 arriving at Victoria Infirmary at 22:01.

PART 2 EVENTS AT THE VICTORIA INFIRMARY, GLASGOW

Accident and Emergency (evidence of Dr Lucy Thomas)

[41] The atmosphere of levity did not continue when Kathryn arrived in Accident and Emergency. She was seen immediately and was classed as "P1" which meant that she was stable but unwell with an underlying condition that required her to be treated as a priority.

[42] Within six minutes, she was seen by Dr Lucy Thomas, who was on duty in the department. Dr Thomas was a Senior House Officer (SHO3) acting as a Registrar. She was, therefore, a middle grade clinician.

[43] Dr Thomas introduced herself to Kathryn and took a history from her parents and from Kathryn. According to Ms Crawford, again she did most of the talking and told Dr Thomas that Kathryn had been suffering from a mild flu-like illness since the Tuesday before. On the Wednesday she had vomited once. Otherwise, she was not eating as much as usual but had been drinking water. She had had "a wee bit of a temperature and a bit of a cough". However, the history, as noted by Dr Thomas on A & E card, was in the following terms:-

> *PC* (presenting complaint) *13 year old female with general malaise*
> *HPC* (history presenting complaint) *unwell for six days with vomiting, flu like symptoms*
> *Spontaneous bruising on limbs*
> *Epistaxis* (nose bleed) *bleeding from gums*
> *This evening mum describes eyes staring, left sided facial droop and dysarthria (difficulty forming words) with confusion. Episode lasted – half hour*
> *c/o neck pain this evening.*
> *PMH* (past medical history) *nil. DH* (drug history) *nil.*

[44] Having noted the history, Dr Thomas proceeded to examine Kathryn. From the outset, she had been concerned about this girl's condition. On examination, she noted Kathryn to be pale and clammy. She was tachycardic with a fast heart rate of 133. Her blood pressure was a little high at 130/83. Her GCS ("Glasgow Coma Scale") was normal at 15.

[45] In the course of their evidence, Kathryn's parents described how, as their daughter was being examined, they noticed bruising appear on her arm and leg. They described this as new bruising which had not been present before and Kathryn's mother described it as "appearing before our eyes". Dr Beattie pointed these out to Dr Thomas and said "these are new". Likewise, while Kathryn was being examined, her parents noted that there was something wrong with the left side of her mouth. Kathryn's mother noticed something different about Kathryn's mouth that she had "a wee different look" and that it was not right. Dr Beattie noticed a slight change in Kathryn's appearance at this time also which he described as a slight asymmetry at the side of her mouth, at the corner. He described it as almost imperceptible.

[46] Dr Thomas noted bruising on Kathryn's left arm and right leg and also noted that she had a slight facial droop. Although slight, it was noticeable. She tested Kathryn by asking her to whistle and to force a smile, which she did. This was a symptom suggesting an abnormality in the VII cranial nerve – the facial nerve – and was indicative of something going on in the brain. Dr Thomas noted no neck stiffness and there was nothing further of significance in her examination.

[47] From the outset, in terms of the history given, the presenting complaints and the results of her examination, Dr Thomas was immediately very suspicious of a possible underlying leukemia and the episode of speech difficulty together with the facial droop made her suspicious of something going on in the brain. Accordingly, she made a differential diagnosis of ?sepsis / ?CNS source (Central Nervous System) or ?underlying haematalogical abnormality/malignancy. It was clear that Kathryn required further investigation. Her plan was to set in train the initial investigation, tests and treatment. She planned to obtain intravenous access, give fluid resuscitation, take routine bloods including cultures, arrange for a chest x-ray, give intravenous antibiotics (ceftriaxone) and refer Kathryn to the medical team. She did all this. She also required a urine specimen and instructed that Kathryn be catheterized. Dr Thomas noted on the A&E record card that Kathryn needed a CT scan of her brain. Unless associated with trauma, emergency doctors did not instruct CT scans. That was a matter for general medicine. Accordingly, Dr Thomas referred Kathryn to the on-call physician for admission.

[48] Kathryn's parents challenged Dr Thomas' evidence on a number of matters which are considered later at paragraphs [128]-[138]. As with the evidence of the paramedic, they disputed that what was recorded about Kathryn's history and presenting symptoms was not an accurate reflection of what they had reported.

First blood results

[49] The first blood results became available at 23:12 hours and were brought to Dr Thomas' attention. These results were alarming as they indicated a grossly abnormal haematology. In accordance with standard practice, the abnormal results were

telephoned directly to A & E from the laboratory. Significantly, the results showed a raised white cell count (WCC) of 31 where the normal reference range would be between 4 and 11. (A raised white cell count is commonly a sign of acute infection.) Secondly, the haemoglobin (Hg), normally expected to between 115 to 165, was low at 8.2. That meant that Kathryn was, indeed, anaemic. Thirdly, the platelet count was 13 ($13x10^9$/L or 13,000). Platelets are sticky substances that form an essential part of the body's clotting mechanism. Again, this was abnormally low and well outwith the normal reference range of 115 to 400. This represented a severe thrombocytopaenia (low platelet count).

[50] Dr Thomas had suspected that this would be the case. As soon as she had seen Kathryn she knew that there was a significant underlying diagnosis and the blood results fitted with both her differential diagnoses. Dr Thomas noted the first blood results on the A&E card and discussed them with Kathryn's parents. Dr Beattie was shocked at the low platelet count. He was anxious that Kathryn received a transfusion of platelets.

[51] It was at this point that, according to the family, they were advised by Dr Thomas that Kathryn had been diagnosed as suffering from leukaemia. Dr Thomas emphatically denied this. It remained an issue of fundamental dispute which is discussed below at paragraphs [139] – [146].

Referral to general medicine (evidence of Dr Weir)

[52] Dr Weir was the senior house officer on call for general medicine that night. He responded immediately to a paged message, arriving in Accident & Emergency at approximately 23:00 hours. Neither Dr Thomas nor Dr Weir specifically recalled where

and how the handover took place – whether in person or by phone – although Kathryn's parents recalled that Dr Weir arrived in the Department and immediately spoke to Dr Thomas. However the handover occurred, I was satisfied that Dr Thomas would undoubtedly have explained the reason for the referral and would have given Dr Weir a full account of the presenting complaint, the history, the principal findings on examination and would have discussed her plan with Dr Weir. Dr Weir, in turn, accepted the referral and from then on Kathryn was under his care, responsibility having passed from Accident and Emergency to General Medicine.

[53] Dr Weir spoke to Kathryn and to her parents and proceeded to take his own history and perform his own examination of Kathryn. It was his recollection that he did not have access to the A&E notes on his arrival. This was not particularly unusual and, in any event, did not alter his normal practice which was to take a history himself and carry out his own examination of the patient. It is worth noting that this practice was universally approved by the various consultants and experts who gave evidence. Dr Weir did not make any contemporaneous notes in the medical records but wrote these up later at around midnight. Dr Weir estimated that he would have spent 35 or 40 minutes taking a history and performing an examination and then about 20 to 25 minutes or so later he would have completed his notes.

[54] He noted the following :-

Presenting complaint A & E Referral with pyrexia, raised white cell count, low haemoglobin, low platelets and left facial weakness. He recorded the following history from Kathryn and her parents: *Background normally well. Seven days ago noticed some bruising on limbs. Five days ago history of flu like illness, with vomiting. Unable to keep foods down. Nose bleed two days ago.* On examination, Dr Weir noted that Kathryn was *pale* +++ and recorded *acute deterioration over the last few hours. Increased temperature. Developed left facial weakness.*

Drooping of left side of the mouth. No neck stiffness. No rash. No confusion. Parents phoned 999. A & E – raised temperature, tachycardia and bloods as on A & E card.

[55] Again, the accuracy of Dr Weir's record of the nature of Kathryn's presenting symptoms was challenged by her parents and is considered later at paragraphs [128]-[138].

[56] His findings on examination were recorded as follows:-

> *Very pale and unwell. Temperature 37.8. Heart rate 130 per minute, regular. BP 130 over 83. Sats (oxygen saturation) 99% on air. Chest is clear. Cardiovascular system, JVP (jugular venous pressure) normal."* That pressure can reflect a raised or depleted intravascular volume. *The CNS examination revealed a normal reading – a GCS of 15 out 15. Pupils were equal and reactive to light and consensual reflects. Fundoscopy was normal. Cranial nerves – there was a right upper motor neuron lesion of the seventh cranial nerve and there was some drooping of the left side of the mouth.*

[57] From memory, he did not think that the drooping was particularly marked. He documented left-sided facial weakness and drooping of the left side of the mouth under history of presenting complaints. He had received information during his history-taking that perhaps there had been a sort of more than marked or severe episode at home that had prompted the call for the ambulance. All other CNS examinations were normal.

[58] After completing his examination Dr Weir started to form his own differential diagnosis at that stage. He was keen to see the blood results. He retired to the doctors' room to write up his notes. He recalled at some time in the course of his examination of Kathryn that Dr Thomas had passed through and informed him of some of the results – he recalled the haemoglobin result which was low and the white cell count which was very high. He was sure he had never been aware of the platelet count at that time because he was struck by it when he saw the results on the A & E card which was in the

pigeon hole in the doctors' room. According to Dr Weir, that was the first time he had access to the results. He certainly had the full, confirmed blood results by midnight.

[59] At 23:35 he prescribed potassium chloride, probably, he said, because the biochemistry lab had phoned the abnormal result and that information was passed on to him while he was still completing his examination. The fluid that had been prescribed since Kathryn's presentation to A & E did not contain any potassium whatsoever. It was logical that having had over a litre of fluid without potassium her potassium would now have been even lower so he replaced it.

[60] While writing up his notes, Dr Weir had access to the results on the computer system and he documented the urinalysis. Those were normal. He also noticed that there was a LDH (lactose dehydrogenase) result on the system that had not yet been included in the biochemistry results already noted and for completion he recorded it. He could see that all the results had been confirmed as true results and he likewise noted that.

[61] Dr Weir was aware that the platelet count was very abnormal and very concerning. It stuck in his mind which is why he remembers that it was only later that he became aware of it. However, had he appreciated that the platelets were abnormally low at an earlier point, he did not think he would have done anything differently.

[62] By this time, Dr Weir was very concerned indeed about Kathryn's condition. He remembered discussing Kathryn with Dr Thomas. He, too, had reached a similar differential diagnosis of ?sepsis / ?underlying haematological malignancy. His main

suspicion was of sepsis but he recalled Dr Thomas suggesting that it might be leukaemia. He was aware that an urgent CT scan was needed to see what was going on inside Kathryn's head and it was the practice to obtain ratification of any out of hours scan by the on-call consultant. In any event, he wished to discuss Kathryn's case with his consultant. That was one of the reasons that he had carefully complied his notes – so that he could provide a full and accurate summary to the consultant without missing out anything important.

Contact with consultant (Dr McAlpine)

[63] Dr Weir telephoned Dr Howard McAlpine, one of the consultant physicians. He thought that Dr McAlpine was on call. As it happened, it was Dr David Vernon who was the on-call consultant that night. It was not clear what led to Dr McAlpine's involvement: whether the hospital switchboard erroneously put Dr Weir through to the wrong consultant or whether, more likely, Dr Weir mistakenly believed Dr McAlpine was on call and asked switchboard to contact him.

[64] Dr Weir's recollection as to the content of the conversation which took place differed from that of Dr McAlpine. According to Dr Weir, although he could not recall the exact details of the conversation, he was certain that he gave a full account of Kathryn's condition to the consultant, including history, presentation, examination, blood results and differential diagnosis. He recalled that Dr McAlpine agreed with conclusions and with his plan to carry out an urgent CT scan. Indeed, it was in discussing Kathryn with Dr McAlpine that it became clear that an urgent CT scan would be required to determine whether Kathryn was bleeding intracranially. Dr Weir noted that part of the

conversation. He recalled that it was only at the end of the conversation that Dr McAlpine told him that he was not actually on call.

[65] Dr McAlpine recalled the conversation differently. Some eight years on, he remembered a call which, according to him, lasted just about a minute. Although a cardiologist, Dr McAlpine participated in the on-call rota for acute general medicine. It was his recollection that he had immediately explained to Dr Weir that he was not the on-call consultant but he asked what the problem was in case there was a cardiac issue with which he could help. He realised as soon as he had the history, examination findings and the blood results that this was a complex clinical picture which required Dr Vernon, the consultant who was on call, to be contacted.

[66] His recollection was that there was no discussion of any differential diagnosis or of haematology involvement although he recalled being told that a blood film would be available in the morning. Nor did he recall specifically discussing the management plan. He believed the primary reason for the call was to obtain his agreement for an urgent CT scan and he agreed with Dr Weir that such a scan was necessary, urgently. Dr McAlpine deliberately kept the conversation brief as his advice was that Dr Weir should contact Dr Vernon. His recollection was that the case was presented to him as a septicemia and at that time did not consider underlying haematological malignancy. It sounded to Dr McAlpine that there might have been some sort of thrombotic event (rather than a haemorrhage).

[67] Dr McAlpine was certain that he did not discuss a management plan for Kathryn nor had he discussed any differential diagnosis. It was his recollection that Dr Weir did not ask for his advice but was simply seeking permission to carry out CT scan. He anticipated that Dr Weir would have contacted Dr Vernon immediately afterwards.

[68] The exact details of this conversation are not particularly important. However, I was surprised that Dr McAlpine, some eight years on, was able to recall the terms of a telephone conversation which, according to him, lasted only a minute. He told the court that he was first asked about the phone call several years after the event which made it all the more surprising. He had apparently no reason to have remembered it except for the fact that it was unusual to have been contacted when he was not actually on call. In these circumstances, I preferred Dr Weir's recollection as the more accurate. He had carefully collated his notes prior to calling the consultant and was therefore primed to provide detailed information. Had the call lasted for only a minute as a result of which no advice was given, I consider that Dr Weir would almost certainly have telephoned Dr Vernon immediately thereafter. However, Dr Weir seemed satisfied with the response from Dr McAlpine and acted as if he had received endorsement of his plan. He clearly did not consider that there was a need to phone Dr Vernon at that stage, having spoken to Dr McAlpine. Therefore I accepted Dr Weir's account as the more reliable.

Contact with on-call haematologist

[69] Rather than telephone Dr Vernon, Dr Weir decided to expedite the further investigations that were required and immediately contacted the on-call haematologist,

Dr Jan Bunemann. Dr Weir did not know Dr Bunemann nor was he aware of his seniority or grade. The call was made at around 00:15 hours.

[70] Dr Bunemann was off-site at home when he received Dr Weir's call. As a result of their conversation, Dr Bunemann authorised the release of one pool of platelets in order to bring up Kathryn's platelet count. He also advised that Kathryn should be give two units of packed red cells to address her anaemia. Dr Bunemann contacted the haematology laboratory to authorise the release of the products. Platelets were not immediately available on site at the Victoria Infirmary but had to be ordered and obtained from the regional blood bank at Gartnavel Hospital, a short distance away. A request was made with them at 00:26. The two units of packed red cells were immediately available from the laboratory.

[71] Dr Weir recalled certain aspects of the conversation with Dr Bunemann and was assisted by rough notes that he had jotted down at the time. Dr Bunemann had no recollection of the phone call at all. Accordingly, he relied solely on the records of the products that were released. He could only surmise what information he had been given by reference to what he did or did not do according to the notes. This was unfortunate given that the content of this conversation was a significant part of the evidence concerning Kathryn's care.

Dr Weir's account of the conversation

[72] According to Dr Weir, he telephoned Dr Bunemann at about quarter past midnight. Not only did he require to contact haematolgy for the release of blood products, he wanted haematology advice because of the grossly abnormal blood results and because

there was a degree of coagulation defect. He was also worried that there might be a bleed and he wanted advice in relation to the possible differential diagnosis of sepsis and underlying haematological malignancy. He wished to make the haematologists aware of the situation and obtain their input into Kathryn's care. According to Dr Weir, he gave Dr Bunemann all relevant information he had and explained his concerns about the patient. Although he had no clear memory of the conversation he was sure that he discussed the differential diagnosis with Dr Bunemann. He believed that he would have used the term underlying haematological malignancy as he did not feel that he had the haematology expertise to suggest to a haematologist what the underlying diagnosis might be – the term leukaemia would have narrowed it down too much. According to Dr Weir, he would have supplied Dr Bunemann with any information he had requested. He was certain that he made Dr Bunemann aware of the urgent CT scan in case of an intracranial bleed. He was sure that he would also have told Dr Bunemann that he had discussed the case with Dr McAlpine.

[73] He thought that Dr Bunemann had understood his concerns and said that he would make one pool of platelets available and recommended two units of packed red cells. He suggested that Dr Weir re-contact haematology in the morning. Dr Weir took that to mean the following morning when the day staff had arrived for duty. It was Dr Weir's impression that Dr Bunemann did not wish to be contacted again. Dr Weir found this slightly surprising as he would have expected an invitation to re-contact the haematologist had he felt the need. Under the heading of his discussion with haematology, Dr Weir noted four lines in the medical case notes in relation to his contact

with Dr Bunemann. Those four lines reflected the full advice that had been imparted to him by Dr Bunemann by the end of the conversation. The entry in the records reads:-

D/W Haematology
→ Platelets
→ 2 u PRC
→ Recontact in a.m.

[74] That reflected the authorisation to release one pool of platelets and two units of packed red cells ("PRC").

[75] Apart from that entry in the medical records, Dr Weir did not make any formal note of the conversation but he did make jottings in the course of the telephone call. These were not a reflection of the whole conversation but just some rough notes he made while he was listening to Dr Bunemann. These notes were jottings that Dr Weir had made on a separate sheet. These read:

Gentamicin
CT
Haematological

- *Platelets >10*

Ok
unless bleeding

- *PRC*
- *2u*

- *ESR* *BM*
- *LDH*
- *LFTs*

[76] These jottings reflected part of the conversation when Dr Bunemann said that a platelet count of above ten was okay unless there was bleeding. Dr Weir could not say at which point in the conversation that had been said. The series of letters noted related to certain tests: ESR (erythrocite sedimentation rate, which is a general indicator of infection); LDH and LFTs (liver function tests). Dr Weir could not now recall why he had

written these results down but suggested that they may have been results that Dr Bunemann was interested in hearing and in respect of which he had specifically asked for results. He believed that he had the liver function tests available and the LDH results. He did not have the ESR results but had CRP results which were for the C-reactive protein, a non-specific marker of inflammation. Further across the page the initials "BM" were noted which refers to a particular test for blood sugar. There was a formal blood glucose result which was available. That these were results asked for by Dr Bunemann was what Dr Weir thought was the most likely reason for having written them down specifically but he had no direct memory of it.

[77] Asked if he was reassured by Dr Bunemann's advice, Dr Weir replied that as he was speaking to the haematology specialist, he had to go by what he said but he was not particularly reassured by it. However, he refused to elaborate on that comment.

[78] Dr Weir did not think there was any discussion of cryoprecipitate but believed that he did ask about the appropriateness of fresh frozen plasma (FFP) which is plasma which contains clotting factors. He thought that Dr Bunemann did not think that FFP was appropriate. No advice was given to continue to monitor the coagulopathy. Packed red cells would be given to address the anaemia. At no time during the conversation was the question of examining a blood film raised. It was Dr Weir's position that blood films were not routinely requested by general medicine and he himself had never before requested one. Indeed, at that stage in his career Dr Weir did not believe that he would have known when a blood film would have been appropriate.

Dr Bunemann's evidence

[79] Dr Bunemann had no recollection of the telephone call at all. He explained that he had not known anything about what had happened to Kathryn and had not otherwise been involved in her care. He had not learned that she had died until years later in 2009. He could only comment on the case by referring to the notes and the very limited entries therein that could be related to actions that he had taken that night. Otherwise, having been taken through the information contained in Dr Weir's notes, he could only surmise that because he did not take certain steps which would have been required in light of that information he could not have been provided with that information. Otherwise he would have undoubtedly acted differently. For example, he said that the LDH results would have been "a red flag", as would any mention of leukaemia or underlying haematological malignancy. In these circumstances, his next phone call would have been to his consultant. He would have realised that he was dealing with something serious that was beyond his ability. In the event, Dr Bunemann did not order further investigation. Nor did he contact his consultant, Dr Anne Morrison.

[80] Dr Bunemann immediately contacted Gartnavel Hospital and ordered one pool of platelets. The platelets were dispensed without delay and sent to the Victoria Infirmary by taxi arriving at the haematology laboratory at 00:54.

[81] At around this time, Dr Weir had contacted the on-call radiologist to make the necessary arrangements for the scan. After his telephone conversation with Dr Bunemann, Dr Weir then telephoned the bacteriology laboratory in the belief that they might look at a blood film for bacteria in the case of severe sepsis. While a culture could

be grown to see if there were bacteria present, Dr Weir understood that results from examination of a blood film would be more readily available. This is an entirely separate issue from the preparation and examination of a blood film by the haematology laboratory. It clearly related to the reference by Dr McAlpine to the discussion concerning the blood film which would be ready in the morning.

[82] Kathryn's condition had remained stable in the meantime. Whilst in A&E she appeared bright and talkative and was conversing with the nurses. Significantly, she was fully alert with a GCS of 15 throughout.

CT Scan

[83] Dr Lorraine Duffy, the on-call radiologist for the Victoria Infirmary, received Dr Weir's telephone call at home. Although she could not recall the exact time of the call, it must have been shortly after midnight. She readily agreed that the scan was necessary and made her way in to the hospital, arriving there some twenty minutes later. She had also called in the duty CT radiographer. The scanner had to be switched on and warmed up ready for use. Once ready, the patient would be sent for. The scanning room is on a different floor from A&E. Kathryn was called to the scanning room at about 00:50 hours according to Dr Weir's recollection. At this point, Dr Weir checked to see if the platelets had arrived from Gartnavel. They had not. Accordingly, Dr Weir decided that the CT scan should not be delayed and accompanied Kathryn to the scanner. He did not recall Dr Beattie being with them.

[84] Dr Duffy remembered Kathryn arriving in the scanning room. Somewhat unusually, she was also accompanied by her father who was allowed to remain with her

during the procedure. Dr Weir and Dr Duffy were in the viewing room and were able to examine the images as they appeared. The scan was carried out at 01:07 hours according to the time noted by the computer on the actual images.

[85] Both Dr Duffy and Dr Weir were alarmed at what the scans revealed. The findings were later recorded by Dr Duffy in the medical notes at 01:45 hours in the following terms:

CT Brain – Provisional Report

Vertical axial CT brain
Four focal areas of acute haemorrhage as follows:
- 1.1cm intracerebral haemorrhage to the L temporal lobe with surrounding mass effect.
- 1.1cm intracerebral haemorrhage in the R basal ganglia with slight mass effect.
- 9mm intracerebral haemorrhage in the R occipital lobe.
- 2.9cm x 2.8cm intracerebral haemorrhage (mixed density) R parietal lob with significant surrounding mass effect."

- *Midline shift to the left of approximately 3.0mm.*
- *The fourth ventricle/basal cistern patent.*
- *Cannot confidently identify the 3rd ventricle.*
- *Sulci possibly effaced.*
- *Effacement of the temporal horn, R lateral ventricle.*
- *Possible enlargement of the R temporal horn.*

Results discussed with neurosurgeon and images sent to the Neuro-Institute

The report contained a reference to "midline shift". Such an observation is significant as it can be an indication of raised intracranial pressure. Dr Duffy measured the midline shift as 3mm which she considered was significant in a young child. The haemorrhages in Kathryn's brain were extensive in Dr Duffy's opinion. On viewing the scans her primary concern was that Kathryn had multiple acute haemorrhages which in her opinion were causing mass effect (adding to the volume inside the skull). This was indicative of a build-up of pressure in the brain and was potentially dangerous. This prompted two courses of action: first, urgent contact with the neurosurgeons at the

Institute of Neurological Sciences at the Southern General Hospital; and, secondly, Kathryn's immediate transfer back to the safer clinical environment of A&E resuscitation department in the event of sudden deterioration in her condition. A slight change in her condition while in the scanning department reinforced these decisions.

Episode in the scanning room

[86] Whilst in the scanning room, Dr Beattie described how Kathryn seemed to have some sort of episode when she told her father that she felt funny. She appeared to faint, losing consciousness for a few seconds and she vomited on the floor. She recovered quickly but the incident gave Dr Beattie a fright. This episode was not witnessed by either Dr Weir or Dr Duffy from their position in the viewing room. Neither could recall this particular incident, nor could Nurse Tait, the A&E nurse who also accompanied Kathryn to the scan and who remembered her. Her recollection was that the scan proceeded normally and was uneventful.

[87] Dr Weir recalled that at the end of the scan Kathryn seemed to be less well and not to be quite as alert as she had been prior to the scan. However, after speaking to her and rousing her a bit, she was able to open her eyes, speak and, after some prompting, she was able to obey commands. He made a retrospective note of that in the medical records. Nevertheless, Dr Weir was anxious that Kathryn be returned to a proper clinical area as the CT scanner was not a good place for acutely ill patients. Dr Duffy was equally concerned and considered that Kathryn needed to be in a more clinically safe area. Dr Duffy was concerned that Kathryn might develop clinical symptoms of raised intracranial pressure. If that happened, she was not in a safe, well-staffed environment to deal with any emergency that might arise. Accordingly, to allow Dr Weir to continue to

make further telephone calls and so that there was a doctor in attendance throughout, Dr Duffy accompanied Kathryn back to A&E and into the resuscitation area.

[88] Nurse Tait, too, was concerned and felt that Kathryn needed to get back to resus in A&E. She also recalled some deterioration in Kathryn's condition after the scan. She could not remember whether it had started when Kathryn was removed from the scanner back onto the trolley but she certainly remembered that Kathryn was more confused whilst they were *en route* to A&E. She described Kathryn as being "vague". She remembered asking her if she was okay but could not remember the response. When she asked Kathryn if she knew where she was, Kathryn was not sure. Kathryn's verbal response was the only aspect of her GCS that Nurse Tait checked as they were mobile at the time. She did not make a note of this but advised the medical staff on return to the A&E department. Dr Duffy advised Dr Thomas that Kathryn was back in the department as Dr Weir had remained in the scanning room making further telephone calls.

[89] A set of observations carried out on Kathryn seemed to indicate that she was back in A&E by 01:30 when her GCS was noted to have returned to 15. Dr Weir made a retrospective note in the following terms: *Patient more confused in CT scanning, but GCS 15. Plan to transfer to A&E resus.*

Contact with neurosurgeons

[90] Having examined the scans while Kathryn was still in the scanning area such was Dr Duffy's concern that she advised Dr Weir to contact the neurosurgeons. Dr Weir, too, was "very shocked and very, very worried" by what he saw on the images. Without

delay, he contacted the Southern General Hospital Institute of Neurosciences and spoke to the on-call neurosurgical registrar, Mr Ahmad. He timed the call at around a quarter-past one.

[91] Contact with the neurosurgeon involved several phone calls. It was not surprising, some eight years on, that there were discrepancies between Dr Weir and Mr Ahmad as to the exact number and content of the conversations which took place. Mr Ahmad also contacted his consultant, Miss Jennifer Brown, on several occasions in between these conversations. Mr Ahmad, in particular, had only a vague recollection of the calls and most of his evidence was reconstructed from the medical records. A great deal of time and effort was spent in somewhat futile examination of these witnesses as to their recollection of the minutiae of these conversations. Given that their memories were so vague, this was a fairly pointless exercise. However, I was able to discern the gist of the conversations. Decisions about Kathryn's management developed over the course of several conversations between Dr Weir and Mr Ahmad; Dr Duffy and Mr Ahmad; Mr Ahmad and his consultant, Miss Jennifer Brown. Two distinct stages could be identified: (i) initial contact between Dr Weir and Mr Ahmad and his subsequent contact with his consultant, Miss Brown which resulted in a decision to accept Kathryn for transfer to ward 66 of the Southern General Hospital for observation; and (ii) further discussions following the deterioration in her condition which led to a revised decision to transfer Kathryn to ward 61, neuro-intensive care, for surgery.

Stage 1: Initial contact and discussion

[92] Dr Weir wished the advice of the neurosurgeons in relation to Kathryn's scan and was, in fact, referring her to them to see if they would accept her for transfer. In the course of his initial phone call to Mr Ahmad, Dr Weir gave a full resume of Kathryn's history, presenting complaints, examination and findings. This included her deranged bloods results and her coagulopathy. I was satisfied that he also gave an indication of the differential diagnosis of sepsis or underlying haematological malignancy. Mr Ahmad was also made aware of the transitory episode in the scanning room. When it came to describing the findings of the scan, Dr Weir handed the phone to Dr Duffy so that she could provide a more accurate and detailed description to the neurosurgeon. Dr Duffy then reported the findings and arranged that the scans be shared electronically with the Institute so that Mr Ahmad could view them. During the conversation with Dr Duffy, Mr Ahmad requested that three further referrals be made: a consultant radiology opinion, a haematology opinion and a paediatric opinion. Beyond recalling those requests, Dr Duffy could not remember any further detail. She believed that she would have passed on the requests for the further opinions to Dr Weir. (Dr Weir had already given evidence and was therefore not asked about this.) As requested, she later contacted her own consultant, Dr John Calder, at home to obtain his advice. She could not remember the details of that conversation, nor the exact outcome other than that Dr Calder had no further input or advice. Therefore she assumed that he must have been satisfied with her interpretation of the scans.

[93] Dr Duffy then left to accompany Kathryn back down to A&E, together with Kathryn's father and Nurse Tait. Dr Weir remained behind in the scanning room and

continued his discussion with the neurosurgeon. At the conclusion of the conversation, Mr Ahmad told Dr Weir that he would discuss the case with his consultant and phone Dr Weir back. However, prior to the transfer of any patient, he required to discuss matters with his consultant.

[94] By this time, Dr Weir was "incredibly concerned about Kathryn" and was worried that Kathryn was not going to be accepted for transfer to the Institute. As he was waiting for the return call, he decided that it was an appropriate time to call Dr Vernon as the clinical picture had developed. He wished to discuss Kathryn with him: he was very anxious about her and felt that he "didn't have a lot else to offer" and therefore wished to check that there was nothing further he should have been doing and to obtain his consultant's advice and reassurance. His telephone conversation with Dr Vernon took place between 01:30 and 01:40.

Contact with on-call consultant (Dr Vernon)

[95] Dr Weir gave Dr Vernon a full resume of events, including that he had already spoken to Dr McAlpine. He discussed the scan result and conversation with neurosurgeon, the differential diagnosis and the discussion with the haematologist - "Basically I told him everything I could about the case because I was wanting his opinion and wanting him involved." Dr Weir estimated that the call had lasted about 10 minutes, finishing between 01:40 and 01:50. Dr Vernon agreed with the management of the patient. Dr Weir was reassured that Dr Vernon had confirmed that the plan was appropriate and that there was nothing else that Dr Weir should be doing and nothing

that he had missed. Dr Vernon did not offer to come in, nor did Dr Weir specifically ask him to do so.

[96] Dr Vernon remembered the call. He could not say from memory when he received it but from the records he worked out that it must have been around 01:30. As he recalled it, Dr Weir was not really seeking his advice because by then a possible working diagnosis had been made and contact had been made with the neurosurgical unit at the Southern General. His impression was that Dr Weir was looking for confirmation that this was a sensible plan for a case that was extremely difficult where a girl was not at all well. Septicaemia had, according to his recollection, been the initial working diagnosis but that after the scan showed haemorrhage in different areas of the brain together with the blood results Dr Weir had an underlying haematological malignancy as the more likely working diagnosis. From the presenting history and examinations as explained to him, which included the blood results and a description of the CT scans, Dr Vernon thought that it suggested some form of myeloid leukaemia although it was not clear whether he said that to Dr Weir at the time. Dr Weir had made him aware of the localising neurological deficit with a left facial palsy. Dr Vernon was aware that at the time of the telephone call Kathryn's GCS was 15.

[97] He believed that Dr Weir told him that he had contacted the haematologist who had said he would issue a pool of platelets and some whole blood. He advised that a blood film should be ready in the morning and that he should be re-contacted again in the morning if further platelets were required. As for the reference to a blood film being ready in the morning, Dr Vernon could not recall whether this came from the

haematology laboratory. From Dr Weir's evidence, however, I was satisfied that this reference to a blood film was in fact a reference to the bacteriology investigations, not haematology.

[98] Dr Vernon approved Dr Weir's actions. The plan for Kathryn seemed entirely reasonable given that this was a girl with one very significant bleed in the brain that was causing some distortion of the brain and she also had, as he recalled it, three or four other areas of bleeding. He checked that Dr Weir had organised blood products, including platelets, and he asked about the transport arrangements to the Southern General.

[99] Although he could not remember whether he had either offered or had been asked to come in to the hospital, it was his normal practice to offer to do so. He did not go in and thus assumed that on this occasion he assumed that Dr Weir did not particularly want him to come in.

Contact with on-call consultant neurosurgeon

[100] At the stage at which he phoned Miss Brown, Mr Ahmad's initial plan was to transfer Kathryn to Ward 66 in the Institute, the paediatric neurosurgical ward. He did not anticipate surgery at that stage because Kathryn was stable and her "neurology" had not shown any significant change. Nevertheless, there was a risk that neurosurgical intervention would be required if Kathryn's intracranial pressure rose. Immediately following his conversation with Dr Weir, Mr Ahmad telephoned his consultant at home and explained the situation to her.

[101] Miss Brown remembered that she had been contacted more than once - she recalled at least two conversations – about Kathryn. It was her recollection that the results of the scan were known by the first call. She believed that she had initially been told that this was a child with a scan that showed several areas of focal neurological deficits and that several areas had been interpreted as haemorrhage. At the time of the referral she was conscious. That was the detail Miss Brown could recall but she knew that more information would have been discussed at the time. There was a concern that this child had an underlying illness: there was low haemoglobin and a high white cell count. The precise figures of the blood results she could not now recall (although she would almost certainly have been given the platelet count). The concern was about a differential diagnosis of sepsis or a haematological malignancy. The latter was also based on clinical signs of a coagulation problem as suggested by the history. Epistaxis, bleeding from the gums and spontaneous bruising were the features that she could recall. This was information which she believed had been passed on to her by Mr Ahmad. He would have seen the scans by that time and described them to her.

[102] From the witness box, Miss Brown described the scans to the court and confirmed her impression that there were features on the scan that suggested raised intracranial pressure, a degree of midline shift being one of those signs. These scan results were described as "very worrisome". There was a difficult clinical picture of a child with a bleeding diathesis (abnormal clotting and a predilection to bleeding) therefore prone to bleeding with multiple sites of intracranial haemorrhage associated with signs of raised intracranial pressure. The fact that these areas of bleeding were all within the brain

rather than on the surface was extremely worrisome. She considered that the full clinical picture was of great concern.

[103] According to Miss Brown, the initial plan was to bring Kathryn to the Institute to ward 66, the paediatric ward. The purpose was for observation. She explained that there was a potential for the sort of deterioration which Kathryn did, in fact, undergo. The plan was to have Kathryn in the Institute should that happen and there were a need for surgery. She would be in the best place to perform these observations, close to the site where any possible surgery would take place. Miss Brown explained that, in such circumstances, deterioration could happen very precipitously. During that period of observation, treatment for the underlying condition and the replacement of abnormal clotting factors could take place as far as possible. Accordingly, Miss Brown authorised Kathryn's transfer on that basis although events were to supersede those initial plans.

[104] Mr Ahmad contacted Dr Weir to let him know that they had accepted Kathryn's referral and that arrangements should be made to have her transferred to Ward 66 where a bed was available. Dr Weir was very relieved. He estimated that he received this call shortly after he finished his discussion with Dr Vernon and about the time he returned to A&E. He thought that to have been at approximately 1:50am. He could not remember whether he was still in the scanning room when he took the call or whether he had already returned to A&E.

Deterioration in Kathryn's condition

[105] On his return to A&E, Dr Weir immediately became aware of the presence of the ITU team around Kathryn, assessing her condition. Dr Weir did not know at that time

who had called them or why. However, he was told that Kathryn's GCS had dropped. Dr Weir approached the ITU team to let them know about the result of the scan and that he had been in touch with the neurosurgeons.

[106] It was not clear from the evidence how it came about that the ITU team was called to A&E but it can reasonably be assumed that it was in response to a sudden and precipitous deterioration in Kathryn's condition and a call for urgent assistance from A&E. There was no relevant entry in the medical case notes but the nursing observation notes document a drop in GCS from 15 to 8 (a person with a GCS of 8 is considered to be in a coma), blood pressure rising to over $^{130}/_{70}$ and an increase in her respiratory rate. Her right pupil was noted as unchanged in size but not responding.

Intubation and preparation for transfer

[107] Kathryn's parents described sudden activity around Kathryn and Dr Rosaleen Beattie described it as "a bustling event". Their evidence was that the family had had no warning about this. Both Dr Beattie and his sister were adamant that they witnessed no deterioration in Kathryn's condition before this and insisted that right up until Kathryn was anaesthetised by the ITU team she was fully conscious and alert, so much so that at the point of intubation she said to her father words such as "Stay with me, Daddy" or "Don't leave me, Daddy". This did not reflect the situation as described by the ITU team. That was not the situation according to the consultant anaesthetist in charge, Dr Allan Davidson, who described Kathryn as in a coma immediately prior to intubation, that being the reason for the procedure. This is considered later at paragraphs [366]-[373].

[108] First to respond to the call was Dr Diana Raj, then an SHO in anaesthetics who was on call for ICU. She took the call from A&E to the effect that they needed help with a child there. She had little direct memory of events and what memory she had was fairly vague but was aware that she had at once asked for her consultant, Dr Davidson, to be called in as she did not have the requisite experience in paediatric anaesthetics. She did remember that there were a lot of people around Kathryn. Of Kathryn's condition, she recalled that her eyes were not opening spontaneously and there was no verbal response. She was aware that Kathryn was to be transferred to neurosurgery at the Southern General and it was clear to her that Kathryn needed to be intubated and ventilated for transfer so as to secure her airway. She had a vague recollection of having been told about some deterioration in the scan but other than that she could not recall any detail of the incident. Much of her evidence was couched in terms of what she would have done in such circumstances. She herself did not take part in the intubation process.

[109] Dr Davidson, Intensive Care Consultant Anaesthetist, was on-call at home when he was contacted – he thought at about 01:30-ish - from the charge nurse in intensive care. The call was about a young girl with a suspected intracranial bleed who was not very well. Aware that she was to be transferred to the Southern General, he asked the charge nurse to call in Dr Stephen Jeffrey who was the senior on-call anaesthetic registrar who would have to accompany Kathryn in the ambulance.

[110] He immediately made his way into the hospital arriving in A&E sometime around 02:00. On arrival he saw that Kathryn was not conscious. Her GCS was 7-8: she was not verbalising or vocalising, she had some degree of movement but it was not normal

movement and she was not eye opening. He made the decision to intubate: the low GCS level combined with a worrying deterioration with an intracranial bleed in the face of a worsening situation meant that she clearly needed to go to the Southern General. For Kathryn's safety during transport – to protect her airway and facilitate ventilation – and also to start to treat the potential rise in intracranial pressure, it was decided to induce anaesthesia. Dr Davidson was also at that time aware of Kathryn's blood results and of the four discreet areas of haemorrhage in Kathryn's brain. Dr Davidson described the situation to which he was called as "really quite desperate". It was a very worrying situation and the priority of the anaesthetic team was to get Kathryn safely across to the Southern General for treatment.

[111] Dr Davidson thought that he had administered the anaesthetic drugs while it was Dr Jeffrey who performed the actual intubation. He was in A&E for about thirty to forty-five minutes before Kathryn was transferred. It was pointed out during this time that ward 66 would not accept an intubated patient and that she would have to go to ward 61, neurosurgical intensive care. It was arranged that Dr Jeffrey would accompany Kathryn in the ambulance.

Further contact with neurosurgeons

[112] As all this was happening, Dr Weir again contacted Mr Ahmad to advise him of the significant deterioration in Kathryn's condition. Mr Ahmad was given full details about the changes in her condition. He recalled that he was told about the change in her blood pressure and that her pulse rate had dropped to 48. He recorded in the cases notes

Before transfer she dilated her R pupil with sluggish reaction and also drops her pulse to 48 when she was give atropine.

[113] A few lines above that entry where he noted information about Kathryn's presentation to A&E, he has inserted a reference to the deterioration in her level of consciousness:

Dropped her GCS to E1 V1 M5 – in other words a score of 7 from 15.

[114] Thus he was advised that Kathryn's conscious level had dropped to 6 or 7 and she had been given Atropine to increase her heart rate. On receipt of this alarming information, he again contacted Miss Brown.

[115] Miss Brown, with reference to the medical records, timed this call as having been received somewhere between 02:00 and 02:30 hours. She was informed about the change in Kathryn's neurological condition. She was informed of a combination of events that were highly suggestive of a now life-threatening raised intracranial pressure. These events were a papillary (pupil) asymmetry, at the same time a drop in conscious level and changes in the blood pressure and pulse rate. She could not remember the exact details of the pupil sizes but recalled that the pupil on the right side, the side of the largest haematoma, had increased in size (dilated) and was non-reactive. That indicated that the largest of the haemotamas was symptomatic and could shift from one side to the other impinging on the third (III) nerve on the right side which in turn would cause the right pupil to enlarge and become unreactive.

[116] Things were happening quickly at that time and at the stage of the second call, Kathryn was either intubated or was in the process of being intubated. Miss Brown had been made aware of the event in the scanning room but this call was about the much

more severe event. She remembered having been told that Kathryn's heart rate had dropped to 48 and that she was in a coma. In these circumstances, Miss Brown considered that surgery was necessary at this point to relieve a rise in intracranial pressure which was immediately life-threatening. *Everything changed with that deterioration.*

[117] Kathryn's destination was changed from ward 66 to ward 61, the intensive care ward. Miss Brown advised Mr Ahmad that Kathryn needed immediate transfer and that she should go immediately to theatre for a right frontal craniotomy. She explained in court that this was almost certainly a situation where time was of the essence and, consequently there would not be enough time to confirm normalisation of clotting parameters prior to surgery. She instructed that all available blood products should be transfused and a post-transfusion repeat full blood count to be done. More blood, platelets and FFP should be arranged.

[118] Mr Ahmad passed that information to the Victoria and also instructed that Kathryn be given Mannitol (100ml of 20% given stat – at once - over a period of 20 minutes) to reduce pressure in the brain. Kathryn left the Victoria Infirmary at 02:45.

[119] Several aspects of Mr Ahmad's evidence require comment. First, in the course of his evidence Mr Ahmad made reference to a "blood film". However, it quickly became clear that he was confusing terminology and he was referring to blood results. He was told about a suspicion of "some haematology". First he said that a blood film was referred to during his first conversation with the lady doctor. Then he corrected that and

said that it was in the course of the second conversation that a reference was made to a blood film.

[120] Secondly, he said "I was told that there is a haema…like, a, acute myeloid leukaemia." When asked if this reference to acute myeloid leukaemia was made in the first or second phone call, he could not remember, nor could he say that it was a male or female doctor who had told him that. He became hopelessly confused and his answers to very specific questions were incomprehensible. It seemed to me that when first referring to this he started to say "a haematological malignancy" which would have accorded to the evidence of Dr Weir and the case notes from the Victoria Infirmary. He changed that mid-word to "acute myeloid leukaemia". I did not accept that part of his evidence. It seemed to me that the most likely explanation for this was that he had transposed what he later read in the notes into what he thought was his recollection. His evidence on this was totally confused and unreliable. Moreover, it was telling that nowhere did he write down that he had been told anything about acute myeloid leukaemia. Rather, his notes contain the phrase "?haematological Malignancy". Nor had he conveyed any such information to Miss Brown at any stage. Accordingly, I am entirely satisfied that no mention of AML was made to Mr Ahmad at that time. This confusion on his part was most unfortunate as it appeared to the family that this confirmed their position that a diagnosis had been made at the Victoria Infirmary. I make it clear that I have rejected Mr Ahmad's evidence about this.

[121] Likewise, I have rejected as unreliable his reference to a blood film. On more than one occasion he appeared to be confusing blood *film* with blood *results*. I was unable to

make sense of his answers about this. Once again, his evidence gave rise to unwelcome obfuscation about a disputed matter. I was, however, entirely satisfied that no mention was made of haematology blood films during his conversations that night.

[122] Mr Ahmad was adamant that he told the referring doctor to involve a senior paediatrician and that they had to sort the blood problems out before they would consider any neurosurgical intervention. Repeatedly he volunteered this with great emphasis during his evidence. Likewise, he said many times that it was not for him to sort out the blood problem but he "expected the primary team to carry downward my initial instruction…to correct the abnormality of the blood". I regarded this evidence with considerable skepticism particularly as he did not appear to have followed this up or clarified the position at any time once Kathryn was transferred into his care.

[123] Mr Ahmad's notes contained references which were not mentioned anywhere else. This was surprising as Mr Ahmad's history was compiled second-hand from information obtained from his medical colleagues: he did not take an independent history from Dr Beattie or Ms Crawford and, of course, Kathryn herself was unconscious by then. His notes referred to a "mild left-sided hemiparesis" (a weakening of the left side) which was not recorded anywhere else. Neither was there any mention elsewhere of there being a suggestion that Kathryn's planter reflex was in the upwards direction. His notes also contained a reference to "polymenorrhea for 9 days". This in itself is inaccurate terminology referring to periods that are too frequent instead of heavy periods which was what he meant to record.

[124] Mr Ahmad volunteered that in his training he had always been told that if a female patient had "deranged blood" a question should always be asked about their menstrual periods. This could give an idea whether the situation was acute or chronic. Accordingly, it was Mr Ahmad's recollection that he had asked specifically about Kathryn's periods and had been told by the referring doctor that she had polymenorrhea for nine days. This formed part of the history which he recorded in Kathryn's notes. This is the only reference to Kathryn's periods in her case notes. Although there was a suggestion during the course of the evidence that Kathryn had experienced heavy periods, Kathryn's mother confirmed that while Kathryn's periods had started, she denied that she there had been had been any problems of heavy periods. I was unable to reach any firm conclusions about this. However, I did not regard any of this to be of particular significance.

Transfer to Institute of Neurological Sciences, Southern General Hospital

[125] Dr Jeffrey could not remember the case at all. This was unfortunate as no notes of the actions of the anaesthetists were available. According to Dr Davidson, these must have been lost because they undoubtedly did write down exactly what the findings were, what drugs were given and their plan. These notes would have been filed loosely on single sheets and not bound together. It was his experience that such notes did go missing from time to time. However, he explained that normally no notes are made of the actual transfer. The practice is for there to be a verbal handover between the transferring and receiving anaesthetists.

[126] There was one entry in the prescribing notes where Dr Jeffrey had signed for Tazocin, a broad spectrum anti-biotic at 02:30. Other than that, Dr Jeffery could recall nothing of his involvement with Kathryn. He explained that an anaesthetist summoned to A&E to help with the intubation of a patient would usually separately examine and assess the patient with a view to deciding the best management of that patient. He would normally have made notes of this process in separate continuation sheets (simply bits of lined paper) that would be added to the patient's medical notes. These case notes would normally remain with the patient. He would include details of any drugs administered, particularly in connection with the anaesthetic, his anaesthetic assessment and the procedure so that all the information about the anaesthetic would be kept in the one place. It was clear that at 02:30, when he prescribed and administered Tazocin, he had access to the Kathryn's medical records. It was also clear that at about that time the records would have been away for photocopying so that a set was available to go with her to the Institute.

[127] Dr Jeffrey explained that medical records would normally accompany the patient on transfer. However, on arrival, the patient would be met by the ICU anaesthetist and there would be a verbal transfer between the accompanying anaesthetist and the receiving one. The handover would involve passing on a full clinical history and what so far had been done with the patient, including details of anaesthetic drugs administered.

Disputed evidence

[128] Before moving on to consider events at the Southern General Hospital, this is an appropriate point to consider two areas of the evidence arising from events at the

Victoria Infirmary of about which there was fundamental disagreement. The first concerned the information supplied to doctors by Kathryn's parents; the second related to whether a diagnosis of acute myeloid leukaemia was made at that time.

Accuracy of A&E notes of history

[129] Dr Beattie and Ms Crawford were adamant that Kathryn had not suffered from epistaxis (nose bleeds), bleeding from the gums, nor had she had spontaneous bruising or confusion. In the witness box, Ms Crawford was outraged that these references had been included in the notes, variously describing it as "absolute nonsense", "disgraceful" and "unbelievable". Her reaction was fairly extreme. It was Dr Beattie's position that Kathryn could not possibly have had a nosebleed without his having been aware of it. Her eyes had not been staring nor had Kathryn been confused as Dr Thomas had noted. Dr Thomas had misinterpreted information given about the episode of speech difficulty. She had noted that it had lasted for half-an-hour. That was strenuously denied. It was suggested that Dr Thomas had mistaken that for information provided about *when* it had happened - namely, about half an hour before. It was their position that this information had been inaccurately and falsely included in the notes. They had not seen Dr Thomas make any notes at all during her examination of Kathryn. She had did not have pen or paper with her. It was their contention that Dr Thomas had deliberately included these symptoms. Likewise, Dr Weir had also wrongly noted the history.

[130] Dr Thomas' evidence was that she had noted what information she was given at the time. It was her invariable practice to make contemporaneous notes. It was not possible that she had taken no notes at the time. As a doctor in a busy casualty

department where she might see twenty patients in an evening, it would be impossible for her to remember details of all patients and write up their notes retrospectively. Dr Thomas could remember Kathryn's case very clearly in general terms. Some of the detail was understandably vague after so long but she did recall taking notes, just as she remembered "where the family members were stood, sat or lying". Moreover, the fact that she had written 22.07 in the margin indicated that she had recorded the notes at the time. Had she occasion to write anything in a patient's notes after the event, she would round the time up or down and would state that the notes were written retrospectively. I had no hesitation in accepting Dr Thomas' evidence that she had, indeed, noted details of the history and examination contemporaneously in accordance with her invariable practice.

[131] As to the accuracy of what was contained in the notes, it was Dr Thomas' position that she simply noted the information she had been given. As regards the dysathria, it was important to determine how long the incident had lasted. That would have been specific information that she had asked for and the fact that she had written "episode *lasted* – half hour" in the notes showed that she could not have misunderstood the position.

[132] Kathryn's parents described how Dr Weir asked some brief questions but took no separate history. Dr Weir's position was that he proceeded to take his own history, perform his own examination and form his own conclusions. This approach was universally endorsed by the expert witnesses as the correct approach. Dr Weir had also

noted down what he had been told. It was Dr Beattie's position that Dr Weir, too, had fabricated information contained in his notes.

[133] This open attack on Dr Thomas and Dr Weir raised stark issues of credibility. Shortly thereafter, information came to the attention of the procurator fiscal which related to Kathryn's condition in the period prior to her admission. This led to further investigations and the leading of many witnesses who spoke in conflicting terms about Kathryn's condition in the days before she died. However, given that the focus of this Inquiry concerned the treatment that Kathryn received in hospital and given the clear evidence that she was already very seriously ill by the time she presented at the Victoria Infirmary, her condition in the run-up to her admission was not of direct relevance. It should be made absolutely clear that no criticisms were directed at Kathryn's parents. It is well established that by the time clinical symptoms become overt in leukaemia the disease is often at an advanced stage. Furthermore, the early symptoms are often associated with apparently benign viral illnesses or infections. Accordingly, it is neither necessary nor in the public interest that this aspect be explored in depth in my determination. I am satisfied that I can reach conclusions as to what happened on the evening of Kathryn's admission without reference to that rather unfortunate chapter of evidence.

[134] I had no hesitation in accepting the evidence of Dr Thomas and Dr Weir and that the notes were accurately recorded by them. Both were credible and reliable witnesses. Dr Thomas struck me as a very bright, intuitive doctor. She had immediately recognised that Kathryn was very ill and required admission. There was never any suggestion, as

her parents' thought, that Kathryn was going to be sent home. Dr Thomas had set in motion all of the appropriate tests initial tests in order to determine what was wrong with Kathryn.

[135] Dr Weir, too, looked after Kathryn with great concern and diligence. He took forward the additional tests required, including the CT scan and he made appropriate contact with the on-call haematologist. He was very methodical and took great care in compiling detailed notes so that he could fully and properly discuss Kathryn's condition with others, including his own consultants. He was somewhat slow and hesitant in giving his evidence and was less confident that Dr Thomas. However, I accepted his evidence as both honest and essentially reliable.

[136] Their position was, to some extent, supported by the evidence of the paramedic, Mr Fleming, whose colleague had likewise noted that the history provided at home included epistaxis, sores in the mouth and that Kathryn's speech had been slurred and confused. This evidence was not challenged. In his submissions, Dr Beattie suggested that it accurately reflected the position and that the reference to epistaxis must have been something mentioned by his wife that Kathryn had once had a nose bleed in France.

[137] There was evidence, too, from Dr Bruce, Kathryn's GP, that when he spoke to Dr Beattie that night, Dr Beattie had said that Kathryn had being experiencing confusion and speech difficulty over a two hour period. I have to say that from the outset I found it odd, if Kathryn appeared to have been recovering from a mild viral infection, why it was that Dr Beattie was so anxious to contact his sister about Kathryn's condition on the

Sunday night on her return from Orkney. This was apparently prior to the speech event. His evidence was that it was to have her give Kathryn "the once-over" and to get his sister's advice on the next steps in relation to his suspicion that Kathryn was clinically anaemic. That seemed strange given that, as a GP, Dr Beattie himself might have been expected to know what these next steps might be. These were aspects of the pre-hospital events which I found troubling.

[138] Furthermore, I found the parents' challenge about the clinical history quite mystifying. It would have been understandable had their complaint been that these important, highly relevant symptoms had *not* been noted when described, perhaps leading to a delay in diagnosis. But their complaint was that symptoms that *were* important pointers towards the diagnosis of leukaemia had not only been erroneously recorded but fabricated. If, as Dr Beattie avers this was done as some sort of cover-up, then it seemed entirely counter-intuitive and made no sense.

Timing of "diagnosis"

[139] The second issue which was in dispute related to the timing of the diagnosis of leukaemia. It was the family's firm position that the diagnosis had been conveyed to them by Dr Thomas shortly before 23:00. Dr Thomas denied having made any such diagnosis. Such starkly opposing evidence seemed difficult to reconcile.

[140] Kathryn's father and aunt were absolutely certain that Dr Thomas had told them that the diagnosis was one of leukaemia. Dr Beattie described it as a "Kennedy moment". Shortly after receiving the first set of blood results, he recalled that Dr Thomas took another call. From her demeanor he could tell it was bad news. It was then that she came

across to him and said that the results looked as if Kathryn has acute myeloid leukaemia. Dr Beattie asked her if this was just a possibility or if it was certain, to which she replied that it was fairly certain. Dr Beattie was certain that she had not simply mentioned acute myeloid leukaemia but had said that it was a type of AML. He could not say if she had actually used the term "acute promyelocytic leukaemia" because that would not have registered with him at the time. At a later stage in his evidence, Dr Beattie told the court that the first time he had been told of the diagnosis of APL was after Kathryn's death.

[141] Dr Rosaleen Beattie was also certain that the diagnosis had been made while they were in the Victoria Infirmary. It was, she said, a very clear and certain memory. As she remembered it, Dr Thomas had said that Kathryn's white cells were abnormal and it very much looked like she had some form of leukaemia. She said that there was no doubt that it was acute myeloid leukaemia. Ms Crawford remembered that she had been standing a bit back and did not hear all of the conversation. She heard the word "leukaemia". She heard Dr Beattie ask if it was certain, to which Dr Thomas replied something like "almost certainly". They were profoundly shocked by this news.

[142] There was little doubt that Kathryn's parents and aunt genuinely believed that they had been given a definitive diagnosis. The difficulty with that is that, at that stage and on the information then available, a diagnosis was medically and scientifically impossible.

[143] Dr Thomas was equally adamant that she had not had a conversation in which she told Kathryn's parents that the diagnosis was one of acute myeloid leukaemia. She was not qualified now to make such a diagnosis and she certainly would not have been able

to make it eight years ago. In any event, the diagnosis could not have been made without haematological input and a blood film. At that point, the diagnosis was not confirmed – it could still have been a sepsis with associated bone marrow failure. She would never have given a diagnosis of something as catastrophic as leukaemia without being absolutely sure or in circumstances where she would have been unable to provide any further information as to what was to be expected or the treatment involved.

[144] From the haematological evidence, it was abundantly clear that in the absence of a blood film, no diagnosis could have been made. Even then, it could only have been made on a provisional basis. Such a "diagnosis" could only have been made by a haematologist. At the time Dr Thomas was supposed to have given the diagnosis, Dr Weir had not yet contacted haematology. Thus the medical evidence about this was unequivocal. No diagnosis could have been made at that time.

[145] However, it seemed to me that there was scope to reconcile the two accounts. Dr Thomas had some sort of underlying haematological malignancy in mind from the outset. She was not surprised when Kathryn's blood results came back: she had expected them to be very abnormal. From her evidence and that of Dr Weir, it was evident that while both had a differential diagnosis of sepsis or haematological malignancy, Dr Thomas clearly favoured the latter. She said that she thought it might be leukaemia. In answer to questions from the court, she readily accepted that she "might well" have said something along the lines of "I'm very much afraid it looks like leukaemia". That might well have been the "Kennedy moment". That evidence is compatible with Dr Beattie's later evidence when he said that Dr Thomas gave him information about the likely

diagnosis and implied that it was fairly definite. Dr Rosaleen Beattie told the court that leukaemia was a diagnosis that she herself suspected but was trying to put to the back of her mind in the hope that she was wrong. In these circumstances, what Dr Thomas said would still have been devastating news, never to be forgotten, and which, as it turned out, was accurate requiring no revision.

[146] In passing, mention should be made of the contention that information which would have enabled a diagnosis to have been made at that time must have come from Mr McLauchlan, the biomedical scientist. It was suggested that this information could have come from a diagrammatic "scatter plot" produced by the analyser machine. There was no evidence that Mr McLauchlan had looked at any scatter plot that night.

PART 3 EVENTS AT THE INSTITUTE OF NEUROLOGICAL SCIENCES, SOUTHERN GENERAL HOSPITAL, GLASGOW

Arrival at the Southern General

[147] Kathryn arrived at the Southern General Hospital at 03:00 and was admitted to ward 61, the neurological intensive care unit. The receiving anaesthetist at the Southern General was Dr Craig Urquhart. He was a senior house officer on attachment to the ward 61. He was in his fourth year as an anaesthetic trainee. He was the resident anaesthetist but there would also have been two on-call anaesthetists – two consultants or a consultant and a senior trainee.

[148] Dr Urquhart was first warned of Kathryn's imminent arrival by Mr Ahmad. He could not specifically recall but was sure that he would have been given the child's age and the two most relevant features of the presenting diagnosis, namely intracerebral haemorrhage and a low platelet count or thrombocytopaenia. At that stage, Dr Urquhart telephoned his consultant Dr Wagstaff who was on call at home. This was simply a courtesy call to advise her that a patient as being admitted to ICU for possible surgery. Dr Wagstaff was not asked to come in.

[149] Dr Urquhart estimated that transfer time from the Victoria Infirmary would have taken 20 to 30 minutes. Kathryn was brought straight to ICU where, in accordance with normal practice, she was received by Dr Urquhart, her named one-to-one nurse, Nurse Rhona Twaddle and the ICU charge nurse. A verbal handover took place between Dr Jeffrey and Dr Urquhart. Dr Urquhart noted the presenting diagnosis as:

1. *Intracerebral haemorrhage 2. ?sepsis and 3. Thrombocytopaenia.*

and Kathryn's history as:

13 year old girl with 6 day history of malaise, flu-like symptoms

Epistaxis and bleeding from gums

Presented to A&E with L facial droop and dysathria.

[150] The deterioration in her conscious level was recorded: GCS was noted as E4 M6 V5 i.e. 15 prior to her CT scan then dropping to E1 M5 V1 i.e. 7 post-scan and prior to intubation. Dr Urquhart noted that she had been given platelets and transferred. She had been given Gentomycin, Tazocin and Ceftriaxone (all antibiotics). Mannitol and Atropine had also been administered, the first to help reduce the ICP, the second to treat the bradycardia and increase her heart rate. The notes contained no reference to any ?underlying haematological malignancy or any mention of leukaemia of any type. Under the heading "Current Problems and Impression" Dr Urquhart wrote

1. *Intracerebral haemorrhage*
2. *Thrombocytopaenia*
3. *Anaemia*
4. *? cause sepsis / autoimmune???*

[151] Dr Urquhart noted Kathryn's heart rate at 96 and her blood pressure as $^{135}/_{80(110)}$. No reference was made to underlying haematological malignancy as part of the differential diagnosis in Dr Urquhart's notes. However, as Mr Ahmad and Miss Brown were fully aware of the possibility of such a diagnosis, its omission is of little relevance.

[152] Once Kathryn arrived in ICU, a series of observations were taken. These were carried out by Nurse Twaddle within a minute or two of her arrival. It was noted that on arrival Kathryn's pupils were unequal and were fixed and dilated. An entry timed at

03:00 noted on Kathryn's chart recorded her pupils as Right 7, non-reacting and Left 5, non-reacting. In this connection, Miss Brown commented that the effect of the Mannitol given at the Victoria Infirmary was not long in Kathryn's case because her pupils were again asymmetrical by the time she reached the Institute. Her blood pressure was recorded as $^{130}/_{70}$. Mr Ahmad instructed that Kathryn be given 100 mls of Mannitol to reduce brain swelling. This was a second dose and according to Nurse Twaddle it was given, with no effect.

[153] Mr Ahmad examined Kathryn once she was in ITU. He transposed the information he had noted in the course of his conversations with staff at the Victoria into the hospital case notes before noting the results of his own examination.

21/06.04 3:30am
Emergency referral from Victoria Infirmary.
13yrs 10 month old girl unwell for last 4-6 days when she had polymenorrhea for 9 days followed with Mild Intermittent Headache + drowsiness for last 2-3 days, developed spontaneous bruises, epistaxis and bleeding from gums, left-sided facial weakness + confusion earlier in the night when she presented to Victoria Infirmary A.E.
weakness with mild left hemiparesis with Babinski reflexes and ?up going left Plantars and then dropped her GCS to E1 M5 V1

- *No ear discharge but Multiple Bruises over her shin* (or skin) *and thigh.*
- *- Before transfer she dilated her Rt pupil with sluggish reaction and also drops her pulse to 48 when she was given atropine. She was tubed and transferred to SGH.*

On arrival/
-Tubed and Ventilated
Pulse of 94/min BP110/70.
Pupils Rt 5m left 2mm
Dilated and not reacting secondary to Atropine

- ➔ *Bleeding from gums with multiple bruises over the hands and lower limbs.*
- *No Abnormal masses or hepato spleeno megaly*
- *CT Scan. Multiple Intra Cerebral Haematoma with mass effect from Rt frontal haematoma.*

Spontaneous Haemorrhage due to Thrombocytopaenia and Anaemia.
? haematological Malignancy
ITP

-Plan> Platelets transfusion given – 1 pool given
FFP
2 units of blood
Needs craniotomy once platelets above 100,00
Discuss with patient's parents, Father is a GP and Aunt a ?Rehab Consultant.
-Discuss with Miss Brown. Platelets 69000 on repeat count.
- We will take for craniotomy.

[154] Further discussion with Miss Brown took place and she instructed that a repeat platelet count be done. Mr Ahmad noted at this point

Plan. Platelet transfusion – 1 unit given.

FFP

- *2 units of blood.*

– needs craniotomy once platelets is above 100,00.

[155] Certain blood products were prescribed. Kathryn was given one bag of platelets at 04:00 in accordance with the instructions which were to transfuse these after her platelet count had been received. The result of the platelet count was noted at 04:00 which showed that her platelets were 69. The platelets were then immediately transfused. The resulting increment might well have taken Kathryn's count to around a hundred.

[156] There was then a note of his discussion with Kathryn's parents. This was the point at which Mr Ahmad obtained consent to surgery from Kathryn's father.

Assessment by Mr Ahmad

[157] According to Mr Ahmad, by the time Kathryn arrived at the Institute, there had been a significant change in her clinical condition. The deterioration had already begun in the Victoria where her left pupil was small but still reacting. When she arrived at the Southern General a set of observations were taken by the ICU nursing staff. They had recorded that at 0300 hours her pupils were dilated and not reacting: R 7 and not reacting, L 5 and not reacting. About ten or fifteen minutes later, after Kathryn was settled in ICU, Mr Ahmad saw her and noted her pupils differently: the right pupil was

dilated to 5mm having been 3-4 at the Victoria where the left was recorded as 3, reacting. Her left pupil was 2mm. Mr Ahmad 's note read

Pupils Rt 5 mm left 2mm
Dilated and not reacting secondary to Atropine.

[158] Significantly, both pupils were non-reacting. Mr Ahmed preferred his assessment of the pupil size to that of the ICU nursing staff although he conceded that both could have been correct but that there had been a change in the intervening 20 to 30 minutes. A further dose of Mannitol had been given in the intervening period.

[159] It should be noted that some references appear in Mr Ahmad's notes which were not spoken to or noted by anyone else. This was odd as Mr Ahmad took no direct history himself and relied solely on information he had been given by others. The inclusion of such information (for example reference to polymenorrhea, weakness, mild left hemiparesis and to ?up-going plantars) was a matter of concern to Dr Beattie and Ms Crawford. However, years later, it is not something about which I have been able to reach any firm conclusion.

Consent to surgery

[160] Mr Ahmad spoke to Dr Beattie to obtain consent for Kathryn's operation. In the course of the Inquiry, it became clear that the information provided by Mr Ahmad to the family as to the risk associated with the surgical procedure (craniotomy) was incorrect. Mr Ahmad himself accepted that he had made a mistake and that he had also omitted to sign the consent form. It was Dr Beattie's position that his consent had been improperly obtained. His consent was given on the basis that the surgery was necessary in the sense

of life-saving; that Kathryn's thrombocytopenia would be addressed before surgery commenced; that the operation would be carried out by Miss Brown herself; and that the risks were as explained by Mr Ahmad.

[161] Mr Ahmad explained the risk of the craniotomy in the following terms, as noted on the consent form:

RIGHT CRANIOTOMY FOR EVACUATION OF FRONTAL HAEMATOMA

Risk: Infection, haemorrhage, Strokes, Fits, Re-operation and < .01% even death.

Dr Beattie equated that with a 99.99% chance of success.

[162] Mr Ahmad, understandably, had only a vague recollection of the conversation. His evidence on this matter was confused and unclear. I have to say that in general I found Mr Ahmad's evidence very difficult, at times impossible, to follow and that caused me to wonder just how clearly he had communicated with Kathryn's parents. He conceded that he had not signed the form, explaining that things were moving quickly and he "may have omitted some important bits". He admitted that he had put the decimal point in the wrong place on the form. The figure should have been 0.1% reflecting a risk of one in a thousand. This was the figure for the percentage of patients who die during the operation.

[163] The information contained in the consent form did not properly reflect the risk of the procedure. Miss Brown confirmed that the risk of death in Kathryn's case, given her neurological condition, was really very high. She would have put her chances of survival at well below 50%. A craniotomy in a child with spontaneous intracerebral

haemorrhage and a coagulopathy was a high risk operation. This was confirmed by the expert witnesses.

[164] As will be seen, the conditions on which Dr Beattie gave his consent did not materialise. If he had known this at the time, Dr Beattie maintained that he would have refused to sign the consent form. Had that been the case, then it seems clear that Kathryn would have died very soon afterwards.

Surgical Procedure

[165] It had been Miss Brown's intention to perform the craniotomy herself. This was in part because the patient was a child whose prognosis was so poor. She had therefore instructed that she be contacted at home when Kathryn was in the anaesthetic room. It was her aim to arrive just as the operation commenced. It would have taken about twenty minutes to prepare her for surgery once in the anaesthetic room, which was about the same time that it would take Miss Brown to come into the hospital from home. In the event, she did not perform the surgery but was present during it in a supervisory capacity. The procedure itself was carried out by Mr Ahmad. Prior to coming in, her position was that, in accordance with her usual procedure, she would have instructed him to get started. She explained that "getting started" to a neurosurgeon meant planning the craniotomy flap, positioning the patient, shaving, prepping, draping and doing the opening.

[166] When she arrived, Miss Brown changed into her theatre scrubs but, again in accordance with her usual practice, prior to scrubbing and putting on gown and gloves, she went in to the theatre to see what was happening and to be satisfied that she was

happy with the preparations and that there were no minor corrective suggestions that she had to make. Mr Ahmad had just made the initial incision. As he was making good progress and Miss Brown did not feel that there was anything that she would do better or more quickly or safely, she allowed him to continue. She remained in theatre to supervise the procedure and to give advice and suggestions if, and when, required. She was confident in Mr Ahmad's ability and thought that he was doing well. There had been no need for her to intervene. Had she needed to intervene she would have had to go out, scrub and put on a gown and gloves which would have taken about five minutes. She did not consider that anything would happen so precipitously that she would not have had time to gown up if necessary. Thus there was continuous assessment of the procedure and Miss Brown was present in theatre at all times.

[167] Surgery commenced at 04:20 and lasted for an hour and a half. Mr Ahmad's notes on the surgical procedure were written up at 06:30. These notes described the following procedure and findings.

[168] On making the initial incision, the immediate findings were suggestive of raised intracranial pressure. There was "a very tight brain" and for that reason Mr Ahmad could not immediately open the dura (the tough membrane surrounding the brain) but first had to aspirate 25mls of blood from the clot cavity before opening the dura. That allowed the brain to relax and prevented further damage. The dura was then opened thereby visualizing the clot from the haematoma. In total, some 30mls of blood and clot were evacuated via aspiration of fresh blood and suction removal of the clot. There was no obvious major bleed during the procedure or any problem controlling the bleeding.

Haemostasis was achieved showing that there was no active bleeding into the clot cavity, none from the dura, from the bone edges or over the surface of the brain. The bone flap was replaced but left unsecured. That was to allow for further swelling. Staples were inserted and a drain was left in place.

[169] Miss Brown agreed with Mr Ahmad's summary of the craniotomy. She had helped with decisions towards the later stages of the procedure. Decisions had to be made as to whether adequate haemostasis had been achieved and whether to pursue into the deeper, less visible levels of the brain. In Kathryn's case, she considered that there was adequate haemostasis. The brain was soft and pulsatile and did not seem to be under pressure and so to pursue the clot into the deep reaches beyond what was easily seen would have been unwise. This was a judgment call, properly taken by the consultant. Miss Brown explained that in such circumstances, the goal was not to make the scan perfect; the goal was to take away enough volume to give more capacity to compensate. Going deeper into the brain might have started new bleeding which might have been both difficult to see and difficult to control. These were dangerous areas. She explained that one of the most difficult decisions in such operations is to "decide that you are done, when to stop, get your haemostasis and get out". Mr Ahmad followed her direction.

[170] During the procedure Kathryn was given two units of FFP and further platelets. This was primarily under the control of Dr Urquhart who was in contact with the on-call haematologist (Dr Bunemann) during the operation. A repeat blood count at the end of the operation showed that her platelet level was 72 from a sample taken at 06:01. This was considered to be a satisfactory position.

Responsibility to correct coagulopathy

[171] According to Dr Urquhart, it was the job of the anaesthetists to try to normalise things as much as possible. Usually it would be the anaesthetist who discussed things with the laboratory in advance for the lab staff to organise blood products required in conjunction with the haematologist.

[172] Platelets were required to correct Kathryn's coagulopathy and cryoprecipitate for the low fibrinogen results. At the time, Dr Urquhart did not know the underlying reason for the coagulopathy problem. While he accepted that it was important to find that out, he explained that, by and large, anaesthetists try to *correct* the problem as a priority, above determining the actual cause. He himself was not familiar with AML or any sub-types of the disease. Neurosurgeons make the decision to proceed to surgery – the anaesthetists try to optimise the patient as much as possible to facilitate surgery, sometimes asking the surgeons to delay a bit. Then it becomes a question of risk- benefit, weighing up pros and cons.

[173] He explained that it was routine practice where there was a problem with coagulation to obtain the advice of a haematologist. Usually, this would involve a discussion during which the haematologist would want to know the history. Normally he would tell the haematologist the history of the patient – if he did not, the haematologist would ask for it. Frequently further discussions take place during surgery. Dr Urquhart explained that the haematologists were usually quicker to pick up on things

like fibrinogen, suggesting cryoprecipitate and things like that. Abnormal results were generally phoned to theatre. Often the on-call haematologist would come into the lab to give advice.

[174] Dr Urquhart did not note any discussions with haematology. He would have required to contact the haematologist for FFP, platelets and maybe for PRC (packed red cells) also. The relevant taxi sheets from Gartnavel Hospital made reference to platelets coming 03:55. That related to a request for platelets which was entered into laboratory system at 03:00 and requested by Dr Bunemann at 03:35. There would have been further contact. It was a discussion over time because things change, he said– we fairly frequently discuss for further advice and help. Had Dr Urquhart received any specific advice from Dr Bunemann apart from authorisation of products he would almost certainly have made a note of it. He was clear that he would have been phoning the haematologist for advice as well as authorisation. For example, the haematologist would notice fibrinogen and they would quite often have access to other therapies that were not necessarily the first line things anaesthetists would think of.

[175] Significantly, Dr Urquhart did not appreciate that he was talking to someone who was not a haematology trainee. Had he known that, he would have suggested to Dr Bunemann that he spoke to his consultant to get advice.

Anaesthesia during surgery

[176] The following observations were noted in the course of the operation:

BP 125/75 pulse 110 at 0300

Normal saline first but suggestion from nursing notes at p127 that platelets put up about 0400.

PRC 0425

Platelets just after 0430 approx 0435 – 1 pool All that were made available.

Another unit packed red cell ten minutes later at about 0440

MSFP just before 0500

Mannitol just after 0500 - 0505-0510

FFP 0510

PC3 0525

Saline 0535

[177] The operation was uneventful from an anaesthetic viewpoint. Kathryn's condition remained stable throughout and she was also noted to be haemodynamically stable.

[178] A post-surgery blood sample was taken towards end of operation at 06:01. This showed a marginal improvement – Kathryn had received a lot of blood products. She was still technically coagulopathic with an INR of 1.5. These results would have been telephoned to ICU. Kathryn's blood pressure was initially quite high with a systolic of 150 which came down over the course of the procedure to 125-130.

Condition after surgery

[179] Kathryn was returned to the intensive care ward from theatre. She was back on the ward by 06:30. Post-operative instructions included that Kathryn be weaned off sedation. Her platelet count and haemoglobin were to be checked and instructions were left that a platelet transfusion should be given if the count were below 50,000 (50).

[180] Miss Brown and Mr Ahmad spoke to Kathryn's parents immediately after the operation. Although the craniotomy had proceeded uneventfully and the clot had successfully been removed, with successful decompression, the situation, according to Miss Brown, was grave. At that stage, a surgeon could never be sure whether the

patient's condition was poor because of the pressure effect alone and was recoverable, or whether that pressure had been present for a sufficient length of time to have caused permanent damage. Miss Brown tried to convey the gravity of the situation and the poor outlook to Kathryn's parents but, given the understandably highly emotional situation, she was concerned that not all that she had to say had been taken on board.

[181] Dr Beattie's recollection was that Miss Brown said that the surgery had proceeded well but that the outcome was still uncertain and that they would have to wait to see how things progressed. It was his impression that Miss Brown seemed to be giving hope for Kathryn's recovery.

[182] Kathryn was kept under constant observation and continued to be nursed on a one to one basis by Nurse Twaddle and, later, by Nurse Patricia Rooney. An entry in the nursing notes by Nurse Twaddle at 07:00 recorded that on her return from theatre, Kathryn remained intubated and ventilated. Her pupils remained unequal and non-reacting. The right pupil was measured at 7mm, the left at 4mm. Her heart rate was recorded as about 90 and her blood pressure was $^{120}/_{60}$. Her temperature was 37.4 degrees. There was no motor response. Miss Brown was aware of her condition and it was recorded in the nursing notes that she attended and spoke to Kathryn's parents. Nurse Twaddle had present but could not now remember what was said.

[183] In the course of her evidence Nurse Twaddle explained that the ventilator was set at "SIMV at 10 resps per minute". That stood for "spontaneous intermittent ventilation" and meant that if Kathryn had wished to breathe, the ventilator would cut out to allow

her to breathe on her own. At some point between 08:00 and 09:00 the setting on the ventilator had been changed to "ASB" which was "augmented spontaneous breathing" which meant that the patient was breathing but the ventilator was adding a little extra. Thus Kathryn was breathing herself but was also receiving some assistance from the ventilator.

[184] Dr Douglas Walker was the consultant anaesthetist who was on duty in ICU on 21 June. He was the senior intensivist in charge. He came on duty at about 08:00 and, in accordance with normal routine, he would have been made aware of Kathryn's condition. His colleague Dr Pamela Docherty was also on duty and it was she who would have received the verbal handover directly from Dr Urquhart. Dr Walker was made aware of Kathryn's history and the surgical procedure. He was aware at an early stage that an underlying haematological malignancy was part of the differential diagnosis and he knew that "haematology were involved". He had also been made aware – although he could not remember how and by whom – that a haematologist would be arriving to see Kathryn soon. Dr Anne Morrison, consultant haematologist, arrived on the ward shortly thereafter.

[185] Dr Walker was on the ward when Dr Morrison arrived. He could not recall the details of any discussions between them but he almost certainly did discuss Kathryn's condition. That would have been the usual practice. It would be highly unusual for a doctor to visit ICU to examine a patient and leave without discussing the patient with the senior intensivist in charge.

Examination by Consultant Haematologist

[186] Dr Morrison was the consultant on call for haematology overnight on 20-21 June. She had not been aware of Kathryn until she came on duty in the morning around 09:00. In accordance with her usual practice, Dr Morrison checked the computer system to see what results had been generated overnight. Kathryn's blood results were very abnormal and Dr Morrison immediately investigated further. She could not remember whether the blood film – which was a slide of the blood prepared so that it could be examined under a microscope – was brought to her attention specifically or whether she had to look for it herself having seen the blood results. The blood films from abnormal results that had been prepared first thing that morning would have been in a tray ready to be examined by a haematologist in terms of the standard operating procedures (see below paragraphs [281] to [284]). This would have included a blood film from the haematology laboratory in the Southern General prepared from the blood sample that had been taken on Kathryn's arrival timed at 03:42 hours. That was how Dr Morrison found out about Kathryn. Thus she had not been contacted directly by anyone prior to this.

[187] Dr Morrison was immediately concerned when she saw the blood results and the blood film. She would have seen the results that had been generated overnight including the ones from the sample times at 03:42 and the later sample at 06:01 which reported the platelet count at 72. The associated coagulopathy results would also have been available. Dr Morrison at that stage would not have had access to the earlier results from the Victoria Infirmary but would have seen these in Kathryn's notes. From the blood film, Dr Morrison immediately suspected that Kathryn had acute myeloid leukaemia. Rather

than delay and write a report on the blood film, she went straight to intensive care to see Kathryn.

[188] Dr Morrison spent at least an hour examining Kathryn and discussing her condition with staff in ICU. She advised her colleagues in ICU of her suspicion that Kathryn had acute leukaemia and that she needed further supportive care. Dr Morrison asked them what they thought Kathryn's prognosis was at that stage. She was told that she was likely to survive. She asked the question because it was her intention to do a procedure known as a bone marrow aspirate and trephine, a procedure under local anaesthetic where samples of bone marrow is taken by needle aspirate from the bottom of the back. Its purpose is to look at the chromosomes within blast (or immature) cells to identify the type of leukaemia.

[189] Dr Morrison gave instructions that Kathryn should receive more platelets and FFP. Dr Morrison contacted the blood bank at Gartnavel to arrange that and later checked with the blood bank within the hospital that the platelets had arrived.

[190] Dr Morrison made the following entry in the notes:

Blood film in keeping with acute leukaemia, probably AML.
Diagnosis explained to father (a GP).
He is willing to allow bone marrow examination. We will support Hb (haemoglobin*) and platelets and correct coagulation. Further discussion on management will be required and will need to take into account neurological recovery.*

*Bone marrow aspirate and trephine RPIC (*Right posterior iliac crest*). 2% lignocaine. LA* (local anaesthetic). *Aseptic technique.*
Samples for cytogenetic and cell markers sent.
Platelets ordered - aim to keep platelet count > 100x10^{a}l. Please check FBC mid afternoon. There will be a further pool of platelets available.

[191] In fact Dr Morrison ordered two pools of platelets. These were marked as having arrived at the haematology laboratory at the Southern General and available for collection at 11:22. A repeat platelet count from a blood sample taken at 10:55 and authorised at 11:37 showed a platelet count of 53 – this was a platelet count before the transfusion of the platelets ordered by Dr Morrison. A post-transfusion full blood count at 13:56 showed a platelet count of 98. Only one pool of platelets was transfused. The second pool was marked as having been returned. It was not transfused.

[192] Throughout the morning there was little improvement in Kathryn's condition and she did not respond as hoped. The only change was a slight improvement in her motor responses which improved from "none" to "flexing to pain". This was documented in the notes as a severe weakness in both arms and legs. Otherwise, her condition remained the same, with little or no change to the recordings made of regular observations. According to Dr Walker, the lack of pupil response throughout was a poor prognostic sign. During the morning Kathryn was given 2 units of blood (concentrated red cells) over four hours: the first unit at 9:30 and the second commenced at 11:45. At 10:55 she received saline and potassium (20 millimoles) after a biochemistry result was received (potassium was noted as 35 in blood results recorded on the chart timed at 11:00). Her platelet count was 53 and at 11:45 one pool of platelets was transfused over an hour. There was an error in the Infusion Chart: the chart included a reference to FFP as the substance infused when the infusion, in fact, related to the unit of platelets.

[193] Although he could not remember the detail of any conversations he had with Kathryn's parents, Dr Walker was confident that the line he would have taken would

have been to stress that Kathryn's neurological condition remained very poor and, similarly, that her outlook remained very poor.

Deterioration in condition

[194] Kathryn's condition began to deteriorate in the afternoon between 2 and 3 o'clock. At 14:30 her motor responses deteriorated from flexing to no response. Thereafter a rapid deterioration occurred. The observations noted on Kathryn's chart and in her notes do not entirely correspond. The retrospective summary in the nursing notes made at 18:40, after Kathryn's death, record that at 14:30 Kathryn's condition deteriorated rapidly. Her pupils remained unresponsive and the recorded measurements were Right 6 and Left 5**.** There was no motor response. She became cardiovascularly unstable. Her heart rate went up to 180, her blood pressure was $^{160}/_{80}$ and her temperature was raised at 39.2. The ICU observation chart contained no entries at 1430. At 15:00 her motor response had deteriorated to none. No other observations were noted. At 15:00 it was noted that she had a CT scan. However, there was an entry showing her pupils at Right 6 Left 5, both unreactive. At 1600 her pupils remained the same, her temperature was recorded as 39.2, her blood pressure at what appeared to be $^{148}/_{80}$ and her pulse at something in excess of 220 beats per minute.

[195] Such changes were an indication that coning was occurring. Miss Brown was informed of the deterioration in Kathryn's condition and it was decided to take Kathryn for another CT scan to see what was going on in her brain. From the report, the scan was carried out at 14:50, although the time of the scan was noted in the observation chart as 15:30 and recordings were noted of Kathryn's observations in ITU at 15:00.

[196] Kathryn's named nurse was Nurse Patricia Rooney. She gave evidence but could remember little about the events. She explained that her notes were written up at the end of the day when she was relying on her recollection. They were not made at the time the various events occurred. From her memory, she thought that Kathryn's condition began to deteriorate around 14:00.

[197] After the scan, Kathryn was returned to ICU. At about this time, Dr Morrison had returned to the ward to discuss Kathryn's condition and to advise that the bone marrow tests had confirmed that Kathryn did indeed have acute promyelocytic leukaemia, sub-type 3v (hypogranular). However, it was clear that there was a difficult and fraught clinical situation at that point and so she withdrew and did not intervene.

Second CT scan

[198] According to Miss Brown, the scan showed evidence of the right frontal craniotomy. It showed no significant change in the smaller areas of haemorrhage. However, there was a large area of haemorrhage in the right frontal lobe. She described it as a more complex picture than the original haemorrhage which had been more or less an acute clot displacing the brain. There was a change of pattern in the second scan. It was a patchy change rather than a single focus of haemorrhage. What was seen on the later scan was a patchy picture of both oedematous brain and necrotic brain with a haemorrhage of greater volume than before. According to Miss Brown what was seen was not consistent with a single bleeding point that had re-bled. It was a mixture of bruised brain, necrotic brain, swollen brain and blood cells. This was a pattern which was seen where pressure has been high and surgery has been performed to effect a

decompression. The area of haemorrhage included the surgical site but was not restricted to it. There was a more extensive area of damage with a corresponding increase in midline shift despite the bone flap which had been left loose to allow additional room for compensation. The other lesions had not substantially altered. The one exception was the one in the right capsule which had burst into the third ventricle slightly.

[199] Miss Brown discussed the scan findings with Dr Walker. She felt that there was a degree of tissue destruction that was not compatible with life. Both she and Dr Walker concluded that Kathryn had sustained a non-survivable brain injury.

[200] Later on, the scan was formally reported by Dr C Santosh in the following terms which reflected Miss Brown's assessment and with which she agreed:-

There is evidence of a right frontal craniotomy. Areas of haemorrhage and brain oedema noted in the right frontal lobe. Allowing for technical differences, the extent of the haemorrhagic area and the amount of blood has increased when compared with the Ct scan done on 21.6.04. The other areas of haemorrhage in the left inferior frontal and right occipital region are still present and have not significantly changed. There is mass effect with marked compression of the right lateral ventricle and shift of the midline structure to the left. The basal cisterns are all effaced secondary to the mass effect and brain swelling. The area of haemorrhage in the right basal ganglia has been compressed. There is compression of the mid brain. The cerebellar tonsils are noted extending below the foramen magnum secondary to tonsillar herniation. The craniotomy flap is displaced outward once again secondary to the brain swelling.
Focal areas of air noted intracranially secondary to operative intervention.

[201] Once the scan results were known, Miss Brown and Dr Walker spoke to Dr Beattie and his sister. They explained the gravity of the situation and that there was nothing more that surgery could offer. It was felt that the deterioration in Kathryn's condition was such that she needed to be examined in relation to brain stem function and we felt it was unlikely that she would survive. In an entry timed at 16:00 Dr Walker has recorded the conversation as follows:-

Father and sister seen with JB. There is no treatment which will reverse the devastating injury that she has already sustained – ,', for supportive treatment only.
-Not for reventilation] in event of respiratory arrest.
-Not for resuscitation]
No active treatment for DI (diabetes insipidus), *hyperthermia.*

(The latter are two conditions which not infrequently develop in such circumstances.)

[202] Dr Beattie remembered that he had been told that the scan had shown further bleeding at the site of surgery and that recovery now seemed unlikely. He was adamant that no discussion about brain stem death had taken place, as was his sister. She recalled that they were told that there was no hope of recovery.

[203] In his evidence, Dr Walker commented that there had been considerable difficulty in communicating the seriousness of Kathryn's condition to her family. Indeed, he commented that that had been the position throughout the day. He had tried to communicate the gravity of Kathryn's position and her poor prognosis, particularly in respect of the dilated and unresponsive pupils. His impression was that the family were finding it very difficult to assimilate the information they were being given and, particularly, that they had considerable difficulty in accepting that Kathryn's condition was non-survivable. He thought there was a problem in getting across to them that, despite the technical success of the surgery, Kathryn's overall condition remained very poor with a very strong possibility that she was not going to survive. His principal concern was of her neurological condition, rather than her platelet count. Dr Walker explained that, quite naturally, this was a major issue to the family and seemed to be the focus of their concern. However, what he was trying to get across was the rather broader picture of how things were going. Contrary to what might be expected in a parent with

medical knowledge where more insight to the problems might be expected, it seemed to Dr Walker that in Dr Beattie's case, as he put it, he was correspondingly less able to assimilate the information that was being imparted. In the circumstances, he believed that there could in fact have been a possibility that, in spite of all the efforts made to explain the gravity of the situation, that Kathryn's parents did not actually appreciate it. This was a concern echoed by Miss Brown.

[204] Dr Walker informed the court that Dr Beattie had specifically asked that his wife not be told about the diagnosis and outlook. This made communication difficult.

Respiratory arrest and death

[205] Despite the hopeless outlook, Kathryn was still breathing spontaneously. The ventilator remained set at ASB which meant that she was initiating each breath which was then augmented by the machine. However, at around 16:00, that changed and Kathryn suffered a respiratory arrest – she stopped breathing. That was a sign of possible brain stem death.

[206] Brain stem death requires to be confirmed by a number of strict tests carried out independently by two senior clinicians under a strict protocol. Certain important pre-conditions must be fulfilled before brain stem death teats can be carried out. Kathryn fulfilled these conditions. Thereafter, a series of separate tests are carried out to assess the activity in the brain. These were carried out by Dr Walker and Dr Docherty and confirmed the absence of brain stem activity and that brain stem death had occurred. Kathryn had died.

[207] There was particular focus during the Inquiry on the final stages of Kathryn's life and the circumstances in which the terminal clinical events occurred. This was a harrowing chapter of the evidence.

[208] Kathryn's parents and her aunt, Dr Rosaleen Beattie, described what happened in the following terms. Dr Beattie's recollection was that minutes after Dr Walker and Miss Brown had spoken to them, Dr Walker came up to Kathryn's bedside accompanied by another doctor (Dr Docherty) and "announced" that he was turning off Kathryn's ventilator. The family objected most strongly. It was Dr Beattie's position that up until then, no mention of brain stem death tests had been made. This was not Miss Brown's recollection or that of Dr Walker himself. Although neither could remember the detail of the conversation, both were certain that this had indeed been discussed when the news of the scan had been conveyed to both doctors Beattie.

[209] Dr Beattie told the court that it seemed that Dr Walker and Dr Docherty were going to "organise" Kathryn's death. Dr Walker did not listen to the family's objections and continued towards the ventilator whereupon Kathryn's grandmother physically barred his way. The family insisted that they wanted a second opinion before such drastic action was taken. Dr Walker explained that Dr Docherty was there to provide a second opinion but the family were not satisfied. According to Dr Beattie, Dr Walker refused to allow a second opinion which resulted in Dr Rosaleen Beattie leaving the ward in an attempt to obtain a second opinion. Kathryn's family effectively stood guard to prevent any access to her in case Dr Walker and Dr Docherty tried to turn the ventilator off. Dr

Beattie then recalled that a new consultant neurosurgeon, Mr Robin Johnston, arrived in the ward and explained that he would examine Kathryn.

[210] Dr Walker did not accept that he had told the family that they were not entitled to a second opinion. He recalled that this had been asked for and that he had left the ward to try to obtain someone who would be able to give that. He could not recall specifically the point at which the request for a second opinion had been made: whether it was in relation to the need to do the brain stem tests or whether it was in relation to the information they had given to the family after the scan. In any event, his recollection was that he left the ward and tried to find someone in either theatre or x-ray but had been unsuccessful. When he returned to the ward Mr Robin Johnston arrived to give that opinion. His position was confirmed broadly by charge nurse William Main whose recollection was that he was told that Dr Walker had left the ward and had gone to x-ray to find someone to give a second opinion.

[211] Dr Walker was by now unsure of the exact sequence of events. However, he recalled the event when Dr Beattie burst through the curtains around the bed and started breathing down Kathryn's endotracheal tube. (Note – the tube must have been disconnected at that point as he could not have breathed down it otherwise.) He clearly remembered the event – a specifically unusual, indeed unique, event hardly able to be forgotten. He recalled that the family seemed prepared to carry on ventilating themselves. However, he was not certain as to the exact sequence of events. He remembered that the family had asked for a second opinion at some point. In any event,

he made the decision that ventilation should be recommenced to allow things to calm down.

[212] Dr Rosaleen Beattie recalled that she went to get assistance. She made half-a-dozen telephone calls to consultant colleagues of hers in an effort to obtain a second opinion. In the end she resorted to telephoning the then President of the Royal College of Physicians to seek his advice. His advice was to telephone the doctors' common room where she would find someone who was able to provide an opinion. Dr Rosaleen Beattie did so and spoke to Mr Johnston who agreed to give an opinion. She did not know Mr Johnston and did not meet him. She arrived back on the ward with him by chance at the same time.

[213] A yet different account of events was recalled by Mr Johnston. He remembered being in his room on the sixth floor (some distance from ICU) when Dr Walker came in with a female colleague (possibly Dr Docherty). He timed this visit at about 5pm. He remembered it specifically because it was unusual for Dr Walker to visit him in his office. Dr Walker explained the background and Mr Johnston agreed to come down to ICU and carry out repeat brain stem death tests on Kathryn. That was what he thought he was being asked to do. He was clear that Dr Walker had come to ask him in person. (This was not clarified with Dr Walker who had completed his evidence before Mr Johnston was called.) Moreover, Mr Johnston had no memory of having received a telephone call from anyone about this and, specifically, did not recall being contacted by Dr Rosaleen Beattie. Mr Johnston had a clear memory of that: in twenty-five to thirty years, it was the only time he had ever been asked to provide a second opinion on brain stem death. It was a unique event.

[214] Dr Docherty had no recollection of Kathryn or the events that occurred at the stage of the brain stem death criteria being tested. She had not been asked to consider Kathryn's case until 2012, some eight years after.

[215] Charge Nurse William Main appeared to have the clearest recollection of events and his account had the advantage of being from someone not immediately involved in the high emotional drama of the events. Someone came to alert him about what was happening on the ward and that a situation had developed which required his help as charge nurse. He was told that while brain stem death tests were being performed or just after that, Dr Beattie was providing artificial breaths to Kathryn via the endotracheal tube. When he arrived the situation had calmed down somewhat. He was aware that a second opinion had been asked for by the family and that Dr Walker had left the ward and had gone to the theatre suites and x-ray to see if he could find someone. Kathryn was attached to the ventilator. Mr Robin Johnston came into the ward and, in Nurse Main's presence and with his assistance, carried out a set of brain stem death tests which confirmed that Kathryn had no brain stem activity. He recalled that after the relevant tests had been completed, Mr Johnston had stated his intention to leave Kathryn disconnected but to let the family come in and be with her as her heart stopped. As Mr Johnston attempted to explain, as soon as he heard that Kathryn was disconnected from the ventilator, charge Nurse Main recalled that Dr Beattie pushed past calling Mr Johnston and himself "murderers". He was not listening to the explanation being given. According to Charge Nurse Main, he and Mr Johnston did not hold back but stood between him and Kathryn. Dr Beattie was pushing to get past trying to get to Kathryn's

head. Given the circumstances, Mr Johnston instructed that Kathryn be put back on to the ventilator to allow the family to come to terms with what had happened.

[216] The family's account was somewhat different. They had waited outside the ward while Mr Johnston was with Kathryn. About fifteen minutes later, Mr Johnston approached the family and said that he had carried out all the tests for brain stem death and when he had come to do the apnoea test, in order to show that Kathryn could not breathe spontaneously he had left the ventilator switched off. At that point, Dr Beattie jumped up and ran into the ward to Kathryn's bedside to see what was happening. He saw Dr Walker and Dr Docherty sitting at the foot of the bed. Kathryn was disconnected from the ventilator. Dr Beattie said that Kathryn was still nice and pink and he checked her pulse which was nice and strong. He decided to continue artificial breathing via the tube, took hold of the tube and began to breathe down it himself. He said that he was prepared to do that for ever until they put the ventilator back on.

[217] Dr Beattie recalled a huge commotion going on in the ward between the family and staff and eventually a decision was taken to reconnect the ventilator. The family were allowed to remain with Kathryn for some hours. Early in the morning, when it was light, Dr Beattie switched off the ventilator. He said that no father should have to do such a thing; that it was "immoral and disgusting" that he had been left to do it. This was in direct contrast to the evidence of Nurse Twaddle who said that Dr Beattie had become distressed at the thought of the ventilator being switched off by anyone other than by him. He insisted that he be the one to do that. It was highly unusual and, strictly speaking, against hospital protocol but, exceptionally in the circumstances, Dr Beattie

had been allowed to do it. She recalled that the ventilator had been switched off between 4 and 5 am.

[218] So many years later, it is impossible for me to reach a conclusion as to the exact sequence of events and for the purposes of my determination it is not necessary that I do so. The different accounts were quite extraordinary. However, out of all the conflicting accounts, it seemed to me that the evidence of Charge Nurse Main was the most reliable. That account seems to fit with Dr Walker's recollection that the point at which Dr Beattie started to ventilate Kathryn himself was not after Mr Johnston had performed the tests but at the time of the first set of tests. I am reinforced in that view because Mr Johnston had no recollection of seeing Dr Beattie actually breathing down the tube. He was sure that such an event would have been etched in his mind forever if he had witnessed it. I agree. I did not accept Dr Beattie's evidence that he had been left to switch off his daughter's life support and preferred Nurse Twaddle account. Given the high emotional atmosphere at the time of these events, it is not surprising that memories have become confused and doubtless corrupted with the passage of time.

[219] I was satisfied that the events surrounding the performance of the brain stem death tests were unique and did not raise issues of general public interest. However, this chapter of evidence is relevant to my determination in terms of section 6(1)(a) which relates to the time of death. Dr Docherty issued the death certificate in which she wrote the time of death as 18:15. That was clearly wrong. Dr Docherty accepted that there was an inaccuracy on the death certificate but was now unable to provide the court with any explanation as to how that came about.

[220] Mr Johnston's record of the time of death was 17:40. That reflected the time when he completed his set of tests. The time of death should be recorded at the stage at which the first set of brain stem death tests were completed. From the evidence available, it would appear that brain stem death was established at or around 17:00 hours.

[221] The cause of death was certified as 1(a) Intracerebral Haematoma 1(b) Acute Myeloid Leukaemia. The death certificate was signed by Dr Docherty. Cytogenetic test results came back after Kathryn had died confirming that she had Acute Promyelocytic Leukaemia M_3 variant. Accordingly, the recorded cause of death should be amended to reflect that.

Respiratory arrest

[222] Not only were there inaccuracies on the death certificate, the evidence about exactly how and when the respiratory arrest occurred was most unsatisfactory. Even allowing for the urgency of the situation, this was a crucial development in Kathryn's clinical condition that appears not to have been recorded anywhere in her case notes. There was no entry of the event in either the medical notes or the nursing notes with the exception of a retrospective narrative or summary in the ICU nursing notes to document the fact that Kathryn had stopped breathing. The actual clinical charts were silent as to how and when this occurred. Furthermore, there was no note on the ventilation chart to document either the occurrence or timing of the arrest. Neither was there any record to indicate that the mode of ventilation had switched as a result. The chart showed that the mode of ventilation remained as ASB.

[223] The evidence about this was unclear. As to the medical notes, Dr Walker explained that this might have been overtaken by subsequent events in the context of a highly fraught and emotional situation. The fact that it had not been documented was not particularly remarkable. He also described how emotions were running high at that time and he thought that it may have been in response to that which led to ventilation being re-commenced at that stage. That may well have been the case. The circumstances were certainly highly unusual.

[224] These issues came into focus when Dr Beattie's solicitor asked Dr Walker to consider the possibility that no such arrest had occurred, a fact that Dr Walker could not accept given that the brain stem death tests confirmed that Kathryn had, indeed, stopped breathing. Clearly, he said, there had been a respiratory event.

[225] While it was unsatisfactory that there was no record of the respiratory arrest having happened, it clearly did. There is a reference to the event – albeit brief – in the records and the subsequent brain stem death test confirmed the position.

Reporting of Kathryn's death to the Procurator Fiscal

[226] In Scotland, the procurator fiscal has the public responsibility to investigate the circumstances of all sudden, suspicious, unexpected or unexplained deaths. Anyone can bring the circumstances of a death to the attention of the procurator fiscal. Where the death occurs in a hospital setting in circumstances where it requires to be reported, it is normally the duty of the doctor in charge of the patient or the doctor who has the responsibility of certifying the death to contact the procurator fiscal. The circumstances of Kathryn's death were not reported to the procurator fiscal at the time. It was not until

some three years after when, in 2007, Kathryn's parents contacted the fiscal with their concerns about the circumstances of her death.

[227] The fact that the death was not reported until very much later meant, of course, that the procurator fiscal was unable to instruct a post-mortem examination. That undoubtedly would have been informative. Furthermore, no enquiries were made at a time when recollections would have been fresh and comprehensive, reliable information would have been available to assist the procurator fiscal's investigations.

[228] This matter was not explored in any great depth during the proceedings. No witness was called from the Crown Office and Procurator Fiscal Service to give evidence on the matter. Reference was made to the terms of the letter of instruction addressed to Professor Grimwade in which included the following comment from the instructing fiscal: *As the death of a previously well child who did within 24 hours of admission, I would have expected Kathryn's death to have been reported to this office.*

[229] However, it was Miss Adair's position that the death should have been reported, not because Kathryn had died within 24 hours of admission, but because the circumstances were such that a complaint might have been anticipated. That gave rise to a brief consideration of the guidance issued to members of the medical profession. There was a conflict of opinion between the senior staff at the Southern General as to whether Kathryn's was a reportable death.

[230] The guidelines in force in 2004 were contained in a booklet entitled *Death and the Procurator Fiscal*. The guidelines identify the categories of death which require to be reported. Those of relevance here include the following:

- *Deaths which occur unexpectedly having regard to the condition of the deceased prior to his receiving medical care.*
- *Deaths which are clinically unexplained*
- *Deaths which are apparently associated with a lack of medical care… Any death as a result of medical mishap, and any death where a complaint is received which suggests that medical treatment or absence of medical treatment may have contributed to the death…*

[231] Dr Walker was the senior intensivist in charge of Kathryn at the time of her death. Miss Brown was the named consultant in charge of Kathryn's care. Both had joint responsibility in deciding whether Kathryn's death should have been reported. Dr Docherty issued the death certificate and so she, too, needed to be satisfied that the death did not require to be reported.

[232] Dr Walker did not consider that the circumstances of Kathryn's death were reportable. During the course of Kathryn's time in the Southern General, her death was certainly not unexpected. Nor was it clinically unexplained. He was not aware at the time that a complaint about her medical care was going to be made. He did not recall any specific discussion about the matter at the time, with Miss Brown or anyone else. He was not aware of any issues in connection with Kathryn's treatment at the Victoria Infirmary. Had there been any, he would have expected these to have been brought to his attention. As a senior intensivist, he was used to considering whether deaths required to be reported. It was not his understanding that a death which had occurred within 24 hours of admission or surgery required to be reported.

[233] The matter was raised with Miss Brown. She accepted that, as the named consultant she had overall responsibility to decide whether Kathryn's death should have been reported to the procurator fiscal. However, a more direct responsibility lay with the individual who completed the death certificate. She was asked as this was the death of a child who had died after a craniotomy, very major surgery, within 18 hours of admission, was it a death which ought to have been reported to the procurator fiscal. She replied that that would have been her normal practice. Thus, from the way in which the question was put (by the same fiscal as drafted the letter of instruction to the expert witnesses), the inference was that the matter fell within the category of a reportable death because it occurred within 24 hours of admission and/or surgery.

[234] Dr Docherty, as the doctor who issued the death certificate, was not questioned about this. Indeed, this was a matter which was not pursued in any great depth with the relevant witnesses but the evidence was enough to raise a concern that there was a misunderstanding amongst senior members of the medical profession (and perhaps even within the fiscal's office) as to when a death should be reported. It was Miss Adair's submission, however, that the current guidelines were sufficiently clear on the matter. Mr Khurana agreed. Mr Lamont and Dr Beattie included the subject of reporting deaths in terms of their submissions under section 6(1)(e).

[235] Given the profound effect that the non-reporting of this death has had on the timing and quality of subsequent investigations, and having regard to the continuing confusion as to whether a death which has occurred within a 24 hour period requires to

be reported, it may be that the guidance to doctors would benefit from some review. In the absence of clear evidence on the matter, it is not appropriate that I include this in my formal determination. Therefore, I simply note the position in order to bring this matter to the attention of the Lord Advocate for his consideration as to what, if any, action he may consider helpful in order to clarify the position for future reference.

PART 4 THE EXPERT EVIDENCE

[236] This court heard a substantial amount of expert evidence. From now on, reference will be made to expert witnesses and their opinions. Therefore, before proceeding further, it is convenient to outline the role of the expert witness in court, the duty of the court insofar as the expert evidence is concerned and to examine the qualification and skills of the witnesses themselves.

[237] A total of eight experts gave evidence from three separate disciplines; neurosurgery, neuroradiology and haematology. Expert reports had been prepared and exchanged although it is important to note here that not all of these were fully incorporated into the evidence. All those called as witnesses of skill were highly qualified. No objection was taken to their ability to give opinion evidence and I was satisfied that each was qualified to assist the court in that capacity.

[238] As to the role of the expert witness, the classic exposition is to be found in *Davie v Magistrates of Edinburgh* 1954 SC 34 *per* Lord President (Cooper) at p40:

> Their duty is to furnish the judge or jury with the necessary scientific criteria for testing the accuracy of their conclusions, so as to enable the judge or jury to form their own independent judgment by the application of these criteria to the facts proved in evidence. The scientific opinion evidence, if intelligible, convincing and tested, becomes a factor (and often an important factor) for consideration along with the whole other evidence in the case, but the decision is for the judge or jury. In particular the bare *ipse dixit* of a scientist, however eminent, upon the issue in controversy, will normally carry little weight, for it cannot be tested by cross-examination nor independently appraised, and the parties have invoked the decision of a judicial tribunal and not an oracular pronouncement by an expert.

The expert haematologists

[239] Two experts were called by the procurator fiscal in the public interest. They were Professor Beverley Hunt and Professor David Grimwade. Professor John Liu-Yin ("Professor Yin") was called on behalf of Ms Crawford. Dr John Hanley was called on behalf of Dr Bunemann. The four haematologists who gave evidence were impressive witnesses. However, the expert of outstanding note was Professor Beverley Hunt.

Professor Beverley J Hunt MbChB, MRCP(UK), MRCPath, MD, FRCP, FRCPath

[240] Professor Hunt was a hugely impressive witness. She had about her an air of quiet gravitas. She has impeccable qualifications and enjoys an international reputation as an expert in acquired bleeding disorders. I had no doubt as to her eminence in her field. She is currently Professor of Thrombosis and Haemostasis at King's College, London. She also holds a consultant post in the departments of Haematology, Pathology and Rheumatology at Guy's and St Thomas' Foundation Trust, London. For the last twenty years she has been involved in delivering a haematology laboratory service and is currently Clinical Lead in Laboratory Haematolgy, a post which she has held for over ten years. She heads a large research team leading research into coagulopathy in APL. She runs a number of clinical trials and, together with Professor Grimwade, is currently doing research on the molecular side of the disease. She has a big clinical practice and provides advice on a daily basis to clinicians all over the UK and beyond. Indeed, during breaks in her evidence, Professor Hunt was engaged in ward rounds and dealt with numerous telephone and e-mail requests for advice. As well as an illustrious academic and clinical career, Professor Hunt has an impressive array of publications to her name, appearing in prestigious professional journals. She had co-written national and

international guidelines on the management of bleeding disorders. She was modest about her qualification and expertise. Her expert colleagues in court clearly held her in high esteem.

[241] She is a recognised world expert on coagulopathy and is carrying out leading edge research in to a number of matters, including the coagulopathy associated with APL. The Inquiry was privileged to hear about the current thinking behind her research which Professor Hunt generously shared, notwithstanding her research is on-going and, as yet, unpublished. As a leading expert on coagulopathy, her evidence was of pivotal importance in this Inquiry. Professor Hunt was careful to stress that she was not an expert in APL, nor was she an expert in terms of the treatment of the disease. She deferred to her fellow experts in that connection. However, on matters of coagulopathy she was firm that she would not so defer.

[242] Professor Hunt was not only impressive on paper, she was impressive from the witness box. (I use the term "witness box" loosely as Professor Hunt gave evidence from her office in London by live television link.) She gave evidence which was noted for its clarity, thoughtfulness and cogency. It was obvious that she had a commanding knowledge of her subject. I was impressed by the way she listened and responded clearly to the questions she was asked. Her explanation of Kathryn's coagulopathy in terms of active hyperfibrinolysis was entirely logical and persuasive.

Professor David Grimwade

[243] Professor Grimwade was likewise an expert witness with a most impressive clinical and academic background and an international reputation. As the Professor of Molecular

Haematology at Guy's Hospital in London, his special interest lies in the genetics of leukaemia. He has a long-standing interest in APL. He heads research into the biological, molecular and genetic aspect of APL. He leads the UK Reference Laboratory of molecular diagnosis of APL which receives samples from patients all over the UK and from other countries. Professor Grimwade's work is laboratory based – it is about ten years since he was ward-based seeing patients but he remains involved in clinical work and clinical trials. He provides guidance on treatment protocols on a daily basis and as such is an expert on the treatment of APL. He has an impressive list of publications (over sixty articles on APL) and, like his colleague Professor Hunt, was involved in drawing up the 2006 national guidance on APL and had a primary role in writing the 2009 international guidelines.

[244] Professor Grimwade's knowledge and understanding of the disease process was plain to see. The court was left in no doubt as to the complexities of the disease. I was impressed by his obvious expert knowledge, although, like Professor Hunt, he was modest and matter of fact about his academic achievements. His evidence describing the challenges associated with treatment, particularly in high risk cases such as Kathryn's was eloquent and convincing. However, when it came to giving his opinion on the particular circumstances of Kathryn's case, he did not always listen to the questions and tended to repeat the general answers already given in order to emphasise these. That aspect apart, he was a most impressive witness and his enthusiasm for his subject was plain for all to see.

Professor John Yin

[245] Professor Yin was likewise a haematologist of immense experience with a prestigious career. Now retired, he continuous with his research interest into leukaemia. His principal interest is in AML but that includes APL. He was consultant haematologist at Manchester Royal Infirmary and was awarded an honorary chair at the University of Manchester in recognition of his academic work. He has a wide experience in clinical work and teaching and an impressive publication history. He heads research into clinical trials into AML, similar to those run by Professor Grimwade in respect of APL.

[246] Professor Yin's expertise was beyond doubt, as was his "hands on" clinical experience. He gave clear and explicit evidence. However, as a witness I found him to be somewhat dogmatic, appearing to be less prepared to consider opinions which did not agree with his own. Perhaps that was simply a reflection of his own belief in the validity of his views but at times he seemed over-confident and I detected an element of arrogance in his manner when challenged. On more than one occasion he proceeded on assumptions about the evidence which were incorrect. His main problem was that he persistently interrupted questions that were being put to him and gave his answer mid-question. This was an unfortunate habit. Although it was obvious that he was an experienced expert witness and aware of his duty to be independent, I was concerned by some comments he made which were inappropriate. For example, when asked whether Kathryn had a chance of survival he replied *Yes, I would agree that there was a small chance. That's why we're defending this case.* On another occasion in answer to a question about intracranial pressure he replied *So, to some extent it kind of supports our case.* These jarring

responses left me with a sense of disquiet that Professor Yin had approached the proceedings as if they were adversarial in nature rather than inquisitorial.

Dr John P Hanley MBChB, MD, FRCP,FRCPath

[247] Dr Hanley is currently consultant haematologist at the Newcastle Hospital Trust. He has a special interest in Haemostasis and Thrombosis. He impressed as a very active clinical haematologist with high academic qualifications. He had experience at consultant level in Christchurch Hospital, New Zealand and experience in paediatric haematology. He was a consultant in Ninewells Hospital in Dundee prior to his current post. For the past five years he has been Clinical Director of the Specialist Haematology Directorate in Newcastle which is a tertiary referral centre. He is therefore head of department of all aspects of clinical haematology. He is Co-Director of the Newcastle Comprehensive Care Centre. He also holds a postgraduate qualification in medical training and is regularly involved in the training of junior doctors in the management of bleeding disorders. He is an examiner in haematology (Coagulation) for the Royal College of Pathologists. He is extensively involved in research and has published in various professional journals.

[248] Dr Hanley was an expert witness who created a very positive impression. His evidence was clear, measured and pragmatic as well as incorporating his obvious expertise. I am sure that he would forgive me for saying that as an expert witness he was not quite of the eminence of Professors Hunt and Grimwade, but his evidence provided a valuable source of information, not only of the current thinking in relation to APL but how that is currently applied in practice. His opinions were carefully given and I had the

impression that this was a highly experienced haematologist with a realistic approach firmly rooted in common sense. I was able to rely, in particular, on his evidence on matters of training. Moreover, Dr Hanley had been instructed fairly late on in the proceedings. The advantage of this was that his instructions clearly reflected the evidence that had been given in court. I was confident that his opinions were based on an accurate foundation and, as such, were reliable.

The experts in Neurosurgery and Neuroradiology

[249] Four experts gave evidence: two neurosurgeons, Mr Connor Mallucci and Miss Myles; and two neuroradiologists, Dr Wellesley Forbes and Dr Susan Keeley. Mr Mallucci was instructed by the procurator fiscal in the public interest. Miss Myles was called on behalf of the Greater Glasgow Health Board, as was Dr Keeley. Dr Forbes was called on behalf of Ms Crawford.

[250] Both neurosurgeons were highly impressive witnesses. Their evidence about intracranial pressure and its effect was expressed in clear, readily understandable terms. Their opinions in this case were fundamentally important and were given in careful, measured but confident terms. I was able to afford their opinions considerable weight.

Mr Connor Mallucci

[251] Mr Mallucci is a consultant neurosurgeon at Alder Hey Children's Hospital, Liverpool and at the Walton Centre NHS Foundation Trust, also in Liverpool. His specific interests are in the areas of paediatric brain tumours, hydrocephalus and neuro-vascular anomalies. Among his many posts and committees, he is currently Deputy

Editor of the British Journal of Neurosurgery and is Chairman of the British Paediatric neurosurgical Group. He has published extensively on his subject.

[252] Mr Mallucci's evidence was very persuasive. His opinions were cogent and reached on sound logic. Although he was confident in his opinions, his was in no way dogmatic. This was demonstrated by the fact that he readily admitted that in his original report he had omitted to consider some factors which he noted in Miss Myles' report. He promptly produced a supplementary report acknowledging that and incorporating those features into his own revised conclusions. While these did not substantially alter his original conclusions, it demonstrated that he was open-minded and gracious in conceding points where that was merited. His evidence impressed as balanced and unbiased. He was a witness of the utmost integrity, as was his fellow expert, Ms Myles.

Miss Lynn Myles,

[253] Miss Myles is a consultant neurosurgeon in the Department of Clinical Neurosciences at the Western General Hospital, Edinburgh. She has an impressive academic career. She trained as a paediatric neurosurgery in Toronto and was a consultant paediatric neurosurgeon in the Royal Hospital for Sick Children in Edinburgh from 2001 to 2011. She has a string of publications to her name.

[254] Ms Myles stood out as someone who robustly tested her own opinions against other facts and circumstances in the case. Matters were carefully considered with close attention to detail. Consequently, her opinions were well-rounded and cohesive. Like Mr Mallucci, she was a patient witness who readily took on board other opinions and

gave them proper consideration. She was not dogmatic in her views and made it clear whenever certain caveats needed to be applied to her conclusions. She struck me as a very bright and intuitive practitioner. I paid close regard to what she had to say.

Dr Wellesley Forbes MA MB DMRD FRCR

[255] Dr Forbes is Consultant Neuroradiologist at Salford Royal Hospitals Foundation Trust and Manchester University Children's Hospital NHS Trust. He has a particular interest in paediatric neuroradiology and has a medico-legal practice, regularly giving expert evidence. He, too, was highly experienced in his field.

Dr Susan M Keeley

[256] Dr Keeley is currently Consultant Neuroradiologist in the Division of Clinical Neurosciences at the Western General Hospital in Edinburgh. She was formerly a consultant in Duke University Medical Centre in the USA. She lectures in Edinburgh, Dublin and at Harvard University and has extensive experience in interpreting and reporting CT imaging.

Like her colleague, Ms Myles, Dr Keeley presented as bright and perceptive. Her evidence was clear and confident. Likewise her opinions were discerning and supported by the application of logic and experience. She, too, was an impressive witness and I was satisfied that her conclusions were soundly-based and therefore trustworthy.

PART 5: DESCRIPTION OF DISEASE AND PHYSIOLOGICAL PROCESSES

[257] In order to appreciate what happened to Kathryn and to consider the treatment she received, it is necessary to understand some of the underlying physiology and biological processes together with the associated medical terminology which were relevant to her condition. These include the basic elements of the coagulation process and the essential characteristics of acute myeloid leukaemia and the APL sub-type with particular reference to its unique and complex coagulopathy. It is also necessary to understand the mechanism whereby intracranial pressure rises and how the body attempts to cope with that. These issues are complex. The Inquiry had the benefit of hearing evidence from several witnesses who appeared in an expert capacity as well as from consultant haematologists and senior neurosurgical staff from the Victoria and Southern General hospitals. It is convenient to divide this chapter into two parts: the first dealing with haematology matters and the second with matters coming under the neurological disciplines.

MATTERS HAEMATOLOGICAL

Coagulation

[258] Coagulation is the process by which the body reacts to stop bleeding (leakage of blood cells) and prevent haemorrhage. It is the mechanism whereby a clot is formed in order to plug the hole in the blood vessel and stop further bleeding. There are two critical elements in the control of bleeding and clot formation: platelets and fibrin. Fibrin is formed from fibrinogen which is a soluble molecule. When activated, as when bleeding occurs, it becomes insoluble and links with other fibrin molecules to form a

mesh. Platelets are sticky substances which stick to the mesh and complete the clot. Therefore a clot is a mixture of fibrin and platelets, together with some trapped blood cells. The clot, in due course, will be dissolved by the action of a powerful enzyme called plasmin. It acts to degrade and break down the fibrin.

[259] In a healthy situation, the components in the blood that are important to coagulation are very closely regulated. There are two main pathways or arms whereby bleeding is controlled via the coagulation cascade. The *ex*trinsic pathway is measured by the prothrombin ratio or PT test. The *in*trinsic pathway is measured by the APPT ratio – the activated partial thromboplastin time. Basically, these are measures of the time taken for a clot to form. The normal reference range for the PT ratio 0.9 to 1.1 seconds and is 0.7 to 1.2 for APPT. However, it is important to appreciate that although these may be increased and will be an indication that there is something wrong with the coagulation, these tests alone do not inform the practitioner as to what is actually going on. While they are providing information about how a particular pathway is working, they do not give an overview of the coagulation process. Complex coagulopathy problems yet to be fully understood remain a key feature of APL.

Acute Promyelocytic Leukaemia (APL)

[260] It is important to remember that APL is a rare disease – described by the expert witnesses as an *extremely* rare disease. It is particularly rare in children. About 75 new cases are diagnosed in the UK per year but probably only about seven or eight cases in children. APL is a complex disease which, even in 2014, is not fully understood and is

particularly challenging to treat. APL is a sub-type of acute myeloid leukaemia (AML) which is a cancer of the white (myeloid) blood cells. There are numerous classifications of AML of which APL presents as the most acute of all the leukaemias. It is a particularly complex and challenging condition to treat and is associated with high risk of haemorrhage, making it one of the most urgent haematological emergencies. Paradoxically, it is one of the most successful of the leukaemias to treat having a high survival and cure rate in those patients in the less high risk categories or where those with high risk survive to reach and respond to treatment.

[261] Red and white blood cells are produced in the bone marrow, as are platelets. In APL, there is a defect in the early cells which fight infection. The cell acquires a chromosomal re-arrangement which is characteristic of this type of leukaemia. This involves a particular translocation of chromosomes 15 and 17 whereby chromosome 15 becomes fused to chromosome 17. This can be a chance phenomenon or can be linked as a secondary condition following chemotherapy or radiotherapy, and is also associated with certain drugs. However, it may occur spontaneously although it is not known why this happens. The translocation of the chromosomes which results in a swapping of material between chromosomes is considered to be the first event in developing leukaemia. However, simply having the 15/17 abnormality is not enough to develop the full-blown disease. It appears that further, additional abnormalities or mutations in other genes are required before the disease will develop. Recent studies sequencing DNA from a whole series of different leukaemias have always found at least one other mutation in another gene.

[262] According to Professor Grimwade, it takes about 18 months to 3 years for APL to develop after the first "hit" in a cell in the bone marrow. That cell has to accumulate further DNA damage and undergo additional changes before a full-blown leukaemia develops. Where the condition develops spontaneously, it is impossible to know exactly when the disease started, but it is considered legitimate to use these secondary leukaemias as a model and assume that the standard timescale seen there is how long it takes for APL to develop generally. This is reinforced by the fact that the disease, unlike other leukaemias, is not seen in children under 1 year.

[263] In terms of clinical onset, APL can present fairly quickly. Leukaemia can be brewing in the body but the number of abnormal cells may not be such that there are clinical symptoms. It is only when these reach a critical level that clinical symptoms start to manifest themselves. Most often the symptoms come on over a period of a few weeks but the onset can be very rapid. A patient can be perfectly well one week and very sick a couple of weeks later. There are many different presentations of the disease and presentation and symptoms will vary from person to person. Typically, some present complaining of tiredness and anaemia; others present with infection; and other patients may present with bleeding. Like Kathryn, some patients present with more than one of these symptoms.

[264] This abnormal chromosomal re-arrangement (which happens when the genes get broken in half and fuse together) forms an abnormal protein called PML/RARA. The effect is to give the cell a survival advantage so it can start to proliferate. Once it starts to accumulate additional mutations, then a full-blown leukaemia can develop. This causes

the cells to arrest in their development at a certain stage so that they do not fully mature – these are the blasts. This survival advantage over normal allows the leukaemic cells to grow at a steady rate until they push or crowd out the normal cells. Patients will become anaemic because not enough red cells are being produced. They will also have a low normal white cell count and will be more susceptible to infection because their ability to fight infection has been compromised. The cells in the bone marrow which produce the platelets (megakaryocytes) will likewise be crowded out so there is a reduction in the production of platelets. Moreover, the platelets will have a reduced survival time. This combination leads to the patient having bleeding tendencies. The situation is compounded by an increased consumption of platelets in an effort to control bleeding. Therefore, in APL, there are problems relating to production, survival and consumption as far as the platelets are concerned. The patient will have a reduction in circulating platelets and will therefore have bleeding tendencies. In APL, a lack of platelets together with low fibrinogen and other coagulation factors lead to the potential for catastrophic bleeding because there is no possibility of a clot being formed to prevent a haemorrhage.

[265] Haemorrhage is a major complication in patients with APL. It is still thought that up to 20% of patients who have APL have a haemorrhage so significant that they will die of it before they reach treatment. This is a sad fact even today. Bleeding in APL is often found in the brain or the lungs. Both of these sites are potentially lethal. The specific dangers of bleeding within the brain are discussed below at paragraph [299] *et seq*. A major cause of early death in APL, therefore, is intracerebral bleeding.

Coagulopathy in APL

[266] Platelets and fibrin are critical elements in the control of bleeding. Low platelets will result in a bleeding defect as will low fibrinogen. The two together is a very bad combination and that is what occurs in APL. However, the coagulopathy associated with APL is extremely complex and one of its main features is the possibility of catastrophic bleeding. In addition to the low platelets and the low fibrinogen which characterises APL, there is also a further coagulation defect which involves the increased breakdown of clots. It is early days to know what the factors are that contribute to this additional risk of bleeding. Professor Hunt is leading research into this area and there is data that appears to suggest that it is the molecules on the cell surface that are the cause. Professor Hunt's suspicion is that it is all to do with the fact that clot breakdown has been switched on in a very great way with these patients. There are still question marks as to what exactly is happening in the coagulation pathways, but it is recognised that it is hyperfibrinolysis.

[267] It is thought that what is happening in APL is that the molecules on the surface of the leukaemic cells are acting in such a way as to switch on the clot breakdown mechanism. The cells in APL have the ability to dissolve clot due to the over-expression of molecules on the cell surface, which stimulates clot and fibrinogen breakdown, the process known as hyperfibrinolysis. One of the things that they will do if there are no clots to breakdown is that they will also break down fibrinogen. Thus lower than normal levels of fibrinogen might be expected.

[268] Kathryn had the type 3 hypogranular promyelocytic leukaemia. There was evidence that about a quarter of APL cases involve the hypogranular variant. This sub-type is associated with high numbers of white cells. Cases that have a high white cell count – microgranular or hypogranular variant - are associated with a high risk of bleeding. Why some have high white count and some have lower is the subject of active research. The high white cell count, as explained by Professor Hunt, is a reflection of leukaemic cells spilling over into the blood. When a differential count is made of the various types of white cells (see below at paragraph [279]) most of these will be leukaemic blast cells. The effect of the failure of the cells to mature also results in a change to the molecules on the surface of the cells. Professor Hunt's particular interest is to find out whether they are regulating expressing molecules that switch on clot breakdown and possibly that they also stimulate thrombosis at the same time.

[269] These leukaemic cells (called promyelocytes) contain granules. These granules are very evident when looked at under the microscope. When the cells start to breakdown, (which by their nature leukaemic cells do) they release these granules – proteins (protease) - which activate the coagulation system. They also release plasmin, one of the most powerful enzymes in the body, which causes fibrinogen to break down quickly. There is an excess of fibrin and therefore any clot that forms is dissolved. That creates the dangerous situation where the body cannot form a clot.

[270] Although hyperfibrinolysis has been recognised to be one of the main features of the coagulopathy, the full position is not clear and research is ongoing. At the same time as hyperfibrinolysis, disseminated intravascular coagulation (DIC) may also occur. This

is a serious and life-threatening condition in which the regulatory processes that normally control coagulation break down. Numerous little clots are formed throughout the body. When this happens, as the clots are being formed, they use up the other coagulation factors. There is ongoing debate as to whether DIC is part of the disease process in APL or whether it happens independently. In APL, according to Dr Hanley, it is probably a balance between the DIC and hyperfibrinolysis. In some cases the hyperfibrinolysis will be the predominant feature. Professor Hunt's particular interest is to find out whether the effect of the failure of the cells to mature also results in a change to the molecules on the surface of the cells. Whatever the involvement of DIC, it is clear that in some clinical circumstances, there can be bleeding in certain parts of the circulation and thrombosis going on at the same time and this is a well- recognised complication in APL. It is another reason why APL can be a very challenging clinical situation to manage and, in the words of Professor Grimwade, *incredibly difficult* to treat. This view was reinforced, repeatedly, by all the haematologists who gave evidence. Thus the complexity of the coagulopathy of APL and challenges it presents in terms of management and treatment cannot be overstated.

Diagnosis of APL

[271] Acute myeloid leukaemia in general, and APL in particular, can only be diagnosed by morphology - looking at the cells under a microscope. It is not possible to diagnose leukaemia simply by reference to numerical results from blood tests. Low platelets, high white cell count and anaemia may be typical of a number of conditions and are not exclusive to the leukaemias. They are indicative of a number of differential haematological diagnoses and other non-haematological conditions such as septicaemia.

Accordingly, a blood film is essential if the leukaemia is to be diagnosed. Because of its unique chromosomal rearrangement, APL cannot be confirmed without further laboratory tests: cytogenetics to confirm the specific translocation t(15:17), or molecular diagnosis to confirm the PLM/RARA gene.

[272] However, morphological examination of a blood film may give a very clear indication that the disease is APL. It can be difficult to identify but in many cases a blood film will reveal a classic picture of the cells. Where there is a diagnosis of AML with a suspicion that it is that APL sub-type, time is of the essence and treatment must not be delayed to await laboratory confirmation. Accordingly, as soon as APL is suspected, that should be the trigger for the treatment to start, without delay. Thus the critical investigative tool for the prompt diagnosis of APL and early treatment is the blood film.

[273] It follows that for early diagnosis, there must be urgent preparation of a blood film by the biomedical scientist in the haematology laboratory and equally urgent examination of the film by a haematologist. Blood films are routinely prepared and examined in accordance with national guidelines incorporated in standard operating procedure. However, in 2004, a reduced service operated outwith normal daytime hours on weekdays whereby blood films were not routinely prepared. This was the system in operation when Kathryn was in the Victoria Infirmary and accordingly, the out-of-hours operation of the haematology laboratory was one of the main focuses of the Inquiry. The following paragraphs describe the workings of the laboratory in 2004.

Haematology Laboratory Services

[274] The evidence about the working arrangements of the haematology laboratories in the Victoria Infirmary and the Southern General Hospital came from a number of experienced and highly qualified witnesses. The Inquiry had the benefit of the combined experience of these witnesses from both the management perspective and the perspective of the biomedical scientist.

[275] In 2004 there were two distinct haematology laboratories to the Victoria Infirmary and the Southern General Hospital each having its own separate staff. During "routine" or "normal" working hours between 09:00 and 17:00 hours Monday to Friday, the laboratory dealt with all routine hospital tests, emergency tests and requests from local general practitioners. The laboratory was open to process hospital business only on Saturday mornings between 09:00 and 12:00 hours (some witnesses said 12:30) with three staff working. Outwith these times, a reduced out-of-hours service operated to deal with emergencies only.

Routine blood tests

[276] Blood tests carried out by the haematology laboratory as a matter of routine included a full blood count ("FBC") and coagulation screening. Separate samples are required for each of these tests. The full blood count contains about 13 different results, including, the white cell count (WCC) haemoglobin (hg) level, and platelet (P) count. Coagulation screening basically checks clotting times. In 2004 (as now) samples of blood for testing were transported by porters from the requesting department to the

laboratory accompanied by the relevant request form. The samples are then analysed in an automatic process.

[277] At that time, the analyser machine completed a full blood count in a few minutes (variously described by the witnesses as between 50 seconds to five minutes). A coagulation screen took longer than that, again described slightly differently by the various witnesses as anything between 20 to 30 minutes. Generally, however, coagulation results would be available approximately 10 minutes after the full blood count if these were processed at the same time. If the results were within normal parameters, the machine would "authorise" them automatically and would send them to a print queue in order to generate a paper report. Authorisation signifies that the result has been checked and is confirmed as genuine. Once the results were authorised, they could be accessed instantly by any member of medical staff in the hospital via the computer system.

[278] There was a different system for abnormal results. These would not be authorised automatically but would be highlighted by the computer and flagged up on the screen. In due course, such results would have to be authorised manually, once confirmed. In the meantime, in the Victoria Infirmary, any abnormal results would be telephoned immediately to the requesting department prior to being confirmed. No results would appear on the hospital computer system without having first been authorised. The out-of-hours system in the Southern General Hospital was slightly different. Here a system known as "ghost authorisation" operated. That allowed the BMS to release the result

without authorising it. Rather than telephoning results, they would be made available electronically on the hospital computer system.

5-Part Differential

[279] As well as producing the various blood results, the analyser was capable of producing a breakdown of the five major components of the white cell population: neutrophils; lymphocytes; monocytes; eosinophils; and basophils. These are broken down by number and classification and the total of these components should add up to 30.1. A differential should be produced where the distribution and appearance of the white cell population is normal. However, where there were certain abnormalities a full 5-part differential would not be expected. If blast cells had been detected (these are a different size from the more mature cells and often contain cytoplasm) a differential would not be produced. Specifically, the presence of blasts or immature white cells would not normally be picked up and counted. Instead they would be disregarded and "voted out". Any differential would be incomplete and the abnormal result would be flagged up in a text dialogue box. At the same time, a scatter plot would come up on the analyser's coloured screen. This is a diagrammatic representation of the cells which comes about, as Mr Rae explained, because the cells are of different size, volume and conductivity so that they scatter quite differently allowing the individual populations to be seen. A warning flag should also appear on the screen.

Blood Films

[280] A blood film is produced to enable microscopic examination of the cells. It is a crucial step in the identification and diagnosis of certain conditions, including leukaemia and certain infectious diseases, such as malaria. The preparation of the blood film is the

responsibility of the BMS. It involves a two-stage process. The initial preparation involves the spreading of a drop of blood between two slides. This also serves to preserve the sample. Thereafter, the specimen must be stained using several different dyes. This is done on a staining machine and is largely an automatic process. Once stained, the blood film is ready to be looked at by a BMS or member of the BMS or member of the haematology medical staff. Abnormal blood films are placed in a tray to be looked at by a senior member of the haematology staff (usually a consultant or specialist registrar) who will then report the result by adding textual comments on the system. That is then authorised and a paper report produced. That is known as the stage of reporting and the time of reporting is recorded.

Standard Operating Procedures

[281] In common with all the other hospital laboratories, the practices and procedure operating in the haematology lab were set out in documents known as Standard Operating Procedures ("SOP"). These required to comply with national guidelines laid down by the Clinical Pathology Accreditation or CPA. There was a requirement to review these procedures every two years.

[282] The Inquiry did not have the benefit of the relevant SOP in force in 2004. However, the relevant SOP issued on 5 November 2003 (Production 16) was produced and the evidence was that, in essence, it reflected the Standard Operating Procedure which would have been in use in 2004. Under the heading "Procedure" at paragraph 10, it reads:

> *A suitably qualified BMS will examine blood films and after checking that the staining quality and general preparation of the film is acceptable, will report on all the relevant appearances of the film*

and any further action to be taken. In general any blood films that require clinical comment are passed on to medical staff.

At the end of that paragraph it continues:

If clinical comment is required by medical staff, the form and film are placed on a separate tray. If urgently required, eg. new leukaemias, severe thrombocytopenias and anaemias the film and form must be passed directly to a member of medical staff"

A platelet count of 13 would be regarded as a severe thrombocytopaenia.

[283] The current version of the SOP is similar in its terms but more specific. The word "urgently" is used rather than directly. An additional list is included:

All results which meet the criteria set out below on the first patient's sample must have a blood film referred urgently to Haematology Medical Staff for examination; WBC greater than 30; Hg – 60; platelets below 50; any suspect leukaemias any other abnormality which the BMS is unsure/uncertain about.

Although ranges are now set out, it was clear that this was really nothing that a competent BMS should not already have known.

[284] Accordingly, standard operating procedures required that a blood film be prepared and passed, as a matter of urgency, to a member of the haematology staff, in person, for clinical comment. This was the procedure during normal daytime hours. However, the SOP was not fully adhered to outwith normal working hours.

Out-of-hours Service

[285] During these hours, what was described as "a very reduced service" was operated whereby only basic haematology work was carried out. These consisted of three routine areas: full blood counts, coagulation screening and cross-matching blood transfusion work which included the cross-matching of samples, the ordering and release of blood products and liaison with the blood transfusion laboratory at Gartnavel Hospital.

Anything outwith this basic service required to be ordered specifically. Thus, the immediate preparation of blood films was not part of the out-of-hours routine service.

[286] During the night, where results indicated that a blood film was required the on-call BMS would simply carry out the first part of the process and would preserve the sample by preparing the slide. He would not proceed to stain and produce a blood film, but would set aside the preserved sample to be stained by the day staff first thing when they came on duty the following morning. Once ready, any film requiring to be referred to a member of the medical haematology staff would be left in a tray and would be looked at first thing by one of the consultants or a registrar.

[287] Rarely, a blood film would be prepared during the night as a matter of urgency if specifically requested by a doctor. In such a situation the on-call BMS would have to be paged and instructed to make the film. It was usual for this request to come from a member of the haematology medical staff.

Treatment of APL

[288] There are two aspects to the treatment regime for APL and *both* must be instituted immediately the condition is suspected. First, there must be urgent, aggressive support with blood products to counteract the thrombocytopaenia and the hyperfibrinolysis. The aim of haemostatic support is to provide sufficient replacement of the platelets and clotting factors to prevent bleeding or to slow or stop any bleeding that has already occurred. Where there is existing bleeding, treatment is more difficult and efforts tend to be focused on damage limitation. Support from additional blood products will not undo

any damage that bleeding has already caused, but is aimed at preventing further damage. This is achieved by the transfusion of platelets and the administration of blood products containing clotting factors such as fresh frozen plasma (FFP) or cryoprecipitate. The latter preparation forms a good source of fibrinogen and enables efficient delivery.

[289] Outwith leukaemic centres, platelets may not be kept in the hospital blood bank but require to be obtained from the regional blood transfusion centre. The speed at which platelets can be obtained varies in different parts of the country. It is standard practice to "blue light "platelets from the centre to the hospital, or, to transport them by taxi, particularly during the night when traffic is light. Platelets come in "pools" which is a standard dose. They are unstable and have to be stored at a temperature of 27 degrees centigrade and kept in a permanently agitated state. This is normally achieved by placing them on a moving flatbed. They do not survive for long, a few days at most.

[290] Platelets are administered intravenously. They can be delivered quickly over a period of about 15 minutes. Their impact is fairly immediate – they quickly enter the circulating blood. Although it varies from patient to patient, a single pool of platelets might be expected to raise the count by somewhere in the region of 30 – 80 x 10^9/l, typically 50x10^9/l.

[291] In patients who are otherwise stable and not actively bleeding, a transfusion is not routinely given unless the platelet count is under 10. Where the patient is bleeding, the general aim would be to keep the platelet count at 50 or above. However, where there is bleeding in the brain and neurosurgery may be indicated, current national and

international guidelines (many of which Professors Hunt and Grimwade were involved in writing) aim to keep the platelet count at 100 prior to neurosurgery. However, according to Professor Hunt, this is not based on clinical studies and the majority of haematologists would find a platelet count of more than 50 acceptable. There is no literature to support running the platelet count at higher than 50. So, although a lot of neurosurgeons would like it to be greater than 100, there is no evidence for this.

[292] FFP consists of a mixture of several coagulation factors which are found in the plasma of the blood. It is a source of fibrinogen, but less so than cryoprecipitate. As its name suggests, in is stored in frozen form. Once ordered, it takes about 30 minutes to bring to room temperature. There is, therefore, an inevitable delay before the product can be administered. Rapid thawing will destroy the components.

[293] Cryoprecipitate is a preparation which provides a rich source of fibrinogen and is an efficient method of delivery. The court heard evidence that in 2004, it was not the first choice, although it was available. FFP was probably the product of first choice at that time. Today, cryoprecipitate would be used rather than FFP.

[294] The second aspect of treatment for APL is the administration of an antifibrinolytic drug designed to switch off the hyperfibrinolysis so that the clot breakdown is stopped. There are two ways of doing this nowadays: ATRA (all-trans retinoic acid) and an antifibrinolytic drug called tranexamic acid. The latter was not widely available and was not considered to be accepted treatment in 2004. Even now, some debate surrounds its use. ATRA was available in 2004 and was the recognised treatment for APL. However,

as a new and expensive drug with a short shelf-life rarely used outside leukaemic centres, it was not always immediately available from hospital pharmacies. It is a drug in capsule form designed for oral administration although it is possible to dissolve it and administer it via a naso-gastric tube in an unconscious patient.

[295] Giving platelets and other coagulation products alone without ATRA would be very unlikely to stop the bleeding. Such support may stop it temporarily or slow it down but it would not switch the process off and prevent continuing hyperfibrinolysis. Stopping bleeding is a much more challenging and difficult situation than preventing bleeding from starting in the first place. Without ATRA you may stop the bleeding initially with haemostatic support, but without ATRA the bleeding is inevitably going to restart.

[296] ATRA works by causing the leukaemic blast cells to differentiate or mature. They move down their developmental pathway and die. This seems to switch off the bleeding tendency. ATRA is not something that can cure APL overnight. It does not show an instant improvement. Although it can start working quite quickly – within hours of the first dose – it takes some 12 to 24 hours before any correction or improvement is seen in the coagulation parameters. It has to act on the leukaemic cells to make them become more normal. It is important to appreciate that it can be difficult to work out exactly how quickly it is working as the patient is also being supported by platelet transfusions and other blood products. It takes a while for patients to go into remission, typically around 30 days.

[297] There are dangers associated with this treatment. A condition known as differentiation syndrome can happen to patients with APL after ATRA therapy is started. The treatment pushes the abnormal blast cells to "differentiate", that is, move down their developmental pathway to become mature cells and ultimately to die. This takes time to happen and as that process is gone through, the cells aggregate and clump together. At the same time, the cells release their granular content. These granules cause a number of problems for the patient. They cause fever, and infiltrate the lungs. Fluid can collect around the lungs and the heart and kidney function can be impaired. This can result in the patient requiring to be admitted to intensive care unit for supportive therapy to help them through that stage. Although the condition primarily affects the lungs, if untreated, it would lead to multi-organ failure. It is a serious and life-threatening condition and is one of the recognised causes of early death in APL during the first 30 days of treatment. The administration of chemotherapy to reduce the white cell count and steroids in an effort to prevent the cells aggregating can ameliorate the incidence of differentiation syndrome but will not prevent it from happening completely. Thus, while it is important to control the bleeding and put the patient into remission, it has to be appreciated that the serious adverse effects of the treatment can in themselves be fatal. The treatment of APL is, according to Professor Grimwade, "a very, very tricky situation".

[298] Despite these known risks, ATRA represents a huge advance in the treatment of APL and should be commenced as soon as APL is suspected following examination of the blood film. Even if the patient is found not to be suffering from APL, little harm will be done by administering a single dose during the time taken to confirm the diagnosis.

In training others, Professor Grimwade perpetuates the mantra of his own teacher: *never let the sun go down on APL* – in other words, never delay commencing ATRA.

MATTERS NEUROLOGICAL

Intracerebral bleeding

[299] Kathryn had already suffered some sort of neurological event at home on the Sunday night and it was clear that this was consistent with the effect of the area of intracranial haemorrhage in the right frontal lobe. A bleed in that location would interfere with the seventh cranial nerve which is the facial nerve responsible for the slight drooping of her mouth which was noted and would also account for the transient episode of dysathria. Therefore, the neurological aspects of Kathryn's condition and what was happening in her brain were of fundamental importance. The following description is compiled from the evidence of the three paediatric neurosurgeons, Miss Brown, Mr Mallucci and Miss Myles.

[300] Bleeding intracranially is particularly dangerous as it occurs with the enclosed space of the skull. The skull is a rigid, boney container which cannot expand. The skull itself has a fixed volume and some of the contents of the skull are also of fixed volume, such as the brain. Other contents such as the cerebrospinal fluid or CSF do not have a fixed volume and so can be altered. The brain occupies most of the skull: broadly speaking the intracranial contents consist of 70% brain (made up predominantly of water), 10% arterial blood, 10% venous blood and 10% cerebrospinal fluid. This fluid circulates in spaces within the brain called ventricles. It not only circulates within these spaces but spreads over the surface of the brain and down the spinal cord providing a

cushioning effect and acting as a type of shock absorber. In children and young adults, the brain is tight within the skull with little surrounding space thus further limiting its ability to cope with any increase in the volume of its contents.

[301] Anything abnormal such as blood, tumour, oedematous fluid or swelling which takes up space in the brain (a "mass lesion") will increase the volume inside the skull and would be expected to cause a rise in pressure within the skull. Local mass effect occurs where a lesion is taking up space, making the whole area bigger than it was before. Thus when haemorrhage occurs, because of this lack of space, the ability of the brain to accommodate the bleed and prevent a build-up of pressure is limited. Moreover, where a lesion occurs, it will irritate the surrounding brain tissue. Over time, the brain reacts and produces an inflammatory response which breaks down the blood/brain barrier and leads to a collection of fluid around it. This watery fluid is called oedema and also takes up space adding to the mass effect. The inflammatory response does not happen immediately but is a process that builds up over time. It will develop over hours, sometimes days. It reaches its peak inflammatory process at about 48 hours (sometimes as long as 72 hours) until it stabalises.

[302] The brain has compensatory mechanisms which allow it to accommodate lesions, particularly if these are slow-growing and the brain has time to respond and accommodate over time. However, where it is a new volume over a short period of time, there is very little time to compensate and symptoms of raised intracranial pressure can develop very quickly. The brain accommodates in two main ways. First it can either shift CSF from the brain compartment into the spinal canal to create more space. It can

also reduce the production of CSF. Secondly, it can act to force venous blood out of the brain and into the general circulation and by decreasing the flow inwards thus reducing the amount blood in the skull. When it can no longer do that and compensatory mechanisms are exhausted, things are critical and life is in imminent danger.

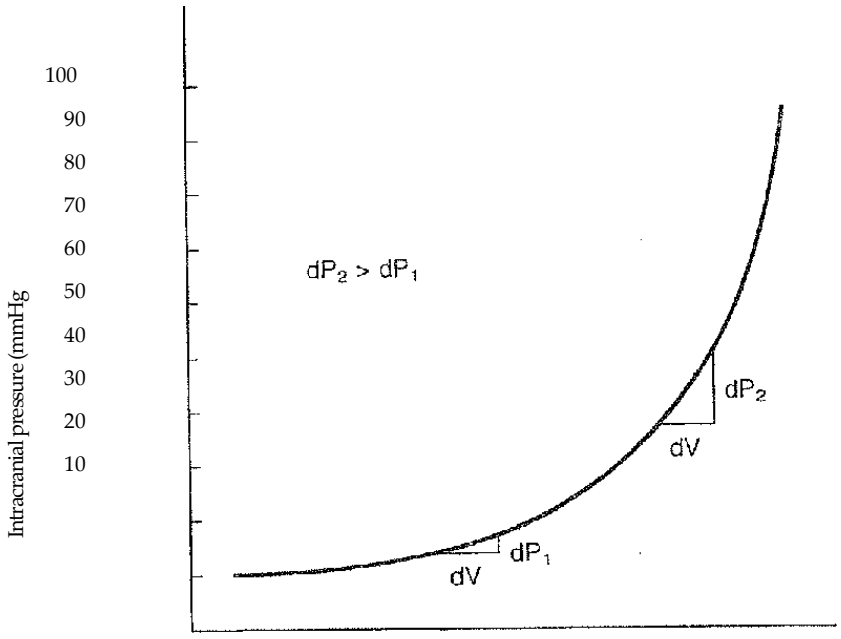
[303] As the compensation mechanism copes, the signs and symptoms of raised ICP may fluctuate with temporary recovery noted. ICP is not a single constant measure but something which changes over time. It can be measured in wave forms. A wave of high pressure may resolve as the compensatory mechanisms work but what is happening is part of a single on-going process.

[304] When there is a significant degree of mass effect, it can cause mid-line shift. This occurs when pressure from a lesion on one side of the brain forces it to encroach into the other side. The structure separating the two brain hemispheres – the midline – then shifts towards the opposite side as it is being put under pressure. If the shift is severe it means that pressure on the side of the head is causing shift to the opposite side. It is a very significant sign of raised intracranial pressure.

[305] Such structural changes are evident on a CT scan. These can include changes to the ventricles which are normally patent and filled with cerebrospinal fluid (CSF). These can

become affected by pressure from lesions and oedema within the brain. They may become squashed and therefore have less ability to accommodate the fluid. Where the intracranial pressure is raised, they may become obscured. Likewise, the basal cisterns which normally contain CSF at the base of the brain, may, in circumstances of increased pressure show signs of effacement or can be obliterated.

[306] The effect of raised intracranial pressure and the mechanisms adopted to compensate are reflected in a pressure volume curve known as the Munro-Kellie doctrine. The following illustration of the Munro-Kellie graph is reproduced from Miss Myles' report. This curve describes the fundamental basis for modern neurosurgery. The whole ethos of modern neurosurgery is to identify patients before they get on to the critical part of the curve.



Relationship between volume addition to the craniospinal axis and intracranial (CSF) pressure.

[307] The horizontal axis represents the addition of volume to the cerebrocranial contents while the vertical axis reflects the intracranial pressure. When the graph is flat, the compensatory mechanisms are active and the pressure does not increase. As the volume of the mass lesion increases, the compensatory mechanisms are overwhelmed and the patient starts to move up into the steeper part of the pressure volume curve. Initially the increase in pressure is slow but rises exponentially.

[308] At this point, when the patient is on the steep part of the curve, any small increase in volume (even 2-3 mls) can cause a large, catastrophic increase in the intracranial pressure. When this part of the pressure curve is reached, the patient is in mortal danger. Miss Myles trains neurosurgeons by repeating what was said to her in somewhat chilling terms when she was being trained: *a patient on the steep part of the curve has one foot in the grave and the other on a bar of soap*.

[309] Thus patient's position is precarious and dangerous. Any small perturbation can cause a precipitous decompensation and the patient can rapidly become comatose. So, the brain will compensate for the first part of that new volume, but at a certain point, extra tiny volumes will cause massive changes in ICP. Thus, the patient will edge along, even though the mass effect is growing, until the point of no return where tiny changes (2 to 3 millilitres of volume) can change the clinical situation rapidly.

[310] Many things can cause decompensation. Miss Myles listed several examples which included the following:-

- An increase in $PaCO_2$ levels (carbon dioxide) which acts by dilating the intracerebral vessels thereby increasing the blood volume.
- Pyrexia or raised temperature which causes the same vasodilation.
- Lying flat as opposed to sitting up as this makes it more difficult for the venous blood to leave the head.
- Straining, vomiting, crying or coughing which increases the venous pressure in the brain and causes the blood to back up in the brain increasing the volume and thus the pressure.
- Re-bleed by increasing the volume of the mass lesion.
- Increase in the volume of oedema surrounding the mass lesion.

[311] The mechanism by which raised ICP causes coma, as explained by Miss Myles and Mr Mallucci, is two-fold. First it causes brain shift and herniation which can compress critical structures of the brain such as the brainstem. This is known as Cushing's Syndrome, or coning, when the brain moves downwards. The brain stem which controls fundamental vital functions passes through a hole in the centre of the tentorium called the tentorium hiatus. The edges of the temporal lobes are very close to the edge of the hole. As the brain is pushed downwards through the hole, the brain stem becomes squashed and the vital centres in the brain stem become compromised in a critical and life-threatening way.

[312] Secondly, it reduces the cerebral perfusion pressure leading to a lack of blood supply or ischaemia. Ischaemia is the final event in any brain injury. When the ICP goes up, because it is a closed box, blood cannot get in thus depriving the brain of valuable

oxygen. The cerebral perfusion pressure drops and that compounds the on-going swelling. If untreated, raised ICP will eventually rise to the same level as the mean arterial blood pressure and at that point, central blood flow ceases and the brain dies. Cells in the brain start to swell thus exacerbating the problem. Mr Mallucci described the process as a sort of vicious deteriorating cycle: brain cells die and the more brain cells die, the worse they swell, the worse the intracranial pressure, the worse the perfusion, the worse the ischaemia, the more swelling.

Signs of critically raised intracranial pressure

[313] Abnormalities in pupil function are a sign of impending herniation syndrome. All changes in pupils must be taken seriously.

[314] Two things can cause a change in pupil size. One is local raised intracranial pressure on the one side or both sides which will initially cause compression of the third nerve. The third nerve is usually responsible for constriction of the pupil. If it is malfunctioning temporarily, this will cause the pupil to dilate. Secondly, as the brainstem malfunctions and/or dies or becomes ischaemic or compressed, then the pupils will become fixed. One of the ultimate signs of brainstem death is non-reaction of pupils. Fixed pupils usually mean that the patient's condition is unretrievable. It is very rare to recover from this. In ninety-five per cent of the cases where the pupils are fixed and dilated the patient will not recover.

[315] The ultimate non-reaction of a pupil is deemed as the near final stage. They can enlarge before they stop reacting which is a sign that things are changing and are very

serious but then they can go from enlarged to small again and non-reacting when things are more terminal.

[316] A sudden bradycardia (slow heart beat) is also an indication of imminent brainstem and brain herniation and indicates critically raised intracranial pressure. It is a very important piece of information. As the pressure goes up, the brainstem will be forced down. It is known as Cushing's Response.

[317] Miss Myles explained that with raised ICP, the perfusion in the brain starts reducing and the brain stem starts to become ischaemic through lack of blood, the blood pressure starts to rise in response. The brain stem controls the heart and blood pressure and it responds to its need for more blood supply by pushing the blood pressure up. Pressure receptors in the heart and the great vessels recognize this increase in blood pressure and respond by pushing the pulse down. Hence the classic picture of critically raised blood intra-cranial pressure: a rising blood pressure in the face of a falling pulse.

[318] Thus, pupilliary change, a rise in blood pressure and bradycardia indicate imminent death.

PART 6 CT SCAN IMAGING

Introduction

[319] In the course of the Inquiry, the interpretation and significance of the findings from the CT scan of 1:06 hours on 21 June were the subject of close scrutiny. Three areas were explored in particular: (1) the size and position of the lesions; (2) whether these were indicative of raised intracranial pressure and, if so, to what degree; and (3) whether the smaller lesions were haemorrhages or chloromas (tumour deposits). I was satisfied that the interpretation of CT scans of the brain does not lie uniquely in the remit of the neuroradiologists but is equally a matter for neurosurgeons. In was clear that, in practice, this is a task performed jointly by these two disciplines. Final reports of scans are often a collaborative effort following discussion between neuroradiologist and neurosurgeon, the latter bringing to the equation the all-important information concerning the patient's clinical condition. Accordingly in addition to the evidence of Dr Duffy, the court had the benefit of several expert opinions from Mr Mallucci and Miss Myles (consultant neurosurgeons); and Dr Keeley and Dr Forbes (consultant neuroradiologists). The court also had the benefit of hearing from Dr Evelyne Teasdale, consultant neuroradiologist at the Southern General Hospital. Although she gave evidence as a witness to fact (she was the consultant who reviewed and reported Kathryn's scans at 10:10am on 21.06.04) it was clear from the evidence that she is a highly regarded expert in her field and so I was able to place significant weight on her evidence, notwithstanding she did not appear as an expert witness. Likewise, I had the assistance of the evidence of Miss Brown, a highly experienced paediatric neurosurgeon.

[320] CT scans show different levels of light intensity. The very white, brightest areas represent bone. The darkest black areas represent air. These are the two extremes of cerebral density seen on CT scans. Structures which do not contain air or bone appear as grey areas. These are the soft tissue structures such as muscles, tendons, brain tissue, fluid, pus, etc. Findings on a CT scan of the brain are also described by reference to brain density. Thus a scan which shows an area of high attenuation, that is, brighter than the brain is described as high density – more solid than the brain. Any area of focal abnormality or lesions may be homogenous (of the same or similar density) or heterogenous (of varying or mixed density showing more than one component). CT scans are essentially pictures of slices of the brain in varying thicknesses. Kathryn's scan contained twenty images. Slides 1 to 8 were taken at a thickness of 5 millimetres; numbers 9 to 20 at 7.5 millimetres. It is possible to calculate the volume of a lesion by reference to scan measurements. Nowadays this is more accurately achieved via digital technology.

Interpretation of CT images

[321] The following represents a synopsis of the combined evidence of these witnesses in connection with the findings of the scan about which there was general agreement.

[322] The maxilliary sinuses in the cheekbones contained material which was either pus or illustrated swelling of the lining of the sinus. This finding was consistent with Kathryn having had a viral infection, as described by her parents.

[323] Kathryn's scan showed four well-defined lesions in her brain. It was possible that there was a fifth lesion situated close to and above the right frontal haematoma but this might simply have been an extension of the large lesion rather than a separate finding. The lesions were on both sides of the brain. The most significant was a large area of haemorrhage on the right side.

[324] This large lesion was situated in the right frontal lobe (the largest lobe in the brain) and was approximately 3 cm in diameter. Dr Forbes measured it at 3.2x2.5x6 mm. There was water surrounding the brain which meant that the brain had had time to react to the lesion being present. It generally takes several hours for fluid to collect around a lesion. There was local mass effect of the large frontal lesion: in other words, it was taking up space. The scan showed a loss of normal sulci, that is, a flattening of the foldings of the brain which give the cortex area its characteristic walnut-like appearance. This is another feature that implies mass effect. The abnormality was causing swelling of the right side of the brain compared to the left, consistent with mild or moderate swelling.

[325] The right frontal lesion was very characteristic of a parenchymal haematoma, that is, a bleed within the brain structures. It was a slightly irregular, well-defined lesion, predominantly hyperdense (white) but with small areas of hypodensity or darker attenuation within. There were one or two darker spots but overall it was bright. Thus it was not consistently homogenous showing that it comprised more than one component. The surrounding oedema was extensive.

[326] The other three lesions were smaller. They were all homogenous in density and were similar in characteristics to each other but qualitatively different from the right frontal mass. They were rounded and fluffy in appearance and much more homogenous. The first of these smaller lesions, measuring 12.1 x 9.8mm, was noted in the anterior right thalamus. It was surrounded by an area of oedema. The region was distorted by substantially increased mass effect. The second was noted in the right medial occipital lobe. It had similar characteristic to the second lesion and was also surrounded by oedema. It measured 30.2 x 11.6mm. The third of the smaller lesions was in the inferior left frontal lobe (just above the eye on the side of the brain). Again, this was a fairly well-rounded structure which was bright (increased density) and was surrounded by area of low density representing fluid. It measured 14.5 x 30.8mm. There was possibly a fifth small lesion just medial to and above the right frontal haematoma.

[327] The main area of haemorrhage together with the other lesions and the associated oedema represented an area of considerable mass effect due to the increased volume in the brain. The combined volume of these was significant.

[328] The scan showed that the pressure in the third ventricle was increased so that the ventricle was being squashed. There was minimal effacement of the cortical sulci in both cerebral hemispheres and partial effacement of the right sylvian fissure. These are structures filled with cerebro-spinal fluid and were partially effaced because of swelling of the right hemisphere. Both the brain stem and cerebellum were noted to be normal.

[329] While there was general agreement as to the size, position and characteristics of the lesions, the expert opinions diverged as to the likely composition of these lesions and whether the scans were indicative of significantly raised intracranial pressure.

Chloromas

[330] In the course of this Inquiry, many hours of evidence were devoted to what was ultimately an issue of limited relevance: this was a debate on whether the smaller lesions present in Kathryn's brain were areas of haemorrhage or whether they were actually tumour deposits called chloromas. Chloromas (otherwise referred to as granulocytic sarcomas) are collections of leukaemic white cells which have infiltrated the brain substance. They are metastatic brain tumours. The presence of a chloroma in the brain is a very rare occurrence. It is not possible to determine whether chloromas are present on normal non-contrast imaging. A contrast image is required. This is achieved by the injection of an intravenous contrast "dye". If there is a tumour the contrast will leak out of the tumour and cause it to enhance. A chloroma would show up as a characteristically intensely bright area – as Dr Forbes put it, it will light up like a light bulb – and be easily identified. Chloromas take some time to form – at least a few weeks. One of the features of such tumours is that they can and do bleed. Thus haemorrhage can be superimposed on the site of a tumour.

[331] Although this was a most interesting feature, ultimately whether these lesions were chloromas or haemorrhage was of little import. What was important was that they were all space occupying lesions which, together, were capable of causing significant mass effect. It was not possible to determine what these lesions were in the absence of

histological examination from a biopsy during life or dissection at autopsy. So, the question was largely academic. However, it did have some relevance in relation to Professor Hunt's opinion on hyperfibrinolysis. Furthermore it seemed to be an issue of significance to the family who seemed anxious that it be established that these were *not* chloromas. Accordingly, it is appropriate to examine this evidence.

[332] It was Dr Teasdale who first raised this possibility of the presence of chloromas. In reporting Kathryn's first scan, she questioned from a radiological point of view whether there were underlying tumour deposits. She commented that the appearances were unusual and that pigmented or haemorrhagic secondary deposits should be considered. In her evidence, Dr Teasdale said that these were almost certainly haemorrhages but later parts of her evidence seemed to contradict this conclusion. She said that the smaller lesions might be secondary deposits. They were very well defined and smaller. It was unusual to get haemorrhages like that spread throughout the brain and therefore secondary deposits must be considered. They had the look of tumour deposits and were certainly likely to have bled as well as being simple deposits. Although the most common high attenuation lesion is blood, some things that looked like blood were not: for example, some tumour deposits with high cellular content that could also bleed as well. Thus Dr Teasdale included tumour deposits as a differential diagnosis of Kathryn's scan. It is worth noting that this was despite the fact that she had no clinical information about Kathryn at all.

[333] The possibility that the smaller lesions were chloromas was put forward by Miss Myles and Dr Keeley. Like Dr Teasdale, Miss Myles commented that it was unusual to

get four spontaneous areas of haemorrhage happening simultaneously. In the absence of some structural cause, that would be highly unusual. The appearance of the other three lesions was similar to each other but different from the main one. They were well-defined, rounded in shape and fluffy in appearance. They were much more homogenous that the main lesion. She thought that the three lesions looked very strange. They did not look to be of different ages, hence the very unusual feature of multiple haemorrhages appearing spontaneously in different parts of the brain.

[334] She therefore considered that chloromas should be included in any differential diagnosis of Kathryn's scan. She could not say categorically that these were chloromas as it was not possible to distinguish between chloromas or haemorrhage in the absence of contrast scans or laboratory further investigation.

[335] When Miss Myles had originally looked at the scans, she sought a neuroradiological opinion from her consultant colleague, Dr Keeley. Dr Keeley was called as a witness and gave evidence which echoed that of Miss Myles. She saw that the three masses were all similar to each other but were qualitatively different from the right frontal mass. She noted the same characteristic as well-defined, ovoid or circular in appearance and homogenous. Had she been asked to report on the scan, she would not have been happy to report the findings as four areas of haematoma alone but would have had to consider the possibility that they represented rare parenchymal chloroma deposits as this would have had an impact on the patient's treatment, management and potentially on prognosis. They may also have been haemorrhages superimposed on pre-existing leukaemic deposits. So from a qualitative assessment, there were sufficient

differences between these three lesions and the larger one to raise a concern that they may not be uncomplicated spontaneous haemorrhages.

[336] Dr Keeley had first-hand experience of a chloromas within the brain in her practice in the USA. This was in a young child of about three or four years old who had died while undergoing treatment for leukaemia. Although Dr Keeley could not remember the specific type of leukaemia or the particular stage of treatment, what was interesting was that a large chloroma had been found at autopsy. This was a large hyperdense mass in the left cerebellar hemisphere. During repeated scanning, it had always been reported as a haematoma. She also referred to an article by Guermazi *et al: Granulocytic Sarcoma (Chloroma): Imaging Findings in Adults and Children* from The American Journal of Roentogenology 178 February 2002 confirming that granulocytic sarcomas have been observed in patients with acute myelogenous leukaemia and may develop during the course of, or as a presenting sign of the disease. It also confirmed that intracranial parenchymal masses have been reported, although rarely. A non-contrast CT image was reproduced of a parenchymal mass in the brain which was found to be a chloroma. That was an example of an intraparenchymal chloroma.

[337] Therefore, in Kathryn's case, Dr Keeley concluded that there were three reasonable hypotheses: first, that the lesions were all haemorrhages; second, that they represented tumour aggregates or chloromas; third, that they represented chloromas that had associated haemorrhage within them.

[338] Dr Forbes gave evidence in similar terms but with slightly different emphasis: he was firmly of the view that the lesions were local areas of haemorrhages, rather than chloromas. A chloroma within the brain was a very uncommon presentation as they were more likely to be on the outside of the brain than infiltrating the brain tissue. He agreed with the observations about the appearance of the three smaller lesions although he thought that one was less rounded and had a more irregular shape. (Miss Myles and Dr Keeley thought that the change of shape noted on one or two of the slides was a feature known as partial volume effect because of the slide's thickness and not that it was actually a different shape.)

[339] During his evidence Dr Forbes repeatedly stressed that haemorrhages were much more common than chloromas statistically and that, on a balance of probability, the lesions were more likely to be haemorrhages and less likely to be chloromas. In this sense he was applying ordinary population statistics.

[340] Professor Yin also supported the view that these were haemorrhages. He was less willing to consider the possibility that they may have been chloromas. He challenged the opinions of Miss Myles and Dr Keeley. He had never, in his thirty years as a haematologist heard of chloromas. That had prompted him to do some research (restricted to chloromas at presentation) in the course of which he was only able to come across one reported case from Japan of a lady who presented with a chloromas in the cerebellum. While he accepted that chloromas can occur in acute myeloid leukaemia, they tend to occur outside the bone marrow and can go into the soft tissues near the spinal cord but he had never heard of chloromas in the brain. He had never seen any

patient with chloromas at presentation and nor had he read that solid tumours like chloromas could be found at presentation. Therefore, chloromas in the brain were exceedingly rare and therefore he would challenge any view that Kathryn had chloromas in her brain. He was strongly of the opinion that all the changes were due to intracranial haemorrhage and, it had to be said, was really not open to the suggestion that they might be chloromas, however rare.

[341] Mr Mallucci's view was that whether the lesions were chloromas or haemorrhages was largely unimportant, a view shared by Miss Brown. From a neurological perspective, in terms of simple volumes, it did not actually matter what they were. The difference would be that a chloroma would probably have built up over time and so Kathryn would have accommodated it and may then have bled into it. He pointed out that the operation note did not describe the evacuation of haematoma and jelly-like tumour which might go against the chloromas theory but it was often difficult to differentiate clot from tumour. Nor was there any pathology to assist as the evacuated clot was not sent for histological examination. However, he considered that the theory that these were chloromas and that they had been present for days was a reasonable one because Kathryn had neurological events at home before she was admitted to hospital. It was possible that there was a chloroma present before which might have had a mild bleed and then a bigger bleed on the day. Miss Myles suspected that this had indeed been the case: that the actual cause of the bleeding was that Kathryn had bled into a pre-existing chloroma in the right frontal region. She commented that this could have happened whether or not the clotting was abnormal. However, both she and Mr Mallucci agreed that the importance for the neurologist was not what they were,

but the fact that they were *space-occupying* lesions which cause intracranial pressure which could lead to decompensation, either by re-haemorrhage or by swelling.

[342] Miss Brown agreed that there was no way of knowing now whether these lesions were haemorrhage or tumour deposits but that a tumour deposit was a possibility. However, like Mr Mallucci, she did not consider it a matter of particular importance. She highlighted the difficulty in identifying such tumours where bleeding had occurred because it was not always obvious that there was an underlying tumour. Although no tumour was identified during Kathryn's operation, that did not necessarily mean that none was there. A relatively small tumour at the base of this clot might not have been obvious at the time of surgery although such a tumour could have been the source of that clot. It could have been at the very base of the clot. This area had not been explored during Kathryn's craniotomy as the decision had been taken not to extend the surgery deeper into her brain. Mr Mallucci likewise confirmed that it is difficult to distinguish clot from tumour.

[343] In the course of this debate, much emphasis was put on the rarity of the chloromas and the far greater occurrence of haemorrhage in the population. That being so, Dr Forbes argued that it was far more likely that these were haemorrhages. Similarly, there was a practice to look for one single diagnosis for all four and, given that it was known that the large area was haemorrhage, then it followed by applying that argument that the others, too, were areas of haemorrhage. Both Miss Myles and Dr Keenley agreed that, of course, haemorrhages were much more common but pointed out the dangers of applying population statistics in such circumstances. One had to be cautious in applying

population statistics to an interpretation of a specific scan. It was vital to interpret the scan in a clinical context, in this case leukaemia. The potential cost to the patient of misdiagnosis was high and therefore it was appropriate that all reasonable hypotheses were considered and differential diagnoses pursued rather than simply relying on what was statistically a more common diagnosis. Likewise, in applying the concept of a unifying single explanation, leukaemia could be a unifying diagnosis, albeit a more rare manifestation.

[344] The relevance of this evidence to Professor Hunt's opinion in connection with the impact of hyperfibrinolysis merits examination. From her evidence, it appeared that she might have understood that the lesions had been confirmed as chloromas. She had been surprised by this. However, it was clear that her theory about the impact of hyperfibrinolysis was not dependent on there being chloromas. It was related to the infiltration of leukaemic cells in the brain. Professor Yin agreed that such infiltration would have occurred: the leukaemic cells would have been "everywhere". Therefore, I was entirely satisfied that the unresolved issue of chloromas did not in any way affect or undermine Professor Hunt's evidence about hyperfibrinolysis.

Conclusion

[345] The proposition that the smaller lesions were a rare presentation of chloromas was a compelling one. I was impressed by the careful and logical assessments presented by Miss Myles and Dr Keeley. At no time did they state categorically that the lesion were in fact chloromas. They simply raised this as a differential diagnosis – one which, although rare, merited consideration. Essentially, that reflected Dr Forbes position also. However,

I was not persuaded by his insistence on the application of population statistics to the issue. There was a clear need to consider the matter with reference to the individual clinical circumstances of the patient. Nor was I convinced by Professor Yin's opinion which seemed to be based primarily on the fact that he personally had never come across this phenomenon. He seemed somewhat dismissive of Miss Myles' and Dr Keeley's evidence on this point. That was so even when he had only carried out limited research on the internet immediately prior to coming to court. That research had disclosed only one recorded case. However, Dr Keeley had personal experience of a chloroma within the brain in her practice and referred to an article which confirmed that interparenchymal chloromas are found as a presenting feature of leukaemia, albeit rarely.

[346] Other witnesses, including Mr Mallucci and Miss Brown found the chloroma hypothesis to be entirely reasonable, even to the extent of Miss Myles' suspicion that the initial bleed could have been into a chloroma in the right frontal region. Although we will never know, the hypothesis that these were a rare manifestation of chloroma cannot easily be dismissed. On the contrary, the case for chloromas was a strong one, particularly when one takes into account the evidence of many of the experts that it was very unusual to see multiple cerebral haemorrhages scattered in both sides of the brain at the same time.

[347] In the absence of a post-contrast scan or histological confirmation, it was clear that there could be no definitive answer to the haemorrhage/chloromas. That being so, it is not appropriate that I make any specific finding. Nor is it necessary for me to do so. As

was pointed out, at the end of the day, it did not matter what these lesions actually were. It was their space occupying properties together with the associated oedema that was of crucial significance in terms of the build-up of pressure inside Kathryn's head.

Did the CT scan at 1:06 suggest raised ICP?

[348] There was some disagreement as to whether the scan images showed evidence of raised intracranial pressure. However, more fundamental was the question whether any reliable indication of the pressure inside the skull could be obtained from looking at a scan alone. Miss Myles explained that a scan is a series of images showing tangible structures and contents of the skull. Pressure inside the skull cannot be measured with any accuracy unless by a special instrument called a pressure bolt which is inserted into the skull. According to Miss Myles, scans are notoriously poor at predicting intracranial pressure with any accuracy. There can be dangerously high ICP with a scan that is apparently normal. Conversely, pressure can be normal even where a scan shows significant mass effect. Moreover, scans required to be interpreted, not in isolation, but in the context of the patient's clinical condition. The other experts agreed with her. Accordingly, I was satisfied that any estimate of the level of intracranial pressure from a scan image required to be approached with a degree of caution

[349] All of the clinicians who treated Kathryn were alarmed by the scan images and worried that they showed evidence of raised intracranial pressure. Dr Duffy was concerned about the mass effect being caused by the lesions. She considered the mid-line shift (which she measured as 3mm) to be significant in a child and believed that the scan was indicative of a build-up of pressure. Such was the risk of Kathryn's conditions

worsening that she wished her out of the scanning area and into a safer clinical environment. Dr Weir was "incredibly concerned" about Kathryn's condition after viewing the scans.

[350] Miss Brown described the scan as "very worrisome", particularly as all the bleeding was within the brain rather than on the surface. She was concerned because lesions were bilateral. She, too, agreed that the scan showed a degree of mid-line shift and features suggestive of raised ICP. It was a dangerous situation where deterioration could occur "very precipitously". Miss Brown's assessment of Kathryn's raised ICP was that she was at a "tipping point". She considered that Kathryn was on the steep part of the pressure volume curve when she was in the scanner. While there, she may have had a transient wave of high pressure when she was in the scan which she survived because of the compensatory mechanisms. Although she still had some reserves, these were being used up.

[351] Dr Teasdale did not consider that there was evidence of raised ICP from Kathryn's scan. She considered that the degree of mid-line shift – which she estimated was nearer 2mm – was not a worrisome feature. She commented that people could walk about perfectly normally with a shift of up to 3mm. In her opinion, where, as here, there was a mass lesion but the third ventricle and the basal cisterns were present, that was an implication that there was no general raised pressure in the head in radiological terms.

[352] Dr Forbes believed that the scan did show evidence of raised intracranial pressure but only to a mild to moderate degree. The third ventricle was still visible although it

was very small from being squashed by the brain. The basal cisterns appeared normal whereas they would be obliterated in circumstances of severely raise ICP. The frontal walls of the lateral ventricles were less full than they should have been and so were slightly compressed, again indicative of a mild to moderate increase in pressure. It was difficult to be certain of the degree of midline shift as the head was slightly asymmetrical but he estimated it at only about 2 to 3 mm of shift. He also noted the minimal effacement of the cortical sulci in both cerebral hemispheres and the partial effacement of the right sylvian fissure. As Kathryn's compensatory mechanisms had not yet been used up, he considered that the scan was not indicative of a critically raised pressure.

[353] Miss Myles and Mr Mallucci had other views. In reviewing Kathryn's scans for the purpose of the Inquiry, Miss Myles had been immediately concerned by what she saw. The main lesion was a serious blood clot causing considerable mass effect. The third ventricle was effaced and there was significant oedema. She also noticed that the lateral ventricle on the right side was starting to become trapped by the blood clot so that it was bulging into the ventricle and preventing a pocket of CSF from draining properly. These were all signs of raised ICP. She identified mid-line shift but totally disagreed with Dr Teasdale's opinion as to its significance. She explained that neuroradiologists did not treat patients, they reported on scan findings. Miss Myles measured the mid-line shift as 3 to 4 mm (being aware that the head was "slightly off-kilter") but what worried her was that the shift was present with lesions on opposite sides of the brain. That was a very dangerous situation where lesions on one side were taking up space and pushing one way while the same was happening on the other side pushing in the opposite direction. In Kathryn's case, the big lesion on the right was slightly winning at that point.

However, in such a situation the midline shift is falsely reduced because of pressure from the opposite direction. She described this as a hugely dangerous position to be in neurologically because it predisposes central herniation. There was an increased the danger of the brain moving downwards as it was squeezed in two opposite directions. In the enclosed space of the skull, there was only one way the brain could go and that was downwards. This would lead to coning at which point there was a risk of sudden death.

[354] As to Dr Teasdale's remarks that a person could walk about with 3 mm midline shift, Miss Myles' view was that, whilst that might be so in certain long-standing conditions, a 3mm shift was very abnormal in a child who had four space occupying lesions in her brain and had to be assumed to be significant. She did not believe that neuroradiologists necessarily understood the implications of that because they did not treat patients. They interpreted the scans but did not necessarily take into account other clinical information or consider consequences. Thus a neurosurgeon would not have dismissed that degree of midline shift as being minor or insignificant because of the lesions on both sides of the brain.

[355] Mr Mallucci was in no doubt that at 1:06 Kathryn had raised ICP and that she was on the steep part of the volume curve and that her overall situation was critical. Miss Myles' opinion was that the episode in the scanner was the start of the decompensation. Kathryn was approaching the vertical part of the curve and things were balanced on a knife-edge at that point.

[356] Indeed, Miss Myles was of the view that Kathryn *might* have been experiencing symptoms of the early stages of increased ICP at home and in the days leading up to her admission. She had symptoms which were consistent with that: headache, vomiting, neck pain and symptoms of a flu-like illness. These were typical prodromal (premonitory) presentations of neurological deterioration in children where raised ICP is frequently mistaken for a viral illness or gastro-intestinal upset. Kathryn also had slightly increased blood pressure and was breathing slightly quickly. The latter would have meant that her carbon dioxide levels were low. Over-blowing is one of the brain's compensatory mechanisms to counteract raised ICP. So Miss Myles thought that perhaps Kathryn had raised ICP in the week before but was compensating. That was a step too far for Mr Mallucci.

[357] He did not go so far as to agree that Kathryn was showing signs of a global raised ICP. There was evidence that she had had a bleed earlier which he thought had caused the reported neurological event. However, he could not say whether that meant that Kathryn had raised intracranial pressure at that home although two or three hours later she had a scan that showed raised ICP. She was clearly building up with raised ICP during those hours in A&E. He thought it likely that she had raised ICP most probably started by an event immediately before that which led to the ambulance being called.

[358] In the course of Miss Myles' evidence, it became apparent that the term "critical" might have been used in different ways by the witnesses. Again, I found Miss Myles evidence clear and cogent. In cross examination, she was asked - repeatedly - whether the fact that there was no evidence of herniation and evidence of some compensatory

mechanisms still available meant that the ICP was not critical at that stage. With understandable frustration, she explained that the scan simply suggested that the brain had not shifted in a critical way yet. The scan showed a picture. Absolute intracranial pressure could not be determined from a scan. Looking at the scan she thought that the pressure was raised and this was a critical situation. The fact that there was no downward herniation did not mean that it could not have happened in the snap of a finger. That is exactly what can and does happen – tiny amounts of additional volume will cause massive rises in pressure and herniation which happens in seconds or minutes. A scan gives a snapshot picture of what is happening at a particular point in time. She said that it was important to distinguish between pressure and herniation. Pressure causes herniation but a scan can only show the physical process of herniation. It cannot show the pressure. At the point when the scan was taken, it did not show that Kathryn had herniated into her brainstem. If what was meant by a critically raised ICP referred to the point of coning then she agreed that the scan did not show that. She explained that once herniation occurred, the patient was almost certain to die. For her, critically raised pressure corresponded to the earlier stage when the patient was half way up the vertical curve but was still compensating so that something could be done about it. The scan showed evidence that Kathryn was up there on that curve – the effacement of the third ventricle, the midline shift and the bilateral lesions were big warning signs.

Conclusion

[359] I accepted the evidence of Miss Brown, Miss Myles and Mr Mallucci and was therefore satisfied that at the time of the scan, Kathryn was in a perilous state as a result of raised intracranial pressure. It was clear that disaster could have struck at any time,

even although her compensatory mechanisms had not been exhausted. It was clear that the transitory episode in the scanning room was an indication that she was running out of the ability to accommodate additional volume. There was compelling evidence that suggested she was on the steep, vertical part of the curve which meant that very little could push her into decompensation. I rejected Dr Teasdale's position that the scan showed no evidence of raised pressure. It seemed to me that Dr Teasdale's evidence took no account of Kathryn's clinical situation. Dr Forbes, too, did not seem to take account of the clinical situation. I did think that there was a question of semantics involved among the experts as to whether the scan showed a critical situation or a critical amount of raised ICP. The important matter was at the time of the snapshot provided by the scan, Kathryn was on that dangerous part of the curve.

Precipitous deterioration shortly before 02:00

[360] What happened to Kathryn shortly after she was brought back to the A&E department was a crucial stage in the sequence of events. There appeared to have been an initial show of symptoms of raised intracranial pressure when her blood pressure rose to $^{130}/_{70}$, her pulse dropped to 70, her respiratory rate increased from 16 to 24 breaths per minute and her right pupil stopped reacting. Her GCS plummeted from a full score of a girl who was fully awake and alert to a score of 6 or 7 which meant that she was in a coma. It the appeared that her condition continued to deteriorate because she became bradycardic with a pulse of 48 and her right pupil dilated. These were signs of an immediately life-threatening rise in intracranial pressure.

[361] The intensive care team arrived and proceeded to anaesthetise Kathryn. She was intubated and ventilated. She was given Atropine to raise her heart beat. On the instruction of the neurosurgeons, Mannitol was administered in an attempt to reduce the swelling in the brain, all measures designed to reduce the pressure and its effects.

[362] Mr Mallucci's opinion was that Kathryn had deteriorated step-wise between 1 and 2 am and then precipitously to coma at around 2am. Despite the occasions when she was alert and talking, this was probably "fluctuate" – in other words, reversible until the deterioration became irreversible by 2am. Miss Myles agreed that the deterioration occurred in a step-wise fashion as part on an on-going process until she could no longer compensate. Both agreed that it was impossible to say exactly what had caused this although it was almost certainly a combination of a number of factors. It was a complex, multi-layered process.

[363] Among these factors was the possibility that Kathryn's haemorrhage had re-bled. Mr Mallucci put a re-bleed – not necessarily a large one – at the top of his differential diagnosis but agreed that other factors, particularly brain swelling and ischaemia might have played a part and that any re-bleed probably happened in conjunction with other things. Other factors including increased swelling round the clot, changes in fluid resuscitation, coughing, lying flat etcetera could have influenced her deterioration. It was impossible to say without a repeat scan. Miss Myles' conclusion was more open-ended: it was not possible to determine what factor or factors actually pushed Kathryn over the tipping point but she considered that it was is likely to have been a combination of a number of things, among which was a re-bleed.

[364] Mr Mallucci clearly suspected that a re-bleed had occurred which, together with other factors, resulted in a serious brain insult which meant that Kathryn's position was no longer salvageable. One of the reasons he favoured a re-bleed was that Kathryn's condition deteriorated relentlessly from then on. Both pupils were fixed and dilated by the time she arrived at the Southern General which meant that she had continued to deteriorate despite the fact that she had been intubated and anaesthetised and that she had been given Mannitol. In retrospect, it appeared to him that Kathryn had suffered some irreversible brain damage as a result of herniation and coning. Had there been a second scan he suspected that it would have shown a much worse picture than the first.

Conclusion

[365] It is impossible for me to determine as a matter of fact what influenced Kathryn's decline at 2am. I am satisfied, however, that it was almost certainly due to a combination of circumstances. It was most unlikely to have been due to one single event. The picture was a complex one. There were a lot of factors at play and it was clear that there any number of things could have contributed to her final decompensation. Among those factors was likely to be a re-bleed. There was much sense in Mr Mallucci's analysis which would have explained why Kathryn continued to deteriorate and would support his proposition that once that had happened, the position was irretrievable. Taking account of Mr Mallucci's opinion, I think it likely that a re-bleed was at least part of the equation.

Disputed evidence about Kathryn's precipitous decline

[366] It was the position of Dr Beattie and Ms Crawford that no such decline occurred. Instead, they believed that Kathryn's comatose state was entirely as a result of the fact that she had been intubated and anaesthetised as a routine precaution for her transfer to the Southern General. Accordingly, the circumstances in which Kathryn was intubated and anaesthetised in the Victoria Infirmary as described by the staff were vehemently disputed by Kathryn's family. Dr Beattie, Ms Crawford and Dr Rosaleen Beattie were adamant that Kathryn had been fully alert and talking right up to the point at which she was intubated with a GCS of 15. Ms Crawford said that she had been told that this was simply a routine precautionary measure for her safe transfer to the Institute. Dr Beattie suggested, late on in the proceedings, that Kathryn's condition was due to the drugs that she had been given.

[367] There are several reasons why I have rejected the family's position. First, children are not intubated without good reason. I accepted the evidence that it was *not* routine practice to do so. Anaesthetising a patient is a procedure which carries its own risks and is not undertaken lightly. I accepted Mr Mallucci's comment that no reasonable transfer/ anaesthetic team would intubate a child unless it were necessary to do so. A patient with a GCS of 15 would not be intubated. Dr Davidson shared that view. Far from being a decision taken routinely in order to transfer patients to the Institute as was suggested, I was wholly satisfied that the decision to intubate was in response to Kathryn's dramatic deterioration and that in the circumstances it was a necessary, life-saving procedure. I

preferred the evidence of Dr Davidson as to the "desperate" situation which had developed.

[368] Secondly, the desperate nature of the situation was evident with reference to the drugs that were administered, specifically Atropine and Mannitol. These are powerful and dangerous drugs that are by no means routine. Mannitol is prescribed on instruction of the neurosurgeons and is designed to reduce intracranial pressure in critical situations. Likewise, Atropine is a powerful drug usually administered by anaesthetists, again in critical situations where cardiac arrest may be imminent.

[369] Thirdly, this rapid deterioration was something which was not entirely unexpected: the potential for Kathryn to have deteriorated in such a fashion had been recognised and prompted her return to the resus area in A & E and it was the basis of the decision to transfer her to the Southern General, so that if she did require immediate surgical intervention in response to such a deterioration, she was in the right place. The expert witnesses all agreed that she was in a very precarious position as far as intracranial pressure was concerned with compensatory mechanism being used up. Disaster could have happened at any time.

[370] Fourthly, the initial plan had been to transfer Kathryn to ward 66, the neurosurgical children's ward, for observation. At that time she was stable with a GCS of 15. I was satisfied that she would not have been intubated in these circumstances. The plan to transfer her to ward 66 could not have been carried out. Ward 66 did not have the facilities to look after an intubated patient and would not have accepted Kathryn.

Then, as now, all intubated patients had to be admitted into intensive care. The change of plan to take her to intensive care was clearly the result of a worsening and critical situation.

[371] Fifthly, the various alternative reasons for the change in Kathryn's condition which were put forward by Dr Beattie were considered but readily excluded by Mr Mallucci. It has to be noted here that these suggestions were never put to the anaesthetists involved. Dr Beattie produced the 2004 British National Formulary. With reference to the usages and contra indications noted in the pharmaceutical guidance, he suggested that the various drugs used were given for reasons other than to counteract a deterioration in her condition - for example that they were used to offset some of the side effects of the anaesthetic drugs. I was left with the impression that these matters had arisen later in Dr Beattie's researches in his dogged attempt to establish that there had been no deterioration in Kathryn's condition.

[372] Lastly, the deterioration that occurred and the mechanisms involved accounted for the fact that the deterioration, as Mr Mallucci pointed out, was relentless and did not respond to powerful drugs. To suggest that she had simply been intubated for transfer and had not suffered such a decline would have meant that her condition had been mistakenly confused for life-threatening intracranial haemorrhage when she was just under routine anaesthesia. That was in effect what Dr Beattie was alleging – that and the fact that in these circumstances she did not required to have surgery at all. That proposition was as absurd as it was impossible and I entirely reject it.

[373] There was no doubt that the decision to intubate and anaesthetise Kathryn was the right thing to do. It was essential for Kathryn's safety during transport and was the correct treatment in order to reduce some of the ICP. It protected her airway during transit and prevented regurgitation of stomach contents. It also controlled her $PACO_2$. Mannitol was given to reduce her ICP by taking water out of the brain and Atropine was given to increase her heart rate. These were the correct responses to Kathryn's condition and were endorsed by the experts.

Haemodilution

[374] Finally, before leaving the issue of Kathryn's sudden deterioration, it is appropriate that the issue of haemodilution is mentioned. This was a matter which arose during the Inquiry and was considered in some detail. The issue was whether intravenous fluids given to Kathryn on admission had caused a degree of haemodilution and, if so, whether that had affected her ability to form clots. It was suggested by counsel for Ms Crawford and by Dr Beattie that while in A&E Kathryn had been given intravenous fluids both inappropriately and in excessive amounts. This, it was suggested, had led to a significant degree of haemodilution and had compromised her clotting ability and thereby contributed to her death.

[375] Haemodilution is the term used when the blood volume is diluted by the administration of fluids, mostly intravenous fluids. The concentration of the components in the blood – the cells, platelets and coagulation factors - will decrease if the amount of blood volume is increased by fluid.

[376] Dr Thomas prescribed fluids shortly after Kathryn had been brought into A&E. Dr Thomas considered that Kathryn's history and presentation were indicative of a degree of dehydration: Kathryn was an ill child who had been vomiting and had a fever during the previous six days; she was clammy and tachycardic on admission with signs of possible infection. In these circumstances, Kathryn's fluid intake was likely to have been reduced while, at the same time, there would have been an increase in her baseline fluid requirement. Accordingly, Dr Thomas prescribed one litre of normal saline in the first instance, her aim being to replace depleted fluid and restore Kathryn's normal fluid baseline. It was delivered via two 500 ml bags. The first was put up at 22:30 hours and infused over a fifteen minute period. The second bag was started at 22:45 hours until 23:10 and delivered over a 25 minute period. Thus the full litre was delivered within a 40 minute period. The fluid balance chart and the prescription chart were unclear as to the exact amount of fluids that had been administered thereafter but it would seem that Kathryn received about 1.2 litres of normal saline and about 500 mls of 5% dextrose (sugary water) when she was in A&E .

[377] It was Professor Yin who was critical of the amount of fluids given. However, at the end of the day, he was merely critical in retrospect. He was satisfied that it was reasonable to have given about a litre of fluid as infection was part of the differential diagnosis in someone who was tachycardic and possibly dehydrated. He did not consider that it could be said that such treatment was dangerous at the time it was given. However, in retrospect, the giving of so much fluid could inadvertently have diluted the essential components for stopping haemorrhage it could – and he stressed he was simply saying "could" – have contributed to her deterioration. In total, Professor Yin's

evidence amounted to the fact that it was possible that Kathryn's ability to form clots could thereby have been compromised. In that regard, he was critical of the decision to prescribe the fluid.

[378] Professor Grimwade disagreed. His evidence on this was that intravenous fluid hydration would be standard practice in the treatment of acute leukaemia in order to reduce some of the major complications of chemotherapy, namely tumour lysis syndrome and possible renal failure once the leukaemic cells started to break down. He considered that commencement of intravenous fluids and antibiotics in A & E was not only reasonable given Kathryn's presentation but would have been the appropriate course of action had the diagnosis of acute leukaemia been known. That was not to say that the fluids might not have had an impact on the coagulation system but everything was a balance and, typically, fluids would be given in such circumstances.

[379] Miss Myles, too, agreed that there had to be a balancing exercise. Although Miss Myles gave evidence in her capacity as an expert in neurosurgery, I was aware that she had a specific interest and qualification in physiology. Miss Myles described in some detail the physiology associated with fluid therapy explaining that although a litre of fluid sounded a lot, attention had to be given as to where that fluid actually ended up. With reference to the percentage of the plasma compartment: extra-cellular fluid compartment ratio, for every litre of fluid, only a quarter of that - 250 mls – could be expected to stay inside the bloodstream. The 5% dextrose would immediately have been distributed throughout the total body water so that only about 80-90 mls of 5% dextrose would actually have ended up in the bloodstream. She also explained that in a young

person, as soon as the fluid goes in, mechanisms immediately work to get rid of the fluid in the urine.

[380] Professor Hunt was asked about haemodilution in her capacity as the expert on coagulopathy. She agreed that there would have been some haemodilution but it would have been present in a minor way because Kathryn had normal renal function and would have been excreting most of that fluid. Thus as fast as the fluid was being put in it would have been passed out in the urine. Therefore Kathryn was not having her plasma volume expanded very much. Similarly, it would have reduced her platelet level in only a very minor way. Professor Hunt was firmly of the view that it was not responsible for all the changes in the coagulopathy, and it did not account for the fall in the fibrinogen level.

Conclusion

[381] I was entirely satisfied there was nothing to support the proposition that Kathryn was given excessive amounts of fluid which diluted her blood to any significant extent. Such dilution as there was would have been minor. Equally, I was satisfied that the giving of fluids was the correct response to Kathryn's presenting condition and that the amounts prescribed were appropriate. Moreover, there was clear evidence from Professor Grimwade that intravenous fluid therapy would have been the correct treatment for leukaemia, albeit coincidentally. Thus the extent to which these fluids further compromised Kathryn's clotting ability was minimal and did not contribute to Kathryn's deterioration or her death in any meaningful way.

Decision to proceed to surgery

[382] The haematologists and neurosurgeons were of the unanimous opinion that, as a general rule, surgery is contra-indicated in a patient with a profound and on-going coagulopathy. Any patient with grossly abnormal clotting risks further bleeding so before embarking on a neurosurgical intervention the surgeon must be confident that the bleeding can be controlled. Accordingly, the general view was that such patients require to be treated primarily by the haematologists with the aim of stabilising the patient by bringing the clotting within normal parameters prior to any surgery. Without that, the risks associated with surgical intervention are obviously high. Thus, as a general rule, it follows that where surgical intervention is contemplated, it should not proceed without input and advice from a senior haematologist.

[383] Kathryn underwent a craniotomy to remove the blood clot from her brain before her coagulopathy had been fully addressed. Miss Brown made the decision to proceed. She was fully aware that Kathryn had serious clotting problems and was particularly concerned that there was a consumption problem whereby the clotting results could shift rapidly. However, given Kathryn's condition when she arrived at the Institute, Miss Brown judged that time was of the essence. Kathryn needed urgent neurosurgical intervention to remove the clot and reduce the pressure inside her skull. This was life-saving surgery. Without it, Kathryn would have died.

[384] Although the target platelet count of 100 had not been achieved and clotting had not been controlled, there had been a good response to the first platelet transfusion when her count had increased from 13 to 69. That being so, it could reasonably be anticipated

that similar increments would be achieved from further transfusion of platelets during surgery. Miss Brown instructed that all blood products released by the haematologist should be transfused so that there was adequate platelet cover during the operation. Accordingly, while her decision to operate did not depend on correction of the coagulopathy prior to surgery, it did depend on Kathryn's good response to the platelet transfusion and the availability of further platelets during the procedure. Miss Brown did not consider that the timescales involved allowed for preoperative normalisation of coagulopathy and laboratory confirmation of the position. Kathryn needed to be systemically stable in terms of blood pressure and pulse but full diagnosis and correction of the underlying medical condition was not something that was possible in the available time frame.

[385] The alternative was to do nothing and make a treatment-limiting decision. In Miss Brown's opinion, there was no realistic chance of Kathryn surviving without surgery. With surgery the chances were small. Given the overall clinical scenario, the risks were high: she estimated the risk of death at about 80%. Notwithstanding that, Miss Brown did not believe that a treatment-limiting decision was reasonable in a child who had been obeying commands perhaps two or three hours before.

[386] Miss Brown's decision to proceed to surgery was reviewed by the experts. From the neurosurgical perspective, Mr Mallucci and Miss Myles supported her decision and felt that it was reasonable. They agreed with Miss Brown's assessment of the clinical situation.

[387] First, they endorsed the view that surgery was necessary and that without it, Kathryn would have died. There was, in Mr Mallucci's view, no other option: there was nothing to lose as the alternative of doing nothing meant certain death. Miss Myles agreed that, without surgery, death was inevitable. There was a chance of survival, however small, by operating to remove the clot where there was a possible pupil reacting and a cough reflex at that time. She believed that the majority of neurosurgeons would have operated in these circumstances.

[388] As she explained, there comes a point of almost no return where, unless the neurosurgeons do something, the patient will die. Put bluntly, the haematologist cannot treat somebody with acute leukaemia if the patient is dead from raised intracranial pressure. Some very hard and pragmatic decisions had to be made in these situations. Once pupils start to dilate and the patient becomes bradycardic and hypertensive, at that stage neurosurgery has to be the driving force. Getting out of that situation is very difficult. As Mannitol stops working, the neurosurgeon is running out of options to reduce the pressure and at that point there really is no choice: you either operate or let the patient die. It would, she thought, be a very, very difficult decision to allow a child to die in front of your eyes knowing that there was even a small chance that you might improve matters. For that reason, Miss Myles could not believe that anyone would have denied Kathryn that chance and not operated.

[389] Mr Mallucci adopted a similar approach. He agreed that once pupils were fixed and dilated, there was very little time and absolutely nothing to lose. However, in his opinion, the chances of success were remote. He estimated that neurosurgery was 99.5%

unlikely to have any effect. That was because he believed that irretrievable brain injury had already occurred. Thus the effect on the eventual outcome of anything that happened after Kathryn's arrival at the Institute was negligible. Mr Mallucci concluded that neurosurgery was actually irrelevant after 3am. However, that was not to say that he would not have operated because, as he put it, "you're always hoping for a miracle, particularly in a child".

[390] The experts agreed with Miss Brown that the situation was one of extreme urgency and that time was absolutely of the essence. Minutes counted. This was an emergency situation and surgery required to be carried out without delay. There was no time to bring Kathryn's clotting within normal parameters and therefore a compromise had to be made.

[391] In that connection, they concluded that it was reasonable to have taken Kathryn to theatre before the coagulopathy had been fully addressed. Their estimate of the platelet level that could reasonably be anticipated reflected that of Miss Brown. With platelets at 69 and in the context of giving more platelets on the way to theatre, Mr Malluccci thought that the decision was "just about reasonable". Miss Myles examined the timescales and procedures involved in some detail. She agreed that, having regard to the first platelet increment, it could confidently be assumed that a second transfusion would raise the level to well above 100, being the target level for surgery. She explained that platelets and FFP could be transfused at the beginning of the operation when there was much time spent preparing before knife was put to skin. The second pool of platelets was commenced in the ward at 04:00 hours and Kathryn was taken to theatre at 04:20

hours. Furthermore, Miss Myles considered that it would reasonably have been expected that the mild PT clotting abnormality would have been reversed by the two units of FFP in the first instance. The important time for this was towards the end of the operation when haemostasis was being achieved, and afterwards in intensive care. Miss Myles also noted that the neither Miss Brown nor Mr Ahmad had considered the operative field to have been particularly bloody and that haemostasis had been achieved by the usual means. Nor was there a large amount of drainage from the wound post-operatively. All of these factors indicated that haemostasis had been adequate. Thus she concluded that the decision to take Kathryn to theatre when they did was the right one. Mr Mallucci agreed. The operation note showed that surgery was unremarkable and straightforward. There did not appear to have been any difficulty controlling the bleeding and haemostasis had been achieved.

[392] From the perspective of the haematologists, there was a profound concern that Kathryn had been taken to surgery at all, and without the involvement of a senior haematologist in the decision-making process. There was a concern, too, that diagnosis and treatment of Kathryn's underlying condition seemed to have been ignored during this time, the entire focus seemed to have been on the neurosurgical dimension, at the expense of the haematological side.

[393] Professor Hunt's view was that a haematologist would hesitate to send a patient with APL to neurosurgery because the haemostatic defect is such that medical management needed to be considered first. Although she appreciated the position of the neurosurgeons in this case, she argued that, as a general rule, craniotomy with an on-

going coagulopathy was contra-indicated as such patients would continue to bleed intracerebrally leading to death. This view was echoed by Professor Yin. In the acute stages, he believed that there was absolutely no place for surgery.

[394] Professor Yin also believed that had a senior haematologist been involved things would have been dealt with very differently and he would have been surprised if the surgery were allowed to go ahead. He believed that Kathryn would have been treated as a medical case and that the neurosurgeons would have "backed out". Mr Mallucci's view did not accord with that. He thought that even if there had been discussion with the haematologists, Kathryn would still have been going to surgery with her clotting problems.

[395] Professor Yin was adamant that the treatment of intracerebral haemorrhage in APL was purely medical. In principle, Mr Mallucci agreed with him. He did not think that neurosurgery had a major role in this particular malignancy. It seemed to him that where there was a haemorrhage associated with this condition, the outcome would be determined by the haematological management, not neurosurgery.

[396] From a haematological perspective, Professor Yin was firmly of the view that Kathryn should not have been considered for neurosurgery at all and that the craniotomy should not have taken place. Had the neurosurgeons been more proactive in questioning the haematologists and had they been told that the underlying condition was leukaemia, it was Professor Yin's contention that they would not have operated. However, he believed that the neurosurgeons were trying to be helpful, albeit

misguidedly. In any case, at the time they operated, it was too late. Accordingly, he did not think that it would have mattered, echoing the conclusion reached by Mr Mallucci.

[397] The issue arose of whether a repeat scan should have been done prior to surgery in light of the deterioration in Kathryn's condition. Some neurosurgeons would have ordered a further scan, others would not. It was a grey area. Both Mr Mallucci and Miss Myles said would have instructed a further scan. However, it was appreciated that this might have involved calling in the duty radiologist from home and warming up the scan. This would take time and might have caused unacceptable delay given the urgency and the fact that Kathryn's life was in danger.

Submissions

[398] The Crown's position was that surgery had not contributed to Kathryn's death and that the decision to operate could not be criticised. However, it was submitted that the neurosurgical team ought to have contacted the haematology department for advice prior to carrying out surgery. No attempt had been made to ascertain the underlying cause of her coagulopathy and it was submitted that there had been ample time to do so in the time between Kathryn's transfer from the Victoria and surgery commencing. While accepting that it had no impact in the critical circumstances of Kathryn's case, it was considered appropriate in the public interest public interest that failure to take haematological advice was not good practice and should not be repeated. The procurator fiscal invited me to include a finding to that effect under section 6(1) (e).

[399] So, too, did Counsel for Ms Crawford and Dr Beattie himself. This was on the basis of the criticisms of the experts. Furthermore, it was Dr Beattie's contention that the decision to proceed to surgery in the absence of haematological input and correction of coagulopathy was a major factor in Kathryn's death.

[400] As to the expert evidence on the contra-indications of surgical intervention in patients with APL, Mr Khurana submitted that the expert view reflected the general approach in such cases but failed to take proper account of the critical urgency of Kathryn's situation.

Conclusions

[401] I was entirely satisfied that the decision to take Kathryn to theatre to evacuate the clot and to reduce the pressure in her brain was justified. It was made in desperate circumstances. Kathryn had critically raised ICP and had started to cone. She was facing imminent death. Not to have operated would have meant that Kathryn would have died. This was a high risk situation with chances of survival, even with surgery, were slim. There was, too, a risk of surviving in severely compromised neurological state.

[402] I did not accept Professor Yin's proposition that there was absolutely no place for neurosurgery in these circumstances. Neurosurgery had to be a legitimate, if last resort in circumstances such as Kathryn's where the only other option was to let her die.

[403] Kathryn had arrived at the Southern General having suffered a catastrophic deterioration during which her brain had started to herniate and the process of coning had started. A second CT scan on arrival at the Institute might have shown that the damage to her brain was considerably more extensive than before. It might have caused the surgeons to decide that surgery was, by that time, futile so that an informed treatment-limiting decision could have been made. We will never know.

[404] The lack of input from a senior haematologist was rightly the subject of much criticism by the expert witnesses. In such cases it was clear that what was required was a multi-disciplinary approach with joint input into decision-making. That depended on whether there was time to do so. I was not convinced that the involvement of haematology would have made any appreciable difference by the stage at which the decision was made to proceed with the craniotomy. I accepted that the urgency was such that if things had been delayed, it was very likely that Kathryn would have died. Minutes did indeed count. The same applied had surgery been delayed in order to normalise Kathryn's coagulopathy. There was simply no time for that. The operation proceeded. Kathryn was given platelets shortly before surgery (which might well have raised her count to within the 100 range) and various blood products were transfused during the operation.

[405] It is important to bear in mind the circumstances in which these decisions were actually being made. Urgency apart, there was no firm diagnosis of leukaemia, let alone the APL subtype. The differential diagnosis remained one of septicaemia and underlying haematological malignancy. Furthermore, Miss Brown was entitled to proceed on the

basis that there was haematology input – a haematologist had been consulted for advice and was actively involved in Kathryn's care at least to the extent of releasing blood products for surgery. The anaesthetist had been in contact at least twice. In the particular circumstances of this case, it seems to me that Miss Brown could be forgiven for thinking that the haematological aspect of Kathryn's care was being covered.

[406] Mr Ahmad's position was slightly different. Although Miss Brown accepted ultimate responsibility for the decisions made, Mr Ahmad was the surgeon who received Kathryn at the Institute and who had been in direct communication with doctors at the Victoria Infirmary. He was keen to emphasise that he had given instructions while Kathryn was still at the Victoria Infirmary that a senior paediatrician and a haematologist needed to be involved in her care. He seemed to expect that her underlying coagulopathy would have been dealt with at the Victoria and to have made certain assumptions in that regard. Once Kathryn had been transferred into his care, it might reasonably have been expected that he would have followed that through to confirm that his instructions had indeed been carried out and to have established which physicians were actively involved in Kathryn's care rather than simply assume that this had happened. Had he done so, it might have become obvious that there was no proper source of haematological advice, nor any involvement of a senior haematologist.

[407] Although Mr Mallucci said that he would have telephoned a haematologist before proceeding, Miss Myles doubted whether she would have done so given the time-critical situation. In any event, I accepted Mr Mallucci's analysis that even if a haematologist had been consulted, surgery would still have proceeded given the desperate situation.

At that point, the immediate threat to Kathryn's life was from the pressure within her skull from which she faced certain death.

[408] It was the Crown's position that there was no question of surgery having contributed to Kathryn's death. However, it was submitted earlier involvement in a less critical case might have made a difference to outcome and that the failure to do so was not good practice and should not be repeated. In the particular circumstances of Kathryn's case, I am not persuaded that it is appropriate to make a finding in terms of section 6(1) (e).

[409] I was satisfied that there was clear evidence in this Inquiry that neurosurgeons and haematologist alike agreed that surgery is normally contra-indicated in such cases. Surgery is usually postponed until such time as the coagulopathy has been corrected. Input from haematologists is important in these situations. The difference in Kathryn's case was that there was no time to do this. She would have died if surgery had been delayed. Accordingly, I do not see that there is any basis for including a general finding proposed by the Crown that this so-called failure to take haematological advice was not good practice and should not be repeated. It was evident that what was good practice was fully understood but the circumstances were such that it was impossible to achieve in Kathryn's case.

[410] Furthermore, the position in Kathryn's case was compounded by the fact that there *was* haematology input. The problem was that it was from a junior doctor who was not a haematologist. The lack of adequate and senior haematology involvement stems directly

from the failure of the on-call haematologist to involve his consultant rather than any failure on the part of the neurosurgeons confronted with a critical the emergency situation.

[411] I accept that had Dr Morrison been contacted, she would undoubtedly have been in contact with the neurosurgeons and able to offer advice. However, I was not convinced that it would have made any difference to the outcome, although clearly it would have been better to have had a multidisciplinary approach and direct senior haematology input.

[412] Kathryn's pupils were fixed and dilated and the evidence was that the chances of recovery from that state were vanishingly small. I accepted Mr. Mallucci's conclusion that, in all the circumstances, her condition was irretrievable after she arrived at the Southern General. *Therefore surgery and her post-operative care were essentially irrelevant.* It appeared that only if physicians had been able to prevent her deterioration at 2am that her death might have been avoided. Thus the real focus of the inquiry settled on her treatment at the Victoria Infirmary prior to 2am.

PART 7 THE CRITICAL PERIOD AT THE VICTORIA INFIRMARY

[413] Several aspects of Kathryn's care and treatment while at the Victoria required to be examined in order to establish whether there were any reasonable precautions whereby Kathryn's death might have been avoided or whether there were any defects in the systems of working which contributed to her death. These included the arrangements in place to obtain an urgent blood film; the operation of the haematology on-call system; the actions of the on-call haematologist; the timing of the diagnosis; the nature and adequacy of the blood support provided; and, ultimately whether the timescales were such that Kathryn's deterioration shortly before 02:00 hours could have been prevented. That was the crucial question.

The out-of-hours laboratory system

[414] The system in place in 2004 whereby a markedly reduced service operated out of normal working hours has already been described at paragraphs [274]-[287]. Essentially, the only tests offered routinely were full blood counts, coagulation screen and cross-matching for transfusions. The full terms standard operating procedure (SOP) were not applied during these hours. In particular, immediate blood films were not produced automatically. If a film were required urgently it had to be specifically instructed.

[415] The Inquiry heard detailed evidence from a number of witnesses as to the operation of the laboratory. Several witnesses gave evidence as to the workings of the laboratories in the Victoria Infirmary and the Southern General Hospital. Two senior managers, Mr John Rae and Mr Tom Moffat, gave evidence about the workings of the laboratory and the procedures in place, currently and in 2004.

[416] Mr Rae was the manager in charge of the haematology laboratory in 2004. Previously, he had been Senior Chief Biomedical Scientist at the Southern General Hospital before moving to the same post in the Victoria Infirmary. He took on this post at a time when there were plans to merge both Victoria Infirmary and Southern General Hospital Departments into one. He was in technical charge of the haematology laboratories for blood testing, coagulation screens and blood transfusions. In 2004 he had almost 40 years experience as a biomedical scientist. Mr Rae was an excellent witness. His vast experience both as a biomedical scientist and in senior management positions made him a most valuable source of information. His presentation was professional, authoritative and confident. He gave his evidence in a straightforward and clear manner. He gave no impression of being in any way defensive or overly cautious about his answers. I placed considerable reliance on his testimony.

[417] Mr Moffat was his successor and is the current Technical Services Manager. He qualified as a biomedical scientist in 1990. He was Chief Biomedical Scientist in Monklands Hospital for NHS Lanarkshire and then the Laboratory Manager at the Royal Hospital for Sick Children in Glasgow before taking up his present post. Most of Mr Moffat's evidence related to his efforts in producing a record of laboratory events in order to assist the Inquiry. I found him to be an honest witness.

[418] Miss Claire McKie is the current Quality Services Manager and Point of Care Testing Manager for the Glasgow South Sector Hospitals which includes the Victoria Infirmary and the Southern General. She held that post in 2004. She, too, has a

background in biomedical sciences and has a Fellowship from the Institute of Biomedical Science. She has specialised in haematology and blood transfusion. Her responsibilities included ensuring that the operating procedures for the various departments met with national standards. Miss McKie was a highly qualified and experienced scientist and manager. I had no hesitation in accepting her evidence: as the current manager she clearly had a good overview of systems and procedures in place now and in 2004. Like the other managers, she had a firm grounding in practical biomedical work.

[419] Mr Alex McLauchlan gave evidence of those matters solely from the perspective of the biomedical scientist. He was the BMS on duty in the Victoria Infirmary on the night of Kathryn's admission. (The on-call biomedical scientist at the Southern General that night was not a witness to the Inquiry.) Mr McLauchlan, too, was a very experienced BMS having qualified in 1979. He has worked at the Victoria Infirmary since his training days and has always worked in the haematology discipline. As a witness, he had a tendency to answer questions very literally and was somewhat pedantic but I had no concerns about his honesty or as to the overall accuracy of his evidence.

[420] Mr Anthony Docherty, himself an experienced biomedical scientist, is currently the Head of the Components Laboratory with the Scottish National Blood Transfusion Service based at Gartnavel Hospital in Glasgow. He described the workings of the Laboratory which manufactures all the blood components and distributes them for stock and emergency supply to the various hospitals in Glasgow and Lanarkshire. He has held that post for nine years and was in post in 2004. Mr Docherty was very straightforward witness who was keen to assist the court.

The evidence

[421] The reduced service had been in place for as long as anyone could remember. No-one could quite recall when or why it had been introduced. Mr McLauchlan said it was "just custom and practice" and that seemed to be the general view. In 2004, the on-call BMS duty was operated on a voluntary basis and did not form part of the core duties of the scientist. The on-call BMS was on duty for a twenty-four hour period and was, therefore, not expected to work a full shift during those hours. Rather than work a full shift of duty during these hours, if things were quiet, Mr Rae explained that the BMS was allowed to relax, watch television, make something to eat and even sleep. He did not simply fill all twenty-four hours by carrying on some of the routine work for the day shift. That was not to say that a BMS could not be extremely busy and fairly fully occupied should there be a major emergency or a high volume of requests but, at quiet times, the BMS was not expected or required to do any further work.

[422] Mr Rae, who was the manager in 2004, gave a slightly different description of the duties of a BMS which suggested a more pro-active role in the out-of-hours situation. As far as blood films were concerned, he pointed out that the preparation of an urgent blood film was a very rare occurrence. He himself could count on the fingers of two hands the number of times he had been asked to prepare one in 40 years. They were so rarely requested that it could not be said to give rise to an issue of resources. Such sporadic requests could readily be accommodated.

[423] Mr Rae explained that, while there was no obligation on him to do so, the BMS would phone the haematologist on call and ask to do the blood film if he thought it was so important that a film should be made. There were, he said, several sources of information which might alert the biomedical scientist to the need for a blood film while on call: the initial telephone conversation requesting the tests; the information contained on the request form; the results produced by the analyser; and discussion in any subsequent phone call to the requesting doctor. In such circumstances it was clear that contact would have to be made with the on call haematologist. This could be done directly by the BMS. Alternatively, the BMS might ask the requesting doctor if he wanted to speak to the on call haematologist.

[424] Mr Rae went on to explain that there were a number of situations in which an urgent blood film would be required, at night as during the day. The type of circumstances in which an urgent blood film would be prepared overnight would be where there was a suspected new leukaemia (that, he said, was an obvious one) or a patient with suspected malaria parasites. There was no definitive list as the circumstances depended on the medical condition of the patient. The fact that the patient was in hospital for the first time (a new patient) and the seriousness of the potential diagnosis would be two factors that would alert the BMS to the need to do a blood film. The purpose of preparing an urgent blood film out of hours was to assist with and, where possible, confirm medical diagnosis.

[425] Mr Rae considered that the abnormal results themselves might also alert the BMS. No decision could be made on the basis of results alone so the BMS would require

further information. Accordingly, Mr Rae would expect the BMS to discuss the result with the person requesting the test. That final conversation was important – sometimes there was an explanation for the result (for example, a low platelet count as a result of severe traumatic bleeding). The requesting doctor might also wish to take the matter further and speak to the on-call haematologist. Depending on the response from the requesting doctor – who might be junior – the on call BMS might contact the on-call haematologist directly.

[426] According to Mr Rae, where there was a result of 13, the BMS would have wanted to ensure that it was taken further to see that the platelet count was being attended to. It was a judgment call in individual cases. The important thing was to pass it on to a member of the haematology medical staff. You would, he said, have to be reasonably sure that something was going to be done about it – a platelet count of 13 was an extremely low result. In these circumstances, someone must speak to the haematologist. Moreover, if this were or had the potential to be a surgical matter then someone had to bring it to the attention of the haematologist who was the only person who could authorise platelets. "At 13,000 you couldn't walk away without doing something. If no one was doing anything I would consider that the BMS would have to call the haematologist."

[427] In a situation where there was a suspected new leukaemia, Mr Rae would be asking the referring doctor: "Are you speaking to the haematologist or am I?" He would have phoned straight to the haematologist because of the platelet count being so low and because it was in keeping with a new leukaemia. This would be to warn the

haematology department at an early stage because subsequently they would expect to get a phone call from the medical staff. "It's an early warning – a heads up that this is coming their way and others would be phoning them to discuss."

[428] This approach – from the person then in charge of the laboratory - seemed in stark contrast to the understanding of others involved with the laboratory. Miss McKie's position was simply that no blood film would be made unless specifically requested by the on-call haematologist. She did not suggest that the BMS would have a duty to take the initiative on seeing abnormal or worrying results. Her understanding was that only a limited, "very basic" service would be operated at night.

[429] Mr McLauchlan shared that view. As he saw it, his job was simply to do the tests and relay the results to the requesting doctor or department. His duty went no further. There was no question of him taking the initiative himself or having any detailed discussion with the physician who had requested the tests (or whoever answered the phone). He would not have given any input on what the physician should do next. That was not part of the job of the BMS. It was Mr McLauchlan's position that it was the physician who knew the clinical circumstances and hence would be better able to interpret the results. Likewise, it was the physician who would telephone the on-call haematologist as he had the clinical details. Mr McLauchlan saw his job as only providing the results. If the physician wanted a blood film, then he could ask for one and it would have been done.

[430] Although there was some variation among the witnesses as to extent of the role of the biomedical scientist in 2004, there was general agreement that diagnosis was no part of the remit of the BMS. That was the responsibility of a medically qualified haematologist. Mr Rae explained that the biomedical scientists in the Victoria Infirmary were trained to read and interpret blood films. In routine cases where they were confident to do so, they could report the films. Anything abnormal or causing concern (Kathryn's results would have fallen into this category) would be passed to the one of the haematologists to analyse and report. While a BMS in the Victoria Infirmary was trained to recognise an abnormal blood film, he was not qualified to make a diagnosis.
The system was different in the Southern General Hospital where the role of the BMS was more restricted. There biomedical scientists were not trained to read blood films. They did not look at blood films, all of which required to be assessed by a member of the medical staff.

[431] It was generally accepted that an experienced BMS would be able to recognise leukaemic cells and therefore a leukaemia - perhaps a myeloid leukaemia. Senior scientists might recognise specific subtypes such as APL but many would not. None of the haematologists would have expected otherwise. Mr McLauchlan himself felt confident that he would be able to recognise leukaemic blast cells but not specifically APL. Therefore, no diagnosis of leukaemia (or provisional diagnosis) could have been made by a BMS in either hospital. That would have required both a blood film and input from a haematologist.

Urgent blood film out-of-hours

[432] It was clear that although the SOP was not followed at night, in 2004 urgent blood films could and would have been prepared out of hours had they been specifically requested. The system was described as a request-led service. However, it was not clear from the evidence exactly how this worked. Nor was it entirely clear whose responsibility it was to give the necessary instruction.

[433] In the course of their evidence, some members of the medical and haematology staff were questioned about their understanding of the out-of-hours system in 2004 with particular reference as to who was responsible for the instruction of a blood film. Likewise the laboratory staff were asked about their understanding of the position. There was a distinct lack of unanimity and the various answers demonstrated a degree of uncertainty and the absence of clear understanding of the arrangements in place at that time.

[434] Of the laboratory staff, Miss McKie was firmly of the view that an out-of-hours blood film could only be requested by the doctor on call for haematology. Mr Rae thought it could be requested by the haematologist who could have been contacted either by the doctor who originally requested the tests or by the BMS. Mr McLauchlan shared the view that it was for the doctor who had requested the initial blood tests to request any urgent film. As the biomedical scientist, it was his job to give the results. Thereafter, it was for the doctor in charge of the clinical situation to give instructions as to what further investigations should be carried out. He thought a blood film could be requested by any doctor, not necessarily the haematologist.

[435] Members of the medical staff were likewise unsure of the position. As Dr Weir was not familiar with exactly what a blood film would show and would not have been in a position to interpret it, he would not have requested one. He was not aware of the procedures whereby an out-of-hours film might be requested. Likewise, Dr Thomas was not familiar with blood films and not qualified to interpret one, had it been within her remit to order such a test as an A&E physician. The two consultant physicians who covered on-call for general medicine, Dr Vernon and Dr McAlpine, were asked about their understanding of the position. Dr Vernon did not know what the out-of-hours routine was in the laboratory at that time. According to Dr McAlpine haematology provided a blood film if they thought it was appropriate. Where results were very abnormal he believed that the technician (BMS) would report back and in partnership with the clinician would suggest what to do or that the clinician made contact with the haematologist for further management advice.

[436] Of the haematologists who were asked about this in evidence, Dr Morrison agreed with Miss McKie that an urgent blood film could be instructed only by the on call haematologist. While Dr Sharp, consultant haematologist at the Victoria, was aware that a blood film would be prepared the following morning unless there had been a specific instruction, he thought that normal practice would be that the doctor requiring the investigations (i.e. the doctor in charge of the clinical situation) would initiate the specific request. In Kathryn's case, he considered that the responsibility would have rested on the doctor in charge of her care at the time, either A&E or general medicine. This was not Dr Macdonald's view: his understanding was that the on-call haematologist would order a

blood film. It was *not* the responsibility of the referring doctor. If the referring doctor had said that a blood film should be looked at, then that would be considered by the haematologist. It was, he thought, an area where it was difficult to establish a foolproof system.

[437] Dr Bunemann's evidence on this subject was equivocal. On the one hand, he did not seem to think that it was particularly his responsibility as the doctor on call for haematology to order a blood film out of hours. He thought that Dr Weir would have been able to do so. However, in general terms he appreciated that either the requesting doctor or the haematologist could instruct a blood film out of hours. His evidence on this was difficult to follow. He could ask for a blood film to be prepared "if I would speak to a technician in the circumstances". When asked about the circumstances in which he would speak to a technician (the BMS) he replied "Well the technician sometimes would have phoned me overnight as the on-call to tell me about that result and tell me, maybe, about a blood film that's been done and I would maybe ask him to look at the blood films in the Victoria. In the Southern General I would have to look at it myself." This seemed to indicate a confused understanding of the position

Discussion

[438] Opinions about the out-of-hours system were sought from the various expert haematologists. All viewed the arrangements that were in place with concern and disapproval. Shortcomings were identified, both in connection with the lack of a clear instruction in place as to the circumstances in which an urgent blood film should be

made out-of-hours and the lack of a clear procedural pathway to identify how such a film should be instructed.

[439] None of the experts would have expected a junior doctor in either A&E or in general medicine to recognise the need to order a blood film. This was a special laboratory test within the remit of the haematologists. Professor Hunt did not consider that the general physician would have the requisite knowledge base to know when it was appropriate to order a film. Thus any system that relied upon a junior doctor who was not a haematologist having to telephone the on-call haematologist in order to ask whether a blood film should be prepared was one which she described as "too tenuous". That, she said, would mean having somebody who did not fully understand when a film was helpful making a decision as to whether or not to phone the haematologist to ask for one. She favoured the practice of an out-of-hours standing operating procedure whereby the onus was firmly upon the BMS to ensure that a film was made in such circumstances. There should, in her view, be set criteria out of hours so that when the laboratory technician telephones the abnormal results he says "this blood count merits a blood film". That way the decision-making is not tenuous but is actually made by the person who is vetting and issuing the results. The technician then proceeds to make and look at the blood film. Once the film is identified as highly abnormal, the BMS should telephone the on-call haematologist. As Professor Hunt saw it, the flaw in the system in 2004 was that the BMS was waiting for a clinician to say that a blood film was required.

[440] Professor Grimwade, did not consider the out-of-hours system in 2004 to have been appropriate because in certain clinical circumstances and within certain clinical

parameters, blood films would be expected as a matter of urgency. He used as an example someone suspected of having malaria. In such a case the physician would want to be able to look at the blood film straight away. There were a number of conditions where different emergency treatments would have been indicated and looking at the blood film would have helped discern the appropriate treatment. Circumstances that constitute an emergency by day cannot be treated differently at night: they are still emergencies. He was not necessarily in favour of standard procedures to cover every eventuality but he believed that it was a matter of common sense. Like Professor Hunt, he would have expected the on-call BMS to have seen highly abnormal results, made a blood film, and looked at it, or identified someone who could have interpreted it. "There is no point in actually having an out-of-hours service if you can't identify what is normal and what is abnormal; what is an emergency and what isn't an emergency." He considered that there was a good chance that an experienced BMS would recognise AML or even APL, but if they did not, they would be sufficiently concerned to bring it to the attention of the haematologist. He would have expected the BMS to communicate any very abnormal blood film to the requesting physician but also to get in touch with the haematology department to let them know that someone from the medical staff would be contacting them.

[441] This process was also approved by Dr Hanley. Like Professor Hunt, he agreed that the problem with the system in place in 2004 was that it relied upon a doctor – who was very often a junior doctor – to recognise the need for a blood film. In these circumstances, it was important that the skills of the biomedical scientist and the medical staff came together. In his experience , this was an area where junior doctors very much

relied on an experienced BMS to guide them and give advice on what to do next. If there were a rule which prevented the BMS for applying his skills and knowledge out of hours, then it made the diagnostic process less likely to work well.

[442] Professor Yin thought that the system was seriously flawed where it did not allow for grossly abnormal results to have been acted upon immediately.

Expert evidence

[443] Before proceeding further, it is necessary to address Mr Khurana's submission on behalf of the Board as to the position of the expert evidence concerning the workings of the laboratory and, in particular, the duties of the biomedical scientists. He cautioned me as to the extent upon which it was appropriate that I rely on it.

[444] First, he submitted that no expert biomedical scientist had been called before the Inquiry. While he took no issue with the ability of the expert haematologists to give evidence on the nature and treatment of leukaemia, he submitted that they were not qualified to give opinion evidence on matters relating to the biomedical scientist. In any event, they were not in a position to compare practices around the country and had limited experience themselves. Secondly, the reasons behind the 2004 arrangements had not been sufficiently explored with the haematology consultants and, in particular, with Dr Sharp as the clinical lead in both hospitals at the time. Nor had the experts been furnished with clear information on that. Thirdly, it had always been the Board's position that the arrangements were adequate and worked satisfactorily. None of the

haematologists had expressed any dissatisfaction and this was never explored with them in evidence.

[445] I, too, had some concerns about the ability of the expert haematologists to give evidence about the role of the biomedical scientist and about certain aspects of the laboratory system. Certainly no expert biomedical scientist was called as an expert witness. Although highly experienced and qualified, Mr Rae appeared as a witness to fact, in his capacity as the then-head of the haematology laboratories in Glasgow. It should be pointed out that no clear objection was raised as to the ability of the haematology experts to comment on the laboratory system although there was an objection on that ground to a question which was not then insisted upon. Nor was I asked to hear that evidence under reservation. Thus the evidence is before the court in its entirety. However, the court has an inherent responsibility to be satisfied that any expert witness is suitably qualified to give opinion evidence and that the witness does not stray outwith his or her respective field of skill.

[446] In fairness to them, they seemed careful not to overstep their remit. Professor Grimwade, for example, made it clear that the workings of the CPA and quality control measures were outwith his area of expertise and so he could not comment on matters relating to laboratory assessment. Nor did he know anything about the particular workings of the Glasgow laboratories and stressed that he had "absolutely no idea" about the operation of laboratories elsewhere.

[447] Professor Yin, too, acknowledged that he only had experience of what happened in his own hospital and that he had no knowledge of, nor had he enquired into, what happened elsewhere. In particular, he had no experience of a system which operated on a 24 hour service as opposed to a full shift system.

[448] Many of the comments of the expert witnesses related to their own earlier experiences when, as junior trainees, they had been required to work in the laboratories. In no sense could this be regarded as expert testimony. In fairness, it was neither presented nor offered as such. I treated that evidence in the spirit in which I believe it was given - simply as anecdotal comment.

[449] I noted and have taken account of Mr Khurana's point that there was no input from an expert biomedical scientist. However, I was satisfied that the haematology experts were able to provide their opinion to the court *qua* haematologists on the service required in order to pursue emergency investigations for diagnostic purposes. Likewise there were matters upon which I required expert input in order to reach my own conclusions, for example, as to whether junior doctors outwith haematology would be expected to appreciate the significance of blood films. To that extent, their evidence was properly admissible. Beyond that, I considered much of what I heard in relation to the workings of the laboratory were matters of common sense in respect of which expert input was neither required nor admissible.

[450] I should add that in considering their evidence I was conscious of the fact that the experts worked in specialist leukaemic centres. For that reason, I had some concern that the expectation of services within specialist leukaemic centres of excellence might be higher in comparison with those in a general hospital - albeit a large teaching hospital – which was not a regional leukaemic centre. Similarly, as far as their expectation of the duties and experience of the BMS was concerned, I wondered whether the bar might have been set artificially high. I was also concerned that the experts had been furnished with incomplete or, at times, inaccurate information as to the details of the workings of the out-of-hours system in 2004. For all those reasons, I considered their evidence on this part of the Inquiry with some care.

Submissions

[451] The Crown submitted that there was a lack of an adequate system in relation to out-of-hours blood films. It was not acceptable in a large teaching hospital accepting emergencies and acute illness that the system of work did not mandate the preparation, staining and examination of blood films out of hours on receipt of blood results within certain parameters. The system as it operated relied too much on the ability of a "junior ward medic" to recognise the significance of and need for a blood film. In the Crown's submission, the active involvement of the BMS was of central importance. Not only would that assist junior doctors but it might also assist less experienced haematologists as suggested by Dr Hanley. It was the Crown's submission that Dr Bunemann might have taken a different approach had the BMS contacted him and advised him of the abnormal blood film. Thus there was criticism of a system which cut out the BMS from

the process and she invited me to conclude that the failure of Greater Glasgow and Clyde Health Board to make adequate provision for the mandatory making, staining and examination of blood films, out of hours, in the Victoria Infirmary haematology laboratory was unacceptable. She invited me to include such a finding under section 6(1)(e), albeit that the position had now been rectified.

[452] In the public interest, Miss Adair also invited me to include this chapter of evidence under subsection (e) insofar as it related to Dr Bunemann's failure to contact a consultant.

[453] Mr Lamont, on behalf of Kathryn's mother also invited me to determine that the system in place in 2004 was lacking, as did Dr Beattie himself. There was no prescribed system for the escalation of abnormal blood results. Mr Lamont made particular reference to remarks made by Mr Moffat about the lack of a clear system to escalate results to a haematologist. Mr Moffat had explained that a perceived lack of a clear system for such escalation had formed part of the reason for the change to the SOP, although this did not appear to be specifically in respect of out-of-hours services. Both Mr Lamont and Dr Beattie invited me to make a finding in terms of section 6(1)(c) and (d) in respect of the restricted laboratory practice: that it would have been a reasonable precaution to have stained and examined a blood film at 2312 hours on the 20 June 2004. Further it would have been a reasonable precaution thereafter to obtain the input of a consultant haematologist as a matter of urgency. Thereafter it was submitted that it would have been a reasonable precaution to arrange for urgent blood support for Kathryn between the period 2312 hours on 20 June and 0200 hours on the 21st. Further it

was submitted on behalf of Ms Crawford that if the court accepted the submission in respect of 6(1)(c) then any defect which delayed the provision of blood support, on the balance of probabilities, contributed to the death. Accordingly, these two defects in the system of working qualified for a finding under subsection (d): that blood films were not routinely examined out of hours and that there was no proper procedure for the escalation of blood results. Thereafter, if the court were satisfied that the lack of a blood film and the lack of consultant input delayed the provision of blood support to Kathryn, then they constituted defects in the system of work which contributed to her death and merited a finding in terms of section 6(1)(d).

[454] Dr Beattie submitted that there were a number of reasonable precautions – he outlined seven - arising from the workings of the laboratory. In brief, he, too, focused on the need for a standing operating procedure to ensure that blood films were made irrespective of timing, the need to have prepared a blood film and to have had it brought to the attention of the on call haematologist and the need to ensure consultant involvement. He included the request-led system for blood films in terms of his submission under (d) as a defect in the system of working.

[455] In her submissions on behalf of Dr Bunemann, Miss Keane was critical of what she described as the "lax management" and the confusion regarding whose responsibility it was to instruct a blood film but she did not consider that it was appropriate that she invite the court to make any finding in that connection.

Conclusions

[456] It was frequently said in the course of the evidence that the relevant SOP did not apply out of hours. Strictly speaking, that was not true. The SOP for the making of blood films *was* recognised and applied out of hours insofar as any results that came within the parameters prescribed in the SOP were acted upon and a blood film was identified as required. The difference was that only the first stage in the preparation of the film was carried out, namely the smearing and preservation of the blood on a slide. The blood film itself was not prepared until the beginning of the next available full shift. Accordingly, the accurate position was that blood films were prepared and examined in all cases set out in the SOP but that there was a time delay out of hours. It was not a question of any of these being missed simply because the initial tests were ordered overnight or at the weekend. In the public interest, it is important to highlight that fact.

[457] Similarly, it was misleading to describe the system as one which did not cater for out-of-hours emergencies. Urgent blood films could and would have been prepared and examined if specifically instructed. So, there was no suggestion of the absurd situation whereby only emergencies which occurred during the day were acted upon as some of the experts seemed to believe.

[458] However, in order for that system to work effectively and reliably, there had to be a means of identifying potential haematological and other emergencies where a blood film was required urgently. I was satisfied that the system in place in 2004 was as described by Miss McKie, Dr Morrison and Dr Macdonald – that an out-of-hours blood film had to

be instructed, or at least ratified, by the haematologist. This made sense: first, junior doctors did not necessarily appreciate the significance of a blood film and were certainly not qualified to interpret it in order to diagnose a haematological condition; secondly, only a haematologist could be relied upon to know the importance of a blood film; and thirdly, it required to be interpreted by a haematologist so would have meant the on-call haematologist coming into the hospital to look at it. However, it depended on someone having contacted the haematologist in the first place.

[459] How, then, was the haematologist to find out about the patient? During normal hours, the BMS fulfilled this role, bringing abnormal or worrying results directly to the attention of a senior haematologist. However, at night, in terms of the system in place in 2004, the BMS did not automatically contact the haematologist. He was entitled to adopt a purely passive role and wait for any further instructions and therefore Mr McLauchlan could not be criticised for doing just that. However, a central player having been removed from the equation, it was not clear on whom the responsibility then lay to contact the haematologist.

[460] Leaving the responsibility to the referring (often junior) physician was an unreliable method. I accepted the evidence that a doctor in such a position could not be expected to appreciate the need for a blood film. For the sake of completeness, I also accepted that a blood film was not the type of initial investigation that would ordinarily have been instructed by the doctor in accident and emergency or a junior general physician. Accordingly, I have concluded that the initial responsibility for identifying the

need for and instructing an urgent blood film out of hours was not that of Dr Thomas or Dr Weir.

[461] However, the problem was that at that stage the duty haematologist, on call at home - possibly asleep in bed - would not know of the patient's existence. The only other person who was aware of the patient, knew of the abnormal results and could have identified the need for an urgent blood film was the BMS. I note here that the BMS would always telephone abnormal results and would therefore be in direct contact with the referring physician (or the relevant department) and, in the event of platelets being authorised, would have direct telephone contact with the on-call haematologist. This happened in respect of Kathryn's blood results. As Mr Rae said, *somebody* had to make sure that the need for a film was identified and acted upon. It was far from clear who would do that out of hours when it was accepted that it was not the role of the BMS. This lack of clarity gave rise to confusion and uncertainty.

[462] The BMS was the obvious person to co-ordinate things. The BMS had a pivotal role in the process. I was satisfied that the removal of the BMS from the equation created a lacuna in the pathway to a blood film and haematological input overnight or at weekends. That was a clear flaw in the procedure. There was much sense in Dr Hanley's remarks about the importance of informative input from an experienced BMS particularly where junior or less experienced doctors were involved. This was all the more important if, as in the Victoria and Southern General hospitals, inexperienced junior doctors were entrusted with on-call responsibility.

[463] As I have concluded later, these shortcomings do not amount to reasonable precautions or defects in the system of working such as would merit their inclusion under subsections (c) or (d). However, I am satisfied that they are relevant to the circumstances of Kathryn's death and raise matters of public interest which merit a finding under section 6(1)(e).

[464] In the particular circumstances of Kathryn's death, it has to be stressed that any flaws inherent in the 2004 out-of-hours arrangements were, in the event, of limited relevance. Notwithstanding the identified shortcomings, Kathryn's results *were* brought to the attention of the on-call haematologist at a relatively early stage. So the need to ensure the involvement of the haematologist was in fact met.

[465] It was universally agreed that urgent advice from a haematologist was required in relation to Kathryn's presenting symptoms. Here was a young girl with grossly deranged blood results which included a severe thrombocytopaenia (low platelets) and a very high white cell count. An intracerebral bleed was suspected. Her differential diagnosis included the possibility that she was suffering from a serious underlying haematological malignancy (which included leukaemia). Urgent consultation with the on-call haematologist was required first to confirm or exclude the haematological part of the differential diagnosis; secondly to authorise the release of blood products, including platelets; and lastly for general advice on treatment and management of the patient. Dr Weir recognised that and made early contact with the on-call haematologist, Dr Bunemann.

[466] That should have been enough to trigger the instruction to prepare the all-important blood film. Sadly, in Kathryn's case, this did not happen. Accordingly, the failure of the on-call haematologist to instruct a blood film out of hours is the critical aspect which impinged on the timing of the diagnosis and the treatment Kathryn received. Why no blood film was instructed by the doctor on call for haematology was a major focus of the Inquiry. In addition to the out-of-hours cover provided by the haematology laboratories, the on-call arrangements as they applied to the haematology physicians came under scrutiny.

On-call haematology arrangements

[467] Dr Morrison, Dr Sharp and Dr Macdonald described the arrangements that were in place in 2004 for the on-call haematology rota. The four consultants – two from the Victoria Infirmary and two from the Southern General - shared the on-call work. The on-call rota was also populated by the haematology trainees, all of whom were specialist registrars or SPRs. The haematology traineeship was a five year course. Those who entered the traineeship were experienced doctors who would normally have done their core medical training which would have involved exposure to a variety of different medical specialties. During this time, they would have been expected to have studied for their Membership of the Royal College of Physicians and many would already have some sort of postgraduate qualification. Although not essential, it was desirable that those entering the programme had previous haematology experience. Accordingly, although the experience of a trainee varied from year 1 to year 5, a year 1 trainee was already an experienced physician, usually with relevant experience.

[468] Dr Morrison explained that there was a further grade of junior doctor attached to the haematology department, namely, the SHO3. They were described by Dr Sharp as junior doctors at least within their third year post-registration. These were young doctors who were just interested in finding out whether they might be interested in pursuing a career in haematology. Dr Sharp described it as an important job in terms of career development which allowed people to find out in more detail what the various sub-specialties had to offer.

[469] There was no formal training for these doctors although they were encouraged to attend the weekly teaching meetings (tutorials and workshops) designed for the trainees. Their attendance was not compulsory but every effort was made to ensure that the SHO3 was freed from other duties so that they could attend these sessions. Their duties were largely clinical: looking after patients on the wards and working in the outpatient clinics. During the day, they would work under the supervision of senior haematology staff, usually the consultants. The SHO3s would not be expected to spend as much time in the laboratory as the formal trainees. They would discuss the interpretation of results with the consultant and when it was appropriate to look at a blood film. However, the SHO3s would not be expected to be competent in examining blood films although they should have an idea of what was normal and abnormal and understand when to ask for input.

[470] Dr Morrison explained that after a period of observation and following discussion with other haematologists, the SHO3s would take calls during the day and be expected to discuss these with senior members of staff before making any decisions. Once an individual SHO3 had spent some time in the department and after an assessment of his

capability, provided the consultants were happy with his performance and decision-making skills, the junior doctor would join the on- call rota. Typically, it would be about four weeks before the SHO3 would be placed on the rota. Dr Sharp confirmed these arrangements. According to him, decisions as to suitability for on-call duties were made on an individual basis. Much depended on the maturity and previous experience of that individual. There was an informal discussion among all four consultants. By and large, if someone had come from a background of general internal medicine and had done a little haematology but not a great deal, he would still be expected to go on to an on-call rota within one to two months of starting the job. He agreed that most of them would undertake on-call duty within a month of starting the job.

[471] The consultants explained that the SHO3 post no longer exists. It was disbanded in all departments and specialities following a national initiative, principally as a result of changes to junior doctors' hours and their career structures. Now the haematology on-call rota consists only of consultants and specialist registrars. First on-call across the city is now a specialist trainee.

[472] Before SHO3s began on-call duties, one or other of the consultants would spend some time with them discussing what types of calls they might have to deal with and a number of different scenarios which might present. There were a number of different situations but Dr Sharp explained that one of the more common scenarios involved being asked for advice in connection with the appropriate use of blood and blood products in cases of bleeding. Dr Sharp would also rehearse with the new trainees (including the SHO3s) what he considered to be the pertinent questions that should be

asked of any doctor phoning for advice: these included the background of the case; past medical history; the symptoms and signs on examination. Such questions were designed to set things into context in an effort to see whether there might be an obvious explanation for the blood count numbers or whether there was something that needed further investigation.

[473] Dr Sharp also explained the type of situations which the on-call SHO3 would be expected to deal with. These included the authorisation of the release of platelets. Much of this was straightforward. Apparently in addition to obstetric cases and trauma cases, those in most frequent need of platelets in the west of Scotland were "alcoholic bleeders". Dr Morrison added that many cases would involve anticoagulation problems where patients were on blood thinners such as warfarin or heparin. The on-call haematologist was also expected to deal with enquiries about in-patients in the wards.

[474] Dr Morrison stressed that there was always a senior member of staff available for advice at any time. A consultant was on call for advice out of hours and junior staff were told quite clearly to contact senior staff early on rather than leaving them to find out about things later.

Dr Bunemann

[475] Dr Bunemann held the German State Exam and was registered as a medical practitioner in the UK in October 2003. Accordingly, in June 2004, he had practised for some 8 months post-registration. During that time he had undertaken locum work in various hospitals. In March 2004 he joined the haematology Department as an SHO to

see if he were interested in pursuing a career in that specialty. Accordingly, as at 2004, Dr Bunemann had no experience in haematology beyond his theoretical training at University. He was not a haematology trainee. He was *not* a haematologist. Dr Morrison agreed that it was more accurate to describe Dr Bunemann as an SHO working in the Haematology Department as opposed to a haematologist.

[476] Although evidence was heard about the post of SHO3, this was somewhat misleading as it would appear from his experience that Dr Bunemann was only 8 months into his post-registration period. Therefore he was not an SHO3 but merely an SHO at the time. This gave rise to some confusion. Not all of the consultants seemed to have appreciated or remembered that Dr Bunemann was not actually an SHO3. Indeed, examination of the witnesses proceeded on that basis that he was. None of the consultants described this more junior post at all. Exactly where an SHO a few months post registration fitted into the scheme of things was not directly discussed. Nor was any evidence led as to how, with such limited experience, Dr Bunemann had become attached to the haematology department. However, it appears that Dr Bunemann acted and was treated in much the same way as an SHO3. He told the court that he had attended the various Wednesday afternoon training sessions along with the trainees and it appeared that his training, like that of his SHO3 colleagues, was largely ward based and in out-patient clinics, working directly under supervision most of the time.

[477] Dr Bunemann commenced on call duties after his first month in the haematology department. Like his SHO3 colleagues, Dr Bunemann would have had sessions with the consultants in order to explain the types of scenarios that would arise during a typical

on-call period and it would undoubtedly have been stressed to him that if he were in any doubt, he should contact the on-call consultant. None of the consultants had any concerns about his ability to do so. Dr Bunemann was described by Dr Sharp as reasonably diligent and it was Dr Sharp's recollection that Dr Bunemann tended to contact the consultants during his on call duties more than the others on the rota.

[478] Dr Bunemann was on call for haematology when Dr Weir telephoned him about Kathryn. Before considering the quality of the advice given, the disputed circumstances of the conversation requires further examination.

The conversation between Dr Weir and Dr Bunemann

[479] The evidence about this has already been noted above in paragraphs [69]-[79]. This evidence had to be examined with some care. Dr Bunemann was in a difficult situation as he had no memory of the conversation at all. That is hardly surprising given the passage of so much time and the fact that apparently Dr Bunemann was not aware of any on-going inquiries into the circumstances of Kathryn's death until 2009. Again, like so much evidence in this Inquiry, his evidence reflected what he thought he would have done in such circumstances. Likewise, he deduced what he must have been told (or not told) from the actions he took (or did not take). The absence of any direct recollection obviously affected the weight that I am able to attach to his evidence. It is worth observing that it was difficult to assess whether Dr Bunemann's evidence had been influenced by the additional experience that he had gained since 2004 rather than reflecting his knowledge and understanding of haematological emergencies as at 2004. In all he spent over two years in the haematology department. I have to say that in general,

I found much of Dr Bunemann's evidence difficult to follow and had the impression that, as a native German-speaker, it stemmed from his excessive use of the subjunctive.

[480] Dr Weir had some recollection of the telephone call but, after almost eight years, his memory of detail was understandably hazy. However, I am satisfied that Dr Weir provided a truthful account to the best of his recollection and that his memory of the gist of the conversation was essentially accurate. Out of all the medical and nursing witnesses who gave evidence at this Inquiry, it was Dr Weir who said that he had often thought about Kathryn in the years since her death. I found this to be a telling comment which fitted in with his evidence that, at the time, he was "incredibly worried" about Kathryn. Her case clearly made an impact on him. I did not take anything sinister from that but simply that this had been a complex and anxious case involving a 13-year-old girl which he dealt with at an early stage in his career as a relatively inexperienced young doctor. For that reason, I am able to attach significant weight to his evidence about the conversation with Dr Bunemann. It was also supported by the contemporaneous jottings he had made of parts of the conversation which I considered provided a vital clue as to the nature of the conversation. I also took account of the fact that, although he was reluctant to elaborate on this, it was clear from his evidence that Dr Weir had not been reassured by the advice given by Dr Bunemann.

[481] In the course of her submissions on behalf of Dr Bunemann, Miss Keane invited me to reach no conclusions as to the nature of the conversation. She attacked Dr Weir's credibility and reliability having regard to his conversation with Dr McAlpine and his own evidence which she considered was self-contradictory. I did not accept that

assessment. I have already indicated that I preferred Dr Weir's evidence as the more accurate reflection of the conversation between himself and Dr McAlpine. In my assessment, Dr Weir was credible on this issue and reliable insofar as he could recall events and had some jottings to assist him. I agree that I am not in a position to make clear findings on the exact content of the conversation but I am satisfied that I can reach a conclusion as to the general nature of the conversation from the evidence before me.

[482] Accordingly I have concluded that Dr Weir *must* have given Dr Bunemann certain detailed information about Kathryn's presentation (including her age and lack of previous medical history) and the results of her full blood count, at least insofar as her low haemoglobin, her severely depleted platelets and high white cell count. I consider it likely that he would have mentioned the differential diagnosis in the course of that conversation. The inclusion in that diagnosis of some sort of underlying haematological malignancy was part of the reason he was phoning for advice.

[483] Frankly, it is inconceivable that he would not have conveyed the important and relevant information about Kathryn's condition to Dr Bunemann. Dr Weir was a junior doctor who was very worried about a young, seriously ill patient in his care. He was phoning specifically for advice as well as authorisation for the release of platelets. It was clear that he was urgently seeking assistance and input from the specialist discipline. It follows, too, that Dr Weir must have given sufficient information to Dr Bunemann for the latter to release platelets. Thus at the very least Dr Bunemann had details of Kathryn's full blood count. The platelet count alone should have rung alarm bells and precipitated further action on his part.

[484] Even if he had not been given more detailed information beyond the blood results (which I do not accept), there was a separate professional onus on Dr Bunemann, as the physician representing the haematology department, to ask for further information and seek clarification if necessary. Dr Bunemann's seniors and the expert witnesses confirmed this. Such a conversation was a two-way affair, a dialogue between two professionals. Dr Bunemann himself acknowledged this responsibility to interrogate and was sure that he would have asked if there was anything else he needed to know or any additional test results he required. Dr Sharp explained how he went into "autopilot" and asked the same tranche of questions in order to elicit the information he required. This was something that came with experience. Professor Yin also stressed the duty of the on-call haematologist to ask pertinent questions. Dr Hanley said that a basic qualification for someone in a position of giving specialist advice was the ability to prompt the person seeking the advice in terms of what they needed to do or needed to be thinking about. The quality of the advice given was dependent on the information given. Dr Hanley explained that giving advice over the telephone was sometimes associated with communication difficulties: how to recognise that and avoid it came with experience.

[485] It was not clear from the evidence whether Dr Bunemann asked for and was given additional information about other test results which Dr Weir had to hand or whether he had ordered further tests. It is not possible to reach any firm conclusion on that. However, I am satisfied that the gist of the advice given by Dr Bunemann was as jotted down by Dr Weir, namely, that a platelet count above 10 was okay unless bleeding, the issue of one pool of platelets and to phone back in the morning if further advice was

required. No blood film was discussed or instructed and no further advice was given. Dr Bunemann neither offered to come in nor did he contact his consultant.

Discussion

[486] There was no doubt in the minds of the experts and his senior colleagues that the quality of advice given by Dr Bunemann was severely wanting. Dr Morrison was clear that he should have ordered an immediate blood film, contacted her to come in and should have come into the hospital himself. Dr Sharp agreed. That view was universally endorsed by each of the experts. Professor Hunt was shocked that no blood film had been prepared given the blood results. Thus there was no question that Dr Bunemann should have ordered a blood film or, at the very least, have contacted Dr Morrison.

[487] It appeared obvious to the experts that Dr Bunemann was inexperienced and unable to deal with the clinical situation with which he was faced. Dr Hanley felt that the advice given indicated that: simply to say "give platelets and see what happens" was indicative of an inexperienced person. Therefore the most likely reason that Dr Bunemann did not contact his consultant was because he did not recognise the situation for what it was. In his opinion, it was inappropriate to have placed Dr Bunemann on call.

[488] Moreover, Dr Hanley did not think that Dr Bunemann was in a position to give specialist advice. His interpretation of Dr Bunemann's actions was that it was clear that he did not formulate a differential diagnosis of the haematological issues which was what was needed at that point. That, he said, was where the specialist haematological way of looking at things would come in.

[489] In terms of what would be expected, Professor Grimwade's view was that the person should be aware of the range of possibilities. The on-call haematologist needed a good medical training as well as a knowledge of haematological conditions in order to have an informed opinion having seen results such a Kathryn's. Specifically, he would need to have an awareness of emergency situations in haematology and what particular conditions would give rise to such abnormal results. APL was such a rare disease in children that he would not have expected it to have been the first thing in the mind of the on-call haematologist but he should at least have considered some form of leukaemia.

[490] There is no doubt that Dr Bunemann's failure to contact the haematology consultant was an astonishing omission. As was submitted by Mr Khurana, no reasonable explanation had been offered for that. No-one could understand why Dr Bunemann did not contact a consultant, including Dr Bunemann himself. It seemed obvious that he should have recognised that the situation was beyond his competence and that he needed to phone Dr Morrison. However, Mr Khurana argued that there was no evidence before the court which would enable me to reach any conclusion as to the reason for his failure to do so. While I do, indeed, accept that Dr Bunemann's failure to escalate matters and involve the consultant was incomprehensible, I do not accept the proposition that the evidence is silent as to the reasons for that. On the contrary, it seems to me that there is evidence from which I can draw the clear inference that, at least in part, this was due to Dr Bunemann's overall inexperience in haematology.

[491] His inexperience was evident on a number of counts, not only by his omissions but also from his actions. He failed to take proper account of the information supplied by Dr Weir or to ask pertinent questions with a view to identifying the underlying reasons for the blood results. He did not seem to appreciate the importance of finding out the reason for the abnormal results. Thus he failed to appreciate the need for an urgent blood film. Similarly, he did not appear to take any account of the possible differential haematological diagnoses, with particular regard to TTP, but issued platelets when it might have been dangerous to do so. It would appear that Dr Bunemann also failed to recognise not only the seriousness of the situation but also the fact that he was dealing with a potential haematological emergency. Hence the need for urgent consultant input. This was evident during the initial contact, again later on when he was alerted by Dr Weir of the proposed neurosurgery (when Dr Bunemann *must* have known about the bleeding in the brain) and later still when contacted by the anaesthetist, Dr Urquhart, for the release of further platelets peri-operatively. Thus there were several stages at which he ought to have realised that consultant advice was required. His actions and omissions were redolent of the response of someone who did not have the experience required to provide the necessary specialist input. Accordingly, I accepted the evidence of the experts in respect of Dr Bunemann's inexperience and that fact that he was, as Dr Hanley put it, out of his depth. That, then, raised the question whether it was appropriate that Dr Bunemann was placed on call.

[492] Mr Khurana expressed concern that this was not an issue which had been raised in the course of the evidence. While I accept that it had not been explored in any great depth with the relevant factual witnesses, it was an issue which was before the court

from the outset albeit that it only came into focus at a fairly late stage with the expert witnesses. It was an issue that was raised early on in the expert reports. It was also a matter about which Dr Morrison, at least, was questioned by the court on two occasions.

[493] Dr Morrison was asked why it was that a person who was not a haematologist would be on call for haematological advice. Her reply to that question was somewhat obtuse. She said that it was partly training and experience for the individual concerned. Some of the calls would be from nursing staff on the wards about haematology in-patients. Some of the most frequent calls would be very straightforward matters, allowing the individual to gain experience in dealing with them. Occasionally there were calls outside the normal run of things. These were the calls that the consultants would be expected to be phoned about and were there to help with. She did not further elaborate.

[494] She was asked directly whether, from her position as a consultant haematologist, she considered it appropriate for someone with Dr Bunemann's experience (as an SHO as opposed to as SHO3) to be placed in the position of responsibility on call. She explained that the on-call doctors were fully supported by the consultants and were made aware that the consultants were there to support them if they had any concerns at all about anything they were dealing with. Both she and the other consultants were satisfied that Dr Bunemann was sufficiently experienced to be placed on call and therefore did not consider that it was inappropriate for him to be on call.

[495] Miss Keane, counsel for Dr Bunemann invited me to conclude that it was not appropriate that Dr Bunemann with his limited experience should have been placed on

call and to include that in my determination under section 6(1)(e) as a matter relevant to the circumstances of Kathryn's death. She recognised that she had some difficulty with that submission in that Dr Bunemann's evidence was to the effect that he was sufficiently aware requirements of the on-call responsibilities. Indeed that formed part of Mr Khurana's argument that there was no evidence to suggest that Dr Bunemann was not capable of being on-call: he had been assessed as competent by the haematology consultants and that by his own evidence he appreciated that he should have contacted his consultant.

[496] Significantly, the procurator fiscal, in the public interest, submitted that if the evidence were accepted that Dr Bunemann should have recognised the need to contact his consultant then it was not appropriate to conclude that he should not have been placed on call. Dr Bunemann had some experience of haematology having been in the department for a number of weeks. In effect, the submissions of the Crown and those on behalf of the Health Board invited me to conclude that Dr Bunemann was sufficiently experienced to be on call but that for some inexplicable reason, he failed to obtain consultant input. On his own evidence he knew to contact a consultant in these circumstances and he appreciated that he had a duty to interrogate Dr Weir had he required further information. For some inexplicable reason, he failed to do either of these things. In these circumstances it could not be said that there was a failure in the system and that it was inappropriate that he be placed on call.

Conclusions

[497] The evidence before me concerned the on-call system as regards consultants, trainees and doctors of the SHO3 grade. There was no evidence to suggest that this had given rise to problems of any kind, far less serious omissions of the nature under consideration. On the contrary, it appeared to have worked satisfactorily. As regards the inclusion of SHO3s on the rota, clear instructions were in place that encouraged early contact with consultants in cases of doubt or difficulty. Consultant advice was readily available at all times. Subsequent changes to the system had been introduced not as a result of local concerns but on a national basis primarily in response to European Directives on junior doctors' hours. Accordingly, there was no evidence that would support any finding under section 6(1)(d).

[498] However, there is clear potential for serious public concern in circumstances where a non-haematologist is on call for the haematology department. It seemed axiomatic that the person on call for haematology should be a haematologist.

[499] The qualities required for an on-call haematologist according to the experts suggested someone with considerably more experience than that of Dr Bunemann. Again, I bore in mind my concern that such qualifications might be what was expected in the centres of excellence and perhaps were not did not reflect the first-line response in a general hospital. However, it was significant that none of the experts had ever come across someone as junior as an SHO occupying such a position.

[500] In order to assess the appropriateness of the on-call arrangements in the Victoria Infirmary and Southern General it is necessary to understand what was required of the on-call doctor. Although I accepted that this could have been more fully explored with the consultants concerned, it seemed clear that the role of those on call as far as the SHO3s were concerned (in which I include Dr Bunemann) was to act as a gatekeeper. As such they had to decide what they could appropriately deal with and what required to be referred on to the senior staff.

[501] In order effectively to perform this filtering task, the person manning the gate, as it were, has to be sufficiently knowledgeable to recognise which cases are capable of being dealt with and which should be referred upwards. I am satisfied that Dr Bunemann had gained enough experience in the haematology department to have recognised that Kathryn's was a very unusual situation which required consultant input. In that sense, it cannot be said that it was inappropriate or unsafe to place him on call.

[502] I accepted Mr Khurana's submission that it was and remains a standard practice throughout the health services for junior doctors to perform on call duties. Although there was no direct evidence to that effect, it is a matter which can be considered to be within judicial knowledge that, particularly in respect of new presentations in the accident and emergency and subsequent initial referrals, junior doctors are routinely the first point of contact for a patient. Likewise, it is obvious that consultants are not on call to deal with routine matters. There was, however, an additional element in Dr Bunemann's position. He was representing a specialist department and was therefore in

the position of being contacted by other doctors, often at a more senior level, specifically for haematology advice.

[503] In such circumstances, it seems not unreasonable that general physicians who call the duty haematologist for the purpose of obtaining specific haematological advice are entitled to expect that the person at the end of a phone is qualified and sufficiently experienced to give such advice. Clearly Dr Weir thought he was speaking to a haematologist.

[504] Neither Dr Weir nor Dr Urquhart appreciated that they were speaking to a non-haematologist at the end of the phone. They were unaware of Dr Bunemann's status and were certainly not aware of the fact that they were speaking to someone who was not a haematologist. In the course of his evidence, Dr Bunemann volunteered that he always told callers that he was in a junior position. Whatever his general practice, I am satisfied that he did not do so on this occasion. Dr Urquhart said in evidence that had he known that Dr Bunemann was not a haematologist and was a relatively junior SHO, he would have asked that he contact his consultant.

[505] The situation was compounded by the fact that Dr Bunemann proceeded to give out advice on behalf of the haematology department, the very advice that was being sought. Thus it seemed to me that it was entirely reasonable for Dr Weir to have thought that he had received the necessary haematological advice, albeit he was not particularly reassured by it. Likewise, Miss Brown was aware that there was on-going input from haematology and was, in my opinion, entitled to assume that it was qualified input. I

have no doubt that had she been aware of Dr Bunemann's position she likewise would have insisted that he contact his consultant or, more likely, that she would have initiated consultant-to consultant contact herself.

[506] Accordingly, while the on call junior doctor might have been performing a very basic duty, unless that was fully understood throughout the hospitals, there was an obvious danger that it would be reasonably assumed that informed haematological input had been obtained through contact with the person on call.

[507] Thus it appears self-evident that haematological specialist advice should be given by a haematologist, particularly where the individual on call is covering two large hospitals serving an extensive catchment area. No matter how accessible the consultant, the fact that, in 2004, the on-call service for the haematology specialty was manned by a non-haematologist significantly more junior than his other SHO3 colleagues left me with a profound sense of unease. For that reason, I have included it as a matter relevant to the circumstances of Kathryn's death in the public interest.

[508] However, in order to assuage any public concern arising from this, it is important to record that the current on-call arrangements are different. Consultants and specialist registrars now perform the on-call duties. In that sense any public concern may be entirely assuaged.

Consequences of lack of adequate haematological input

[509] The focus of the inquiry returns to the crucial hours between Kathryn's admission and her deterioration shortly before 2am. It becomes necessary to consider what the consequences were of the delay in diagnosing her condition and the failure to engage consultant input and whether they affected Kathryn's chances of survival.

[510] In this connection, what actually happened and what might reasonably have been expected to happen had a blood film been made and consultant involvement obtained require to be considered. It is necessary to examine the various timings and treatment in order to reach any conclusions as to whether Kathryn's chances of survival were affected. However, such an examination must be considered in context. There are two contextual aspects which are particularly important.

[511] First, throughout most of her time in A&E, Kathryn's outward clinical condition remained stable. She was bright, alert and talkative (in both English and French). Until her sudden and precipitous decline around 02:00, the only sign that she was less stable was the transient episode in the scanning room which occurred around 01:15 and from which she appeared to make a swift recovery, returning to her previously fully alert state. It must be appreciated that Kathryn was a new patient and that these were early investigations to try to identify what was wrong with her. It was a Sunday night and outwith full hospital working hours. We now know that Kathryn was suffering from an extremely rare and immensely complex disease, unlikely ever to have been encountered by general physicians and rarely seen by haematologists. Many other conditions such as septicaemia were much more likely than this rare leukaemia. It is also important to

appreciate that it was recognised by the expert witnesses that Kathryn's disease was progressing very rapidly.

[512] Secondly, any examination of timing must be pragmatic and realistic. A counsel of perfection is not the test against which the actions of the medical team should be measured. Nor is the "ideal world", a term often used by the expert witnesses to describe a hoped for, rather than a likely, scenario. Rather it is what was realistically and reasonably practicable in a real-life situation. This was recognised by the expert witnesses, all of whom were at pains to highlight that, in real life, things take time. As Ms. Myles said, paging someone takes time, as do the subsequent conversations. Walking along the corridor takes time; transporting patients to scans takes time; filling out forms and writing up notes takes time, etc. These minutes add up but they are necessarily and unavoidably spent. Professor Hunt put it thus: "the trouble with medical times is that there is always slippage". In this connection, the danger of unrealistic over-analysis must be guarded against. I was conscious that, years later, witnesses were being asked to remember things in meticulous detail in order to calculate precise timescales. This was so even where they had made it clear that their recollection was vague. This amounted to little more than a futile academic exercise. I have been invited to reach conclusions based on calculations of minutes. For example, counsel for Ms Crawford invited me to conclude that "by approximately 23. 17 – 23. 41 a blood film could have been examined". Ten years on, it is impossible for me to reach any such precise conclusions.

[513] Before considering these crucial hours in the Victoria Infirmary, it is important that I set out some of my concerns about the quality of the opinion evidence in this connection. During my careful review of the evidence, I noticed that some of the conclusions of the experts were based on information that was at times wrong or incomplete. Some of the conclusions contained assumptions, not all of which were justified. I was aware that during their evidence, the experts were having to revisit their original opinions once they had been advised of the correct factual basis. This is not an unusual feature: by the time the experts give evidence the court has heard evidence from the factual witnesses which may not accord with the information contained in the original letters of instruction. However, this gave scope for confusion and for that reason I considered their opinions with particular care.

[514] Moreover, it seemed to me that the expert haematologists did not always have regard to the context in which the events occurred. Neither did they appear to take full account of the on-going neurological issues which were a key feature in Kathryn's deterioration and death.

[515] It was also difficult to keep separate the 2004 position and the present day position as far as treatment protocols were concerned. Although the court can apply the benefit of hindsight in considering whether a precaution might have been a reasonable one, it is important to have regard to what the medical knowledge and the recognised treatment protocols were at the time that the death occurred. I was concerned that past and present became conflated at times. For example, Professor Hunt, when describing what the Kathryn's treatment should have, been included a reference to tranexamic acid. While

available in 2004, it was not a generally recognised treatment and was therefore not one which would have been reasonably expected to have been used in 2004. Again, fresh frozen plasma *and* cryoprecipitate were mentioned as what would have been expected for fibrinogen replacement despite evidence that cryoprecipitate was not widely used ten years ago. Likewise, the treatment protocols described depended on whether a diagnosis of APL had been made. On occasion it appeared that the experts were describing expected treatment for APL at a time when no provisional diagnosis of APL had been possible.

[516] Again, I was conscious that the experts might have been considering this from the perspective of expectations within specialist leukaemic centres. This was true also in respect of timescales for diagnosis and treatment. Some of the estimates were wholly unrealistic and failed to take proper account of the situation at the Victoria Infirmary. For example, in calculating what he considered a reasonable timescale for platelet transfusion, Professor Grimwade had not realised that platelets were off-site and had to be obtained from another hospital. This involved a recalculation. For these reasons, I have considered this aspect of the expert evidence with particular care in order to be satisfied that any opinions as to timescale and treatment were ones on which I could safely rely.

Timescale in the Victoria Infirmary

[517] Kathryn was attended to immediately on arrival. She was seen by Dr Thomas within 7 minutes. Thereafter she was under the sole care of either Dr Thomas or Dr Weir until she was transferred to the Southern General. She required to be examined at first by

Dr Thomas. She underwent a thorough examination. From the outset, Dr Thomas had recognised that Kathryn was very unwell. She suspected that there was some serious underlying condition and she ordered the appropriate initial investigations, including blood test for a full count and a coagulation study. Dr Thomas properly referred Kathryn to the receiving physician for admission and further investigations. The time taken by Dr Thomas was considered entirely reasonable.

[518] Dr Weir responded immediately arriving in the department shortly after 23:00 hours. He proceeded to examine Kathryn himself and take a fresh history. It was Dr Beattie's contention that this was entirely unnecessary and caused delay. However, there was universal approval of his approach by the senior consultants and the expert witnesses. They recognised the importance of a separate examination by the receiving physician on behalf of the medical team. Likewise it was good practice to take a fresh history thereby lessening the chance of perpetuating any previous inaccuracies that might have been recorded. Dr Weir's initial examination, including time taken to write up his notes, was about an hour in total. This, too, was considered to be reasonable when reviewed by the expert witnesses. Accordingly, I was entirely satisfied that there was no basis for any criticism of Dr Weir's actions. He acted appropriately and the time spent examining Kathryn was a necessary part of her admission procedure. Dr Weir then took urgent steps to involve other specialties and organise further investigations.

[519] After speaking with one of the medical consultants, Dr Weir immediately contacted the on-call haematologist, Dr Bunemann. The timing of this telephone call was 00:15 hours. Again this was considered to be within a reasonable period of time. As the blood

film was a specialist haematology laboratory investigation carried out under the auspices of the haematology department, this first contact with the haematologist was the appropriate starting point in considering the timings of the haematology investigations.

[520] It follows that the earliest point at which Dr Bunemann could have instructed a blood film or contacted his consultant was around 00:20. We know that he ordered platelets at 0026 so he was making telephone calls in respect of Kathryn at this time. Had Dr Bunemann appreciated the need for a film, he would undoubtedly have contacted Dr Morrison - he would have realised that this was something serious and beyond his capabilities. In any event, as he was not able to interpret a blood film, she needed to come in to examine the blood film. Even if he had not thought about a blood film but had simply phoned his consultant for assistance, Dr Morrison would undoubtedly have instructed that an urgent blood film be prepared and she would have come in. Either way, the outcome would have been the same: a blood film would have been made and a consultant haematologist would have been involved in Kathryn's care.

[521] It is, therefore, reasonable to assume that the blood film would have been instructed by 00:30 and Dr Morrison would have made her way in. Platelets might not necessarily have been ordered at that stage until the film had been examined. However, it seems more likely that they would have been ordered in anticipation that they would probably be needed but no instruction to transfuse them could have been given before the blood film was examined given the potential for TTP or other conditions where platelets were contra-indicated. I noted that Dr Morrison suggested that as overt

bleeding was rare in TTP, she might have taken a risk and transfused platelets before having seen the blood film. Professor Yin made reference to that too. However, the inherent risks of such a course would preclude its consideration as a reasonable precautionary measure.

[522] Allowing time for the instruction to be passed to the BMS, the machine to be switched on and primed and for the production of the film, it is reasonable to assume that it would have been ready at about the same time as Dr Morrison arrived at the hospital, sometime shortly before 0100 hours. In her evidence, Dr Morrison said she would have had to liaise with the laboratory, examine the film, and go to see Kathryn. She explained that she would have carried out a full assessment, confirmed the history and carried out her own examination. Thereafter she would have telephoned Yorkhill Children's Hospital to discuss things with a paediatric haematologist. Although she said she would have acted "to get things going as soon as possible", inevitably all these things take time. That being so, I consider it most unlikely that any treatment decisions based on a preliminary diagnosis would have been made much before 0130. In these circumstances, there would have be no appreciable difference in the timing of the first transfusion of platelets. Indeed, had Dr Morrison waited until the blood film had been examined before ordering platelets, they would have arrived at the hospital even later than they did.

[523] Given the depth of the family's concerns, I have also considered an alternative timescale which could be described as a best case scenario had the full SOP been implemented. Thus a blood film would have been prepared automatically on receipt of

the abnormal results and brought to the attention of a haematologist, as would have been the case during normal working hours. In this scenario, it would be reasonable to take as a starting point the time immediately after the coagulation results had been obtained. In the laboratory at night only one BMS was available. Both these tests had to be carried out and both sets of results telephoned to A&E. There was evidence, too, that cross-matching would have been done which would have taken 20 to 40 minutes. So a realistic starting point would have been about 23:30. The blood film results would have been available, at the earliest, shortly before midnight. On the assumption that the BMS, having reviewed the film, then contacted Dr Bunemann to have the film examined, it seems reasonable to suggest that this contact might have been made by midnight. Dr Bunemann would then have had to phone Dr Morrison to ask her to come in and examine the film. There would doubtless have been a conversation between Dr Morrison and Dr Bunemann. As before, allowing time for Dr Morrison to come in from home, look at the blood film, take a history, examine Kathryn and speak to her paediatric haematology colleagues, it appears reasonable to conclude that a provisional diagnosis might have been made at the earliest by about 0030. The same comments as before apply in relation to the platelets. Had they been ordered by Dr Bunemann at once, the timing would have been the same and they would not have arrived. Had he waited to receive instructions from Dr Morrison, they would have arrived even later.

[524] There were suggestions that a provisional diagnosis could have been made by 23:00 and that platelets could have been available by midnight. I have rejected these as totally unreasonable. Such estimates took no account of the logistics involved nor of time taken in the real world. They were estimates which I considered were unsupportable and

unrealistic and had been casually reached. On a best case scenario that a provisional diagnosis could have been made by midnight there had to be time built in to allow Dr Morrison carry out her own examination etc. That could reasonably have taken some 30 minutes or so. She then would have had to issue instructions as to the necessary treatment, these had to be carried out and treatment commenced. Simply saying that diagnosis might have been made by a certain time does not, of course, mean that treatment would have started simultaneously.

Examination of blood film

[525] It is convenient here to consider whether, had Dr Morrison examined the blood film, a provisional diagnosis or suspicion of APL would have been reached in the early hours of that morning. Dr Morrison did not reach such a conclusion when she examined the film from the blood sample taken at the Southern General later on. She identified the generic acute myeloid leukaemia but did not identify the M3v subtype of acute promyelocytic leukaemia. This of course is relevant only to the blood film prepared from the admission blood sample in the Southern General and not the earlier sample at the Victoria.

[526] This was a troubling aspect of the evidence. It was difficult to comprehend why it was that looking at the same blood film a few hours later, Dr Macdonald was able to note some features of APL and to suspect that it was possibly the M3 hypogranular variant. In particular, in his report he identified folded or bi-lobed nuclei, classical signs of the subtype. In his evidence he confirmed that he strongly suspected APL. Yet Dr Morrison, likewise an experienced consultant, did not seem to have recognised these

classic signs. The physical blood film was not produced and was thus not available for independent scrutiny by the experts. This was unfortunate. None of the experts felt able to comment on this in the absence of the film itself. Thus this remained an unresolved issue of some importance. It was difficult to reconcile the two interpretations. However, I considered that it left open the real prospect that APL should have been able to have been suspected from the film at an earlier stage. However, in relation to that film, the earliest that APL could have been suspected was at about 09:00 on the 21st when Dr Morrison first saw it. Given Kathryn's condition by the time Dr Morrison actually became involved, what was identified in the blood film from the Southern General was of limited relevance and, like the experts, I could reach no conclusion on this.

[527] Had Dr Morrison come into the Victoria Infirmary during the night, the blood film that she would have looked at would, of course, have been the film from the first blood sample which had been preserved by Mr McLauchlan and stained the following morning. Although this film was not available either, it had been reported although Kathryn by that time was no longer a patient within the hospital. It was noted as "prepared but not looked at until 0900 hours on 21 June but not reported until 16:37 hours on the 22 June", after Kathryn had died. The report of that blood film was in the following terms: *Majority of white cells are blasts with granular cytoplasm. Appearances would be consistent with Acute Promyelocytic Leukaemia.* The identity of the person who authorised the report was not included in the authorisation box and no witness was led who had examined that report. Although this was a matter of deep concern to Dr Beattie and Ms Crawford, I did not share that concern and concluded that the identity of the person who did so was irrelevant. However, it seems clear that the morphological appearances were such as

would have raised the necessary suspicion of APL for the purposes of identifying the correct treatment protocol. (The later blood film prepared from the sample taken at the end of surgery likewise contained similar appearances.)

[528] No firm conclusions can be drawn as to whether Dr Morrison would have identified APL from the Victoria sample but it seems likely that there was evidence to support the suspicion of APL. Accordingly, I am satisfied, on a balance of probabilities, that a provisional diagnosis of APL could have been made earlier. Even if APL had not been suspected, AML would have been suspected. The only difference in treatment would have been the administration of ATRA. Therefore it is necessary first to consider the quality of the supportive treatment that Kathryn received before considering the implications of ATRA.

The treatment Kathryn received

[529] The initial response to both conditions was urgent provision of blood products to support Kathryn's thrombocytopenia and correct any coagulopathy. Dr Bunemann released one pool of platelets and ordered two units of packed red cells, the latter to address her anaemia. He gave no instructions about repeating the platelet count or otherwise monitoring Kathryn's coagulopathy. However, as it happened, a full blood count and coagulation screen were repeated shortly after she arrived at the Southern General Hospital.

[530] Although packed red cells had been prescribed, it would appear that these were never transfused. The only blood product support Kathryn received in the Victoria

Infirmary was a single unit of platelets. These were not transferred until 02:00 hours. Accordingly, concerns were raised as to the adequacy of the support and the timing of the platelet transfusion.

Adequacy of blood support

[531] This matter was first explored with the various Glasgow consultants. Dr Morrison told the Inquiry that she thought that she would have done the same as Dr Bunemann and ordered platelets and two units of packed red cells. She assessed the fibrinogen level as comfortably within acceptable limits and was conscious of the need to guard against the risk of thrombosis, reflecting the standard approach in 2004.

[532] Dr Morrison considered that it was reasonable to have prescribed one unit of platelets initially. Dr Macdonald thought that he would probably have ordered two. Although Kathryn's platelet increment was excellent, he pointed out that her response would not have been known in advance. Had there been time to do so, Dr Macdonald thought that it would have been reasonable to have ordered two pools but to have repeated the platelet count after transfusing the first pool. There was a need to be mindful the platelets were – and still are – a scarce resource which should not be overused or wasted.

[533] The experts were asked to comment on Dr Bunemann's initial response. Dr Hanley agreed that one pool was sufficient. Typically, he said, you would give one pool and then monitor the increment by re-checking 15 to 30 minutes later. If the target level had not been achieved, then more should be given. Professor Hunt, too, thought that one

pool of platelets was sufficient although she might have ordered a second in case they were needed. However, one pool was sufficient to increase the count.

[534] Professor Yin's view was that you prescribed platelets - "lots of it" - as quickly as possible. He would have ordered three pools of platelets in order to try to get her level above 100.

[535] Regular monitoring of Kathryn's coagulation was an essential aspect of her care. The general view was that this was adequate. Professor Hunt, the coagulopathy expert, considered that even where APL was suspected, repeat monitoring within a 4 to 6 hour time scale would have been appropriate. Because Kathryn was clinically well, the timescales in which the repeat screens were done were fairly satisfactory and in response to the clinical circumstances. There was general agreement about this among the experts.

Timing of platelet transfusion

[536] Dr Bunemann telephoned the regional blood bank at Gartnavel Hospital immediately after speaking to Dr Weir. The request for platelets was received there at 00:26. The order was immediately made up and dispatched to the Victoria by taxi and received in the laboratory at 00:54 hours. That was in accordance with the timings that the Glasgow consultants would have anticipated. The expert witnesses recognised that it was not uncommon for platelets to come from an outside source and the timings appeared reasonable. Therefore the platelets arrived in timely fashion.

[537] Unfortunately, that arrival coincided with Kathryn's CT scan. Significantly, Dr Weir checked with the laboratory to see if the platelets had arrived. On being advised that they had not, he opted to continue with the scan rather than wait for the platelets. While Kathryn's father contended that this further delayed vital treatment, there was no criticism of Dr Weir's decision by any of his senior colleagues or by the experts. It was a judgment call. The scan was an urgent and necessary investigation given Kathryn's neurological signs. It was vital to discover what was going on inside her head. Dr Weir did not know how long it would be before the platelets arrived. As Dr Hanley explained, this was a difficult decision – there was no easy way of prioritising one over the other. While, in retrospect, it might have been better to have delayed the scan until after the platelets have been transfused, I am satisfied that in the situation he was faced with at the time, the decision to proceed to the scan was one that Dr Weir was entitled to make in the proper exercise of his professional judgment.

[538] The platelets were not transfused until about an hour after their arrival. Kathryn had returned from her scan about 01:30, after the transient episode. It is worth noting that the platelets could not been waiting for her in the department as they required to be kept in the laboratory in order to maintain temperature and agitation. Although there was no direct evidence about this, they must have been ordered from the laboratory at some point after she returned from the scan as they were available for transfusion by 02:00. However, during the period between 01:30 and 02:00, nearer to 02:00, Kathryn's condition dramatically deteriorated resulting in coma and emergency intubation and anaesthesia. Thus the period between her return to A&E and administration of the platelets is broadly accounted for. In all the circumstances, the CT scan and Kathryn's

deterioration provided satisfactory reasons for the delay of an hour between the arrival of the platelets and their transfusion. Notwithstanding these coincidental occurrences, it was Dr Hanley's position, bearing in mind that Kathryn only arrived at the hospital at 22:00 and taking account of the logistics involved, that the administration of platelets by 02:00 represented a reasonable degree of urgency. That being so, it cannot be said that there was an undue delay in the transfusion of platelets.

Impact of delay in diagnosis and lack of consultant input

[539] In order to assess the impact of the delay in diagnosis as a result of the lack of a blood film and the failure to contact Dr Morrison, it is necessary to consider the expert evidence about Kathryn's prognosis and the likelihood of her survival. As has already been noted, if Kathryn was to have any real chance of survival, the crucial time was the period prior to 2am. As Mr Mallucci concluded, whether she survived was essentially a matter of haematological input during her time in the Victoria Infirmary. It depended on whether steps could have been taken to stop the bleeding in her brain or prevent a re-bleed in order to avoid the decompensation that occurred shortly before 02:00 hours.

Kathryn's condition at presentation

[540] By the time Kathryn arrived in A&E her leukaemia had advanced to the point where she had already suffered a significant intracranial bleed. Indeed it was Dr Hanley's view that it was what had happened at home in the period before her admission that ultimately determined the outcome. At the time of her presentation at 22:00 hours, she was in a high risk category from the bleed alone. Significant numbers of patients with APL involving intracranial bleeding will die, many in the first 24 hours. Professor Grimwade described how the complication of CNS bleeding was associated

with an increased risk of death so that the haemorrhagic death rate associated with the disease, even after the introduction of ATRA, remains significant. ATRA has not had much impact on early death rates. He agreed with Professor Hunt's estimate that there was a 20% risk of death associated with intracranial bleeding and that some patients die very quickly before treatment can begin and before they reach clinical trials. Death rates based on such trials do not take account of these patients thereby making the true death rate higher than the 20% reported in the trials literature. Professor Yin agreed that in his experience these patients do badly and considered that Kathryn's death was, not inevitable, but very probable. Dr Hanley agreed and considered Kathryn's outlook to have been "very, very poor - pretty bleak".

[541] Moreover, there was indisputable evidence that Kathryn presented in the high risk group for a number of other reasons but with particular reference to her high white cell count. Reference was made to a prestigious publication in Haematologica 2010 by the Italian group Lo-Coco *et al* entitled *Early Haemorrhagic Death and Postpartum Therapy in APL.*

[542] Risk associated with APL is classified in accordance with the "Sanz score". This score assigns patients with APL into risk groups. It rates risk according to platelet count, white blood cell count and LDH levels. LDH is an enzyme associated with high red cell turnover. (The relevant test is not essential to diagnosis but tends to be used as a prognostic test.) The Sanz prognostic scores take account of treatment protocols. If the presenting white cell count is above 10, the risk of death is high (Kathryn's was over 30). Professor Hunt's evidence was that there are some data showing that there is a worse

prognosis if the patient has a high LDH in combination with a high white cell count and a low platelet count, as Kathryn had.

[543] Professor Grimwade explained that the Sanz score is a recognised measure of risk. However, the UK adopts a slightly different approach where the thinking is that the platelet count does not really add anything. Accordingly, in the UK in terms of risk assessment the platelet level is lumped together with other features while in the Sanz score the platelet count is separately included. Under either scoring system, Kathryn was in the high risk category under all three headings. The white cell count is recognised as the key factor in determining outcome. Kathryn was in the group showing the highest risk and the poorest outcome. According to Professor Hunt, Kathryn presented in a condition of high morbidity and mortality. The odds were heavily stacked against her.

[544] Although the combination of Kathryn's risk factors put her at very high risk of death, it did not mean to say that she had no chance of survival. There was general agreement that her death was not inevitable when she presented at the Victoria that night. Paradoxically, APL has a good survival rate if patients at high risk can be stabilised so that they survive long enough for ATRA to start working. Those who do, generally go on to make a good recovery. That is why prompt diagnosis and aggressive support with appropriate blood products was vital.

Available blood support in 2004

[545] Dealing first with the provision of aggressive blood support, I was satisfied that in 2004 there would have been no requirement to have corrected the fibrinogen level on

Kathryn's first blood results. The evidence was clear from the experts and from the various haematology consultants who were asked about this that in accordance with guidelines then in place, a fibrinogen level of 1.8 would have been regarded as within acceptable limits and that no specific measures to raise that level would have been indicated. Professor Grimwade acknowledged that the fibrinogen was not particularly low. Professor Hunt was clear about this. A fibrinogen level of 1.8 was within a normal range and required no additional treatment. The 2006 guidelines - which Professor Hunt was involved in drawing up - was based on the practice in 2004 which was that an additional source of fibrinogen would only be indicated where the level was 1 gram or less. There were concerns at that time of the risk of thrombosis. (In the current guidelines, supplementary fibrinogen would be indicated where the fibrinogen level was somewhere between 1.5 and 2 grams.) Dr Hanley agreed that in 2004, most haematologists would not have given an additional source of fibrinogen at a level of 1.8. The fibrinogen level was not sufficiently low to warrant the giving of cryoprecipitate or any other source of fibrinogen and therefore there can be no criticism of the fact that Kathryn did not receive such an additional source of fibrinogen at that stage. In 2004, that would not have been a reasonable expectation.

[546] I note in this connection that there was evidence, which I accepted, that cryoprecipitate as the best and most efficient source of fibrinogen, would not have been widely used on 2004 outside regional centres. Accordingly it was more likely that any correction of fibrinogen levels would have been achieved by the transfusion of FFP in accordance with the standard practice at the time. It was Professor Hunt's view that FFP

would not have improved Kathryn's coagulopathy sufficiently enough to have stopped her further bleeding.

[547] In any event, by the time the fibrinogen level was noted to have fallen from the sample timed at 03:42 hours thereby indicating a downward trend, it was too late. By then Kathryn had passed the point of no return.

[548] The extent to which supportive measures would have impacted upon Kathryn's coagulopathy has to be examined in the context of the on-going disease process. Having already considered what was happening to Kathryn in terms of her intracerebral haemorrhage, it is also necessary to consider the physiological aspects of her leukaemia. Reference is made to the earlier discussion at paragraphs [258] *et seq*.

The continuing disease process

[549] In this connection, there was a difference in emphasis among the experts as to what was principally responsible for Kathryn's condition, the immediate treatment she required and its likely effect on her chance of survival. I stress that this was a difference in emphasis because there was universal agreement as to the characteristics of the disease itself and the associated treatment of APL. The disagreement arose between Professor Yin and Professor Hunt as the extent to which hyperfibrinolysis or thrombocytopenia was the key feature at work.

[550] Professor Yin considered that lack of platelets was the main culprit whereas Professor Hunt was firmly of the view that the principal problem was hyperfibrinolysis.

Dr Hanley's position was that both were important and that it was difficult to tease out the different aspects that contributed to the bleeding. Thrombocytopenia definitely contributed to Kathryn's bleeding, as did DIC and the fibrinolysis. Professor Grimwade emphasised the importance of platelets. The problem, as he saw it, was that essentially there were two mechanisms to prevent bleeding and in APL both were disrupted. For that reason, he did not disagree with Professor Hunt that the most common reason to bleed in APL was the increase of the fibrin clot breakdown which he agreed was the key issue.

[551] Thus both Professor Yin and Professor Grimwade focused on the need, initially, to ensure that Kathryn received aggressive blood support with particular emphasis on platelets. That, Professor Yin explained, was the way patients survived nowadays - the focus was on the correction of coagulopathy in attempt to avert further damage whilst waiting for ATRA to attack the underlying process and reverse it. He considered that it was the lack of platelets which was responsible for the bleeding in the first place. He argued that if the haemorrhagic area were plugged with platelets, physiologically speaking, further haemorrhage could be delayed. In the longer term, he accepted that unless the coagulopathy was addressed completely, there would be no point in platelet replacement. In that longer term, fibrinogen became more important. However, he considered platelets could have been crucial in the initial couple of hours. Dr Hanley agreed that in Kathryn's case platelets were the most important issue but also recognised the importance of hyperfibrinolysis in APL.

[552] Professor Hunt's position was that it was the activation of the clot breakdown through hyperfibrinolysis that was of crucial importance. She was firmly of the view that the lack of platelets had *not* initiated the bleeding. There were multiple areas of haemorrhage and this indicated that some other factor was causing that. The bleeding was not explained simply by a platelet count of below 50. She had never come across multiple areas of haemorrhage with a platelet count of 13. Indeed, above a platelet count of 10, it was extremely rare to see spontaneous bleeding.

[553] She accepted that research was in its early days but it looked like what was happening was that leukaemic cells in Kathryn's brain were producing lots of factors that were breaking down the clot in the brain. Where there was no clot to work on they would break down the blood vessels and cause local bleeding within the brain. She thought that a lot had gone on in Kathryn's brain for the last twenty-four to forty-eight hours. The coagulation results from the Southern General Hospital showed a classic picture of excessive fibrinolysis activation. This was because the fibrinogen levels were lower than normal but the APTT was normal and the PT only slightly deranged. This demonstrated that fibrinogen was being consumed at a much greater rate than all the other coagulation factors because it was being broken down by active fibrinolysis. Between hours of 10pm and 3am, the fibrinogen levels were falling while the platelet count had improved as a result of the transfusions, yet Kathryn continued to deteriorate. Therefore she considered that hyperfibrinolysis was the culprit: the blood picture implicated an over-active clot breakdown. Correction of Kathryn's platelet levels would have meant that there would have been higher levels of platelets in the circulating blood but that would not help what was going on in her brain very much.

[554] Professor Hunt concluded that although platelets were important, given the unique clotting problems in APL, these problems would not be fully corrected by the use of blood products alone. Giving platelets and fibrinogen was correcting the defects. It was not switching off the proteins which were breaking down the clots. Their action would simply continue.

[555] Professor Yin disagreed with Professor Hunt's analysis of what was happening in Kathryn's brain. In his opinion there was no evidence that in Kathryn's case local fibrinolysis was the major factor that caused Kathryn to bleed. He believed that Professor Hunt was speculating.

[556] Dr Hanley agreed that platelets alone were unlikely to stop the bleeding. A transfusion *might* stop it temporarily or slow it down but it would not switch off the process. He stressed that stopping bleeding that had already started was much more challenging than preventing it from starting in the first place. This appeared to reflect Professor Yin's view also. Despite his disagreement with Professor Hunt's analysis, he recognised the problems associated with fibrinogen breakdown and that, at the end of the day, the bleeding would not stop unless and until the clot breakdown was halted by the use of ATRA. That, however, did not detract from the importance, in the initial stages, of ensuring prompt and aggressive support with blood products.

Conclusion

[557] There was a fundamental difference of opinion between Professor Yin and Professor Hunt about the part played by local fibrinolysis, and as regards what had been going on in Kathryn's brain. Professor Hunt's opinion was based on the most recent but as yet unpublished research. She accepted that this research was on-going and the conclusions were not fully established. The difficulties facing any tribunal of fact in these circumstances were acknowledged in the case of *Dingley v Chief Constable of Strathclyde Police* 1998 SC 548. It is well established that where, as here, there is controversy among the medical profession, it is not for the court to resolve that controversy, to settle scientific dispute or to pronounce on the scientific validity of those opposing views. However, these conflicting opinions must be analysed and assessed against each other and against the whole facts and circumstances of the case. In so doing, two factors in particular must be borne in mind.

[558] The first is that expert medical witnesses apply very different standards of proof. The Lord President (Roger) expressed it thus at p 600:

It is a case where in their evidence the experts refer at times to a standard of scientific proof which is different from the standard of proof which the court applies when deciding matters of fact. For a court, a fact is proved if the court holds that it is more probable than not, even if it is only marginally more probable. By contrast scientific experts will obviously require a much higher standard before it can be proved that something has been established.

Lord Prosser observed of such an expert at p 601:

Mere marginal probability will not much interest him. But it must satisfy a court. It is, therefore, important for the court to keep this different approach in mind when considering the import of the expert evidence and in reaching a conclusion.

[559] With that in mind and after careful consideration, I accepted Professor Hunt's analysis as a likely reflection of what was happening in Kathryn's brain. Her analysis was based on her own cutting edge research and, a leading expert in coagulopathy, she was best placed to comment on the current state of medical thinking. Her conclusions were logical and coherent and well-supported. I accepted her evidence that the areas of haemorrhage could not have been caused by thrombocytopenia alone. That conclusion was supported by other evidence from the neuroradiologists and the neurosurgeons who, likewise, had rejected the idea of four haemorrhages occurring simultaneously. Moreover, if these *were* chloromas, that would further support the case for localised hyperfibrinolysis. The coagulation study from the Southern General appeared, as Professor Hunt said, to reflect a picture of hyperfibrinolysis. I did not agree with Professor Yin's description of Professor Hunt's opinion as speculation. This was not an opinion based on mere conjecture and supposition. It represented the current working hypothesis in a complex area of medicine. As Miss Adair put it, Professor Hunt's opinion was not speculation, it was simply not definitive. Thus Professor Hunt's opinion was highly persuasive and I was convinced that, as she suspected, something more was clearly going on in Kathryn's brain and it was probably to do with localised excessive fibrinolysis.

Could the bleeding have been stopped?

[560] There was universal agreement among the haematologists and the neurosurgeons that once Kathryn's condition had deteriorated shortly before 02:00, her condition was irretrievable. Likewise, it was the unanimous position of the haematologists that, without ATRA (and chemotherapy) Kathryn would not have survived. Therefore there

are two critical questions that arise: first, could anything have been done to have prevented the decompensation which occurred as a result of her critically raised intracranial pressure?; secondly, could her condition have been stabilised sufficiently to have allowed ATRA to have been administered and long enough to have allowed it to start to work? Both of these depended on whether the bleeding could have been stopped (or a re-bleed prevented) before that critical point was reached.

[561] As previously stated, on the basis of the evidence of Mr Mallucci and Miss Myles, I was persuaded that the bleeding would require to have stopped, not just slowed. This was on the basis of their opinion that only tiny increase in volume, such as 2 or 3 mls, would have been enough to tip Kathryn into decompensation.

[562] The overwhelming conclusion from the expert evidence was that it was highly unlikely that the bleeding could have stopped in the time available. Timescales were accepted as being, in the words of Professor Hunt,"incredibly tight". There was "the smallest possible interval, a tiny period" in which to have done anything. Professor Yin agreed – "It's really very, very tight on the facts, I won't deny that" and Dr Hanley also came to the conclusion was that there simply was not sufficient time.

[563] Professor Grimwade thought that it was *possible* that the bleeding might have stopped but he could not say how big that chance was. If the diagnosis of APL had been made in a timely fashion and if FFP, cryoprecipitate and platelets had been administered by midnight then such measures *might* have prevented further bleeding. However, in his opinion it was just impossible to know. It might have slowed down or reduced any on-

going bleeding but he could not say how likely it would have been that it would have stopped by the time Kathryn deteriorated. He acknowledged that there was a lot going on and that the situation was very dynamic. He believed that in the hours between 1 and 2 am there was not time to have stopped the bleeding and even had Dr Morrison been called in at 1am, it was still too late.

[564] Professor Yin believed that with platelet support it might have been possible to stem the flow of bleeding. Later in his evidence he said that he did not think that platelets would have stopped the bleeding but they might have done so temporarily or have slowed it down.

[565] Professor Hunt focussed on the need to give support so that there was time for antifibrinolytic drugs to have started to switch off the process of clot breakdown. From this there arose two further questions: whether ATRA could have been administered within the times available and, if so, whether there was enough time for it to act.

ATRA

[566] Although it was not entirely clear, it seemed that ATRA would not have been immediately available from the pharmacy at the Victoria. In 2004, not all hospitals kept the drug. At that time, it was a new, expensive drug, not often used outside regional leukaemic centres and it had a short shelf-life. Dr Morrison and her fellow consultant colleagues did not think that it was available off-the-shelf in the hospital. Therefore, in order to obtain it, the on-call pharmacist would have had to be telephoned at home in order to come into the hospital and source a supply from elsewhere. It was estimated

that ATRA could have been obtained within half a day. Sadly, that would have been too late for Kathryn.

[567] Likewise, ATRA is usually delivered with chemotherapy. At the Victoria Infirmary, it was not possible to administer chemotherapy during the night. Chemotherapy requires to be administered in a sterile environment. Thus the necessary arrangements could not have been put in place until the following morning.

[568] Even if ATRA *had* been immediately available and even if it *had* been administered during the early hours of the morning, from the evidence it would not have had time to have any real effect on Kathryn's coagulopathy in terms of switching off the leukaemic cells. Although there was evidence that ATRA was quickly absorbed, reaching peak therapeutic levels on average within two hours or so of administration, in terms of switching off the leukaemic cells, it took about 12 to 24 hours before it had any appreciable effect on the coagulopathy. Sadly, that, too, would have been too late for Kathryn who would undoubtedly have continued to haemorrhage in the intervening period. Without ATRA, it was agreed that she would have continued to bleed and her death was certain. Dr Hanley agreed that it was not possible to administer ATRA within the timescale available. Things were moving too quickly.

Kathryn's chances of survival

[569] The experts were asked for their opinions on Kathryn's chances of survival. In the course of their evidence, they translated her chances into percentage terms, something to which Dr Beattie took exception. He argued that it was beyond the proper remit of the

Inquiry to take account of estimates of survivability and he invited me to disregard that evidence in its entirety.

[570] Dealing first with Dr Beattie's submission, I was satisfied that this evidence was not only relevant but was of some importance in terms of assisting the court in determining matters under section 6(1)(c) and (d). I appreciated that this evidence was not a matter of scientific accuracy and it was certainly not presented as such. However, I was satisfied as to the qualifications of the witnesses to make such estimates and content that assessments of matters concerning prognosis, morbidity and individual patient outlook are standard aspects of medical practice. Accordingly, this was evidence to which I could have regard in forming my own conclusions.

[571] Professor Hunt estimated Kathryn's chances of survival at presentation to be "very low". She assessed them in single figures at 5% or less. She based this estimate on her world-wide knowledge of patients who presented with APL and associated intracranial haemorrhage and knowing how poor their survival is. She had also factored into her equation Kathryn's bad prognostic signs: her high white cell count of greater than 30, her low platelet count and her raised LDH.

[572] It was significant that her estimate of 5% was on the basis that Kathryn had presented at a haematology day ward and had been given treatment very quickly, rather than at the A&E department of a general hospital at 2200 hours on a Sunday night. It was also on the basis that Kathryn had received some "sharp, quick treatment to halt the progression of the clot breakdown and hyperfibrinolysis". That included an

antifibrinolytic drug such as ATRA or tranexamic acid (although the latter was not accepted treatment in 2004.) If the antifibrinolytic were taken out of the equation, she assessed Kathryn's chances of survival at presentation as only 1 or 2%, decreasing with every passing minute. She acknowledged that in the real world, time elapses: the clock was ticking and Kathryn's chances of survival were getting less and less. By 2am they were well down from 5% to about 1 or 2%, what she described as "a tiny chance". Although Professor Hunt was not asked specifically to recalculate this equation without the antifibrinolytic element, it could reasonably be assumed that if the revised starting point were 1 or 2%, then by 2am, four hours later, her chances of survival would have been negligible.

[573] Professor Yin considered that Kathryn had a higher chance of survival at the time of her initial presentation. At that point, he estimated that she had a 20 - 25% chance of survival. However, that was based on the assumption that it was known at presentation that she had APL and was given prompt treatment. It was of course not known, nor could it have been known, that she was suffering from APL when she presented as a new patient at 2200 hours. Between midnight and 1am, with optimal treatment, he considered that Kathryn's chances had dropped to between 10 and 15%. He associated optimal treatment by 1am with a 10% chance. However between 2am and 3am, when Kathryn's GCS had dropped, he considered that her chances of survival by then were less than 1% and that her death was almost certain. With ATRA, there was just a "tiny, tiny chance" that she might have survived.

[574] Professor Grimwade was reluctant to place numbers on Kathryn's chances of survival. Instead he chose to emphasise the challenging aspects of the disease and treatment. He was at pains to point this out stating that APL was such an "incredibly difficult" disease to treat. This was with particular focus on the problem with bleeding which he stressed was "really, really, really a problem". Where the patient presented with a high white cell count in 2004, their chances of survival were already at down at 50%. Professor Grimwade had taken account of Kathryn's clinical circumstances and had reduced her chances of survival to 25% at presentation. However, he later revised that to somewhere in between 5 and 25%. Again this assumed timely diagnosis, prompt treatment and was based on the assumption that bleeding would have stopped or been reduced. He described this as extremely poor and a "very, very bad situation" in which 2am was "the end stop" – when decompensation occurred and when Kathryn's condition became critical.

[575] Professor Grimwade acknowledged that even with all the supportive care and with the best efforts, patients still die, a sad fact reflected in the opinions of all of the haematologists. Professor Yin admitted that "despite our best efforts, sometimes we can't win because patients die in the first 24 hours because ATRA hasn't had time to act". Professor Hunt, too, acknowledged that, despite the advances of ATRA, there is still a considerable loss of life.

Submissions

[576] The procurator fiscal invited the court to prefer the evidence of Professor Hunt. It was she who possessed the expertise about coagulation which was the main issue

associated with APL. She commended Professor Hunt's evidence as logical and cohesive. On that basis, Kathryn's chances of survival were so low that it was the Crown's position that nothing could have been done to have prevented Kathryn's death.

[577] Kathryn's chances of survival were minimal, even in optimal surroundings. From the outset in the Victoria Infirmary, they were only at one or two per cent. In that connection, Miss Keane, too, invited me to accept Professor Hunt's evidence. As far as ATRA was concerned, it was simply impossible to achieve effective treatment in the time available. Thereafter, it would have taken up to twenty-four hours to work. Even if a consultant had attended, the outcome for Kathryn was unlikely to have been anything other than fatal.

[578] On behalf of the Greater Glasgow Health Board, Mr Khurana invited the court to prefer the evidence of Professor Hunt. Like the procurator fiscal, he commended her expertise: she was the only authoritative expert to give evidence on the coagulopathy associated with the M3 variant of APL. Kathryn's chances of survival were one to two per cent at best. Professor Hunt's evidence on analysis was entirely logical. The basis of her opinion and reasoning was very clear. When testing the validity of her views, one could look at the medical literature: there was produced only one anecdotal article, from the entire world-wide medical literature databases, of survival in a case with some potential similarities to Kathryn's.

[579] He submitted that Professor Grimwade was speculating as to Kathryn's chances of survival. He put the figure somewhere between five and twenty-five per cent. His

approach was not capable of withstanding logical analysis. Mr Khurana was critical of Professor Yin's approach, in particular with regard to an anecdotal case which he relied on as proof that Kathryn could have survived. That case, however, was of limited relevance as it turned out not to involve APL. Professor Yin should not be considered as reliable in terms of his assessment of Kathryn's survivability. However, ultimately, Professor Yin's evidence was in accordance with every other haematologist who gave evidence: that Kathryn's chances of survival were extremely low.

[580] Only Mr Lamont, on behalf of Ms Crawford, invited me to make findings in terms of sections 6(1) (c) in connection with Kathryn's chances of survival. Mr Lamont asked me to prefer the evidence of Professors Yin and Grimwade in assessing Kathryn's chances of survival. All that was required in terms of section 6(1) (c) was that there was a *possibility* that Kathryn might have survived and all experts agreed that she had a chance, however small. It was not part of his submission that Kathryn *would* have survived, only that she *might* have. That was what was required in terms of section 6(1) (c). It was submitted that I should apply the ordinary meaning to the word which simply meant a possibility. All of the experts gave Kathryn a chance of survival so the possibility clearly existed.

[581] In his submissions, Dr Beattie repeatedly referred to "egregious failures" on the part of the medical staff and the hospital in relation to Kathryn's care. Although, as he put it, Kathryn was "teetering on the brink of disaster" when she presented at 2200, it was his position that the presenting emergency was treatable, the underlying condition curable and the fatal outcome avoidable. It was his submission that the downward spiral

of Kathryn's underlying condition could have been halted at any time by the infusion of two units of platelets and two units of FFP.

"Might" in terms of section 6(1) (c)

[582] Before a finding in terms of section 6(1) (c) can be made the court must be satisfied that, on a balance of probabilities, there was a reasonable precaution whereby the death might have been avoided. It is clearly not necessary for the court to be satisfied that the proposed precaution would in fact have avoided the accident or the death, only that it *might* have done. It is well-settled that the precaution must have been reasonable at the time of the death. As to what is meant by the use of the word "might", I was referred to the oft-repeated words of Sheriff B. Kearney in his determination of 17 January 1986 into the death of *James McAlpine* where he said:

> *The phrase 'might have been avoided' is a wide one which has not, so far as I am aware, been made the subject of judicial interpretation. It means less than 'would on the balance of probabilities have been avoided' but rather directs one's mind in the direction of the lively possibilities.*

This definition of "lively possibility" has been applied with approval in many determinations since and I adopt it here.

[583] Thus it is clear that "might" must have meaning beyond a mere possibility. To suggest that a finding under section 6(1)(c) could be made simply on the basis that something was theoretically possible would render it entirely meaningless. It has to be remembered that such a finding in terms section 6(1)(c) represents a positive judicial determination. It also forms one of the principal findings under legislation which has as

one of its principal aims the future avoidance of fatal accidents and sudden deaths. Thus the term 'might' should be applied in a sense that incorporates the notion of something qualitatively more than a remote possibility: a possibility with some substance and potential rather than a fanciful or notional possibility.

Conclusions

[584] Addressing first the issue of whether Kathryn's bleeding could have been stopped in time to prevent her deterioration, I am satisfied, on the basis of the expert opinion that such an outcome was highly unlikely. While blood support might have reduced or slowed the bleeding, the universal view was that it would not have stopped it or prevented a re-bleed. Thus in the short time available, her deterioration could not have been avoided.

[585] Earlier diagnosis was unlikely to have happened before midnight at the earliest. Allowing for the timescales involved in order to commence treatment, any window of opportunity to correct Kathryn's coagulopathy would have been too small. ATRA would not have worked quickly enough. Even with prompt administration, it would have taken hours to work. Thus I was satisfied that in the time available – which was the narrowest of windows - the chances of Kathryn's deterioration being averted by stopping her bleeding or preventing a re-bleed were very slim and the chances of her surviving long enough to receive ATRA and for it to start to reverse her coagulopathy were negligible.

[586] On the matter of Kathryn's survivability generally, I preferred the evidence of Professor Hunt. I have already concluded that it was likely that there was excessive local

fibrinolysis happening in Kathryn's brain. As before, I found Professor Hunt's evidence highly persuasive. I considered that she was an outstanding witness, both in terms of her position of eminence, the cogency and fluidity of her evidence, the careful manner in which she considered the various aspects of Kathryn's condition and clear realism and common sense which complemented the academic aspects of her opinions. I agreed with Dr Hanley that of all the experts, Professor Hunt was the one best qualified to give an opinion on Kathryn's chances of survival.

[587] On the basis of her evidence, even when she first presented at the hospital, Kathryn's chances of survival having regard to her risk factors and the fact that she had already had a significant intracranial bleed were at best slim. The odds were truly stacked against her.

[588] When considered together with Kathryn's precarious neurological condition and the fact that she was on the steep part of the pressure volume curve with little scope to accommodate any rise in pressure from whatever source, I have concluded that Kathryn's chances of survival during her time in the Victoria were vanishingly small. I am satisfied that nothing could reasonably have been done that would have saved her.

[589] Accordingly, I have concluded that in terms of section 6, there were no reasonable precautions whereby Kathryn's death might have been avoided and no defects in any system of working that caused her death or made anything other than a contribution that was *de minimus*, that is, without legal consequence.

[590] Although I have concluded that no findings are appropriate in terms of section 6(1)(c) and (d), I have included certain matters under (e). These reflect the fact that there were a number of unsatisfactory elements of Kathryn's care and treatment at the Victoria Infirmary.

[591] Dr Beattie, in his detailed and voluminous written submissions, referred to other matters which were explored in the course of the Inquiry. I have given these my careful attention and have concluded that these are not matters which need form part of my determination.

Concluding remarks

[592] In conclusion, I wish to record my thanks to the procurator fiscal and the legal representatives for their helpful submissions and for their courtesy throughout the Inquiry. I include in that recognition of the difficult task undertaken by Dr Beattie in representing himself.

[593] These proceedings have been immensely difficult and harrowing for Kathryn's parents and her family. Their profound grief was tangible. It remains for me, once again, to express my condolences to them, in particular to Ms Crawford and Dr Beattie on the immensely sad loss of their much loved and cherished daughter, Kathryn.

ANNEX

LIST OF WITNESSES TO THE INQUIRY

1 Ms Jean Crawford, Kathryn's mother.

2 Dr Gerald Beattie, Kathryn's father.

3 Dr Rosaleen Beattie, Kathryn's aunt.

4 Mr James Fleming, Paramedic Advisor, Ambulance Service.

5 Dr Lucy Thomas, Consultant in Emergency Medicine and Paediatric Medicine. In June 2004, Dr Thomas was equivalent of an Acting Registrar (SHO3) in Accident and Emergency Department at the Victoria Infirmary, Glasgow.

6 Dr James Weir, Department of Anaesthesia, Crosshouse Hospital. Kilmarnock. In June 2004, Dr Weir was the Senior House Officer in General Medicine at the Victoria Infirmary, Glasgow.

7 Dr Jan Bunemann, Specialty Doctor in Rheumatology, Inverclyde Royal Hospital, Glasgow. In June 2004, Dr Bunemann was a Senior House Officer in the Haematology Department in the Southern General Hospital, Glasgow.

8 Dr Lorraine Duffy, Trainee in General Practice. In June 20104, Dr Duffy was the on-call Radiologist at the Victoria Infirmary, Glasgow.

9 Mr Alex McLauchlan, Senior Bio-Medical Scientist, Victoria Infirmary, Glasgow. In June 2004, Mr McLauchlan was the on-call Bio-Medical Scientist at the Victoria Infirmary, Glasgow.

10 Dr Howard McAlpine, Consultant Physician, Department of Cardiology, Victoria Infirmary, Glasgow.

11 Dr David Vernon, Consultant Physician, Department of Respiratory Medicine, Victoria Infirmary, Glasgow, now retired. Dr Vernon was the on-call Consultant for General Medicine in Victoria Infirmary, Glasgow on 21 June 2004.

12 Ms Claire McKie, Bio-Medical Scientist, Quality Training and Point of Care Testing Manager, Victoria Infirmary, Glasgow.

13 Dr Evelyn Teasdale, Consultant Radiologist, Department of Neuroradiology, Institute of Neurological Science, Southern General Hospital, now retired.

14 Dr Craig Urquhart, Consultant Anaesthetist. In June 2004, Dr Urquhart was a Senior House Officer in Anaesthetics on attachment to the Neuro-intensive Care and Neuro-anaesthesia Unit at the Institute at the Southern General Hospital, Glasgow.

15 Dr Ann Morrison, Consultant Haematologist, Southern General Hospital, Glasgow.

16 Mr Anwar Ahmad, Neurosurgeon. In June 2004, Mr Ahmad was a Senior Registrar in Neurosurgery at the Department of Neurological Sciences, Southern General Hospital, Glasgow.

17 Miss Jennifer Brown, Consultant Neurosurgeon, Institute of Neurological Sciences, Southern General Hospital, Glasgow.

18 Dr Dianna Raj, Specialty Registrar in Anaesthetics. In June 2004, Dr Raj was a Senior House Officer in Anaesthetics and Intensive Care at the Victoria Infirmary, Glasgow and was on call for Intensive Care.

19 Mr Tom Moffat, Bio-Medical Scientist, Technical Services Manager, Southern General, Victoria and Yorkhill Hospitals, Glasgow.

20 Dr Steven Jeffery, Consultant Anaesthetist, Royal Alexandra Hospital, Paisley. In June 2004, Dr Jeffery was a Specialist Registrar in Anaesthetics based at the Victoria Infirmary, Glasgow.

21 Mr Anthony Docherty, Bio-Medical Scientist, Head of Component Laboratory, SN Blood Transfusion Service, based at Gartnavel General Hospital, Glasgow.

22 Dr Ian Macdonald, Consultant Haematologist, Victoria Infirmary and Southern General Hospital.

23 Dr Douglas Walker, Consultant Anaesthetist, Neuoranaesthetics, Institute of Neurological Sciences, Southern General Hospital, Glasgow.

23 Dr Pamela Docherty, Consultant Anaesthetist, Victoria Infirmary, Glasgow. In June 2004, Dr Docherty was a Specialist Registrar based at the Neurological Institute at the Southern General Hospital, Glasgow.

24 Mr Robin Johnston, Consultant Neuorsurgeon, (Retired), Institute of Neurological Sciences, Southern General Hospital, Glasgow.

24 Mrs Rhona Twaddle, Enrolled Nurse, Neurological Intensive Care Unit, Southern General Hospital, Glasgow.

25 Mrs Patricia Rooney, Registered Nurse, Neurological Intensive Care Unit, Southern General Hospital, Glasgow.

26 Dr John Davidson, Consultant Anaesthetist, (Intensive Care Medicine), Victoria Infirmary, Glasgow.

27 Mrs Kirsty Allen, Senior Charge Nurse, Accident & Emergency Department, Victoria Infirmary, Glasgow.

28 Dr Ronald Sharp, Consultant Haematologist, Victoria Infirmary and Southern General Hospital, (Retired).

29 Miss Karen Tait, Registered Nurse, Department of Accident & Emergency, Victoria Infirmary, Glasgow.

30 Mrs Melanie White, Registered Nurse, Accident & Emergency Department, Victoria Infirmary, Glasgow.

31 Mr William Main, Charge Nurse, Neuro-intensive Care Unit, Institute of Neurological Sciences, Southern General Hospital, Glasgow.

32 Mrs Patricia Bienkowski

33 Miss Alison Taylor

34 Miss Natalie Laine

35 Mr Gerard McKenna

36 Mr John McKenna

37 Miss Carol Cooke

38 Mrs Theresa Bruce

39 Dr Douglas Bruce

40 Mr James Rae, Haematology Manager for South Glasgow.

41 Mrs Patricia Wallace

42 Mr Liam McLaughlin

43 Miss Abigail Parkins

44 Ms Lorraine Lane

45 Mrs Linda Donaldson

46 Mrs Phyllis Burke

47 Mrs Christine Small

EXPERTS

48 Mr Connor Mallucci

49 Professor David Grimwade

50 Professor Beverley Hunt

51 Professor John Liu-Yin

52 Dr Wellesley Forbes

53 Dr John Hanley

54 Miss Lynn Myles

55 Dr Janet Keeley

A statement of evidence was presented by the procurator fiscal in connection with timeline of the investigations.