This is a third witness statement by

Dr Malcolm Coulthard

in relation to the inquiry into the death of

Adam Strain

15/03/2011

Introduction

I wrote a report on 04/08/2010 giving my professional opinion about the cause of death of Adam Strain. I concluded that he died of cerebral oedema which had been induced by the changes in his plasma osmolality which resulted from inappropriate fluid management during his transplant operation.

I was asked to clarify some of my answers in this first report, and to answer additional questions, which I did in my second report on 04/12/2010.

When I wrote both of these reports I had had access to a summarised version of Dr Taylor's interviews with the police service, made under caution, and I commented specifically on them in the first report, pages 33 and 34. Since then, I have been provided with the full transcript of this day of police interviews. This has allowed me to gain a much fuller understanding of the arguments that he presented on 17/10/2006 in his defence that Adam's death was not due to cerebral oedema.

Although my two reports to date have dealt with many of the points that Dr Taylor has argued, these issues were interspersed among many other detailed debates which did not relate specifically to his position.

For this reason I have written this third report. Its purpose is to attempt to clarify the arguments that I believe Dr Taylor specifically relies upon to reach his conclusions about Adam's death. In it, I list the points which he presents in the police interviews to support his case, and I then present each point in turn along with evidence which I believe refutes it. To minimise repetition, where I have already presented a full, long discussion about a particular point in a previous report, I will merely summarise it here, and refer back to the earlier statement.

13 key arguments which Dr Taylor presents in his police statements on 17/10/2006 to reach his conclusions about Adam Strain's cause of death:

1) Dr Taylor states that Adam's urine losses were a minimum of 200 ml hourly, and he speculates that they had no known upper limit, suggesting at one point that they could, for example reach as high as 500 ml in an hour. To illustrate this point, he repeatedly described Adam's kidneys as being like a bucket with holes in, or like a sieve, which would simply leak out whatever volume was put into the body, specifically using the word unlimited to describe his kidneys' lack of upper limit to making urine.

2) Dr Taylor states that Adam's urine sodium concentrations were known, over the years, to be in the range of 30 – 50 mmol/l.

3) Dr Taylor explained repeatedly that he reviewed and regulated Adam's fluid intake to keep up with the losses from his kidneys.

4) Dr Taylor's interpretation of Adam's CVP recordings is that it only rose moderately by 3 mmHg from 17 to 20 during the operation.

5) Dr Taylor states that he expects pulmonary oedema to occur during most transplant operations, suggesting that this is a normal consequence of this surgical procedure.

6) Dr Taylor identified 0.18% saline (1/5 Normal saline with 4% dextrose) as being the fluid recommended for treating dehydration by the British National Formulary (BNF), and included a copy of the relevant page in his testimony.

7) Dr Taylor repeatedly states that he knows Adam's medical history, problems, and unique responses to fluids particularly very well.

8) Dr Taylor explains that the fact that Adam had previously been given 300 ml of 0.18% saline over 1 hour without ill effects was good evidence that he could handle 500 ml over ½ hour.

9) Dr Taylor argues that the common practice of resuscitating ill children with 1 or 2 boluses of 20 ml/kg fluid is evidence that his fluid management was not extreme or unusual.

10) Dr Taylor explained that there was no other strength fluid available that would have allowed him to administer Adam both sodium and sugar.

11) Dr Taylor explained that 0.18% saline is effectively like an isotonic solution in a child with a low sugar metabolism.

12) Dr Taylor explains that osmosis is a process in which salt passes from areas of high concentration to areas of low concentration.

13) Dr Taylor explains the delays and difficulties of getting laboratory tests done, along with it being a lower priority for him than his many other tasks, blaming lack of theatre staff, difficulty in getting porters, and getting lab workers to come in and do a blood test.

Evidence which I believe contradicts Dr Taylor's specific arguments or statements:

Point (1) that Adam's kidneys had no upper limits to urine flow in response to being given fluid.

Both Prof Savage and I estimate that Adam's urine output is about 1.5 litres daily. The evidence for this is based on his measured fluid daily intakes, and the simple deduction that people need to pass as urine an equal volume of fluid to remain in balance, minus the volumes passed insensibly by evaporation from the skin and lungs, and the tiny amounts lost in stool. This evidence is given in detail on pages 5 & 6 of my second report. A rate of 1.5 litres daily give an average hourly urine loss of 62.5 ml hourly, less than one-third of the rate which Dr Taylor states is Adam's minimum rate of urine loss.

It is not at all clear where Dr Taylor finds his evidence to support his figure of 200 ml/hour. I was unable to find any reference to this figure in his records other than in statements made by Dr Taylor himself. Although some children do have such enormous urine outputs in certain conditions, such as nephrogenic diabetes insipidus (NDI), they never go un-noticed or un-recorded when they occur. To put it in perspective, 200 ml every hour amounts to 4.8 litres, or over a gallon in a day. Scaled up for a 70 kg man this would be over 3 gallons. Boys with NDI typically wake every hour or two every night to drink and void, and are always seen clutching a large bottle of water to keep up with losses. When this occurs, it is very dramatic, and a major management problem. The descriptions of Adam's condition throughout his notes do not correspond with this clinical picture at all.

Kidneys produce urine through two sequential processes. First they filter fluid from the plasma, and then they reabsorb almost all of the water back into the blood-stream again, leaving the concentrated remaining fluid as urine. In end-stage kidney failure (when the kidneys do so little work that the patient needs to have dialysis or a kidney transplant to support them), the kidneys typically leak a larger proportion of the filtered fluid as urine. Rarely, this reaches as much as ⅔ of the filtered fluid in an extreme case. In the first report, I estimate that Adam's GFR, the rate at which his kidneys were able to filter fluid from the plasma, was slightly less than 4.8 litres per day. This means that his maximum capacity to produce urine can be calculated (contrary to Dr Taylor's claims). If we take the extreme case that he might have leaked as much as ⅔ of his filtered volume, it would amount to 3.2 litres per day, or 133 ml per hour, but would be more likely to be much lower than this.

On the above considerations, it can be seen that a rate of 200 ml/hour would be unattainable for Adam, and certainly that volumes as high as 500 ml/hour are 2½-times greater than his kidneys could filter fluid at all.

This is a fundamental point. Throughout his justifications for his decisions to administer high volumes of fluid, Dr Taylor repeats the assertion that his reason for doing this was because Adam had an almost unlimited and incalculably high ability to pass urine. This is simply not possible. The assertion is untrue, and there is no evidence to support it anywhere in his medical records.

Point (2) that the ranges of Adam's urine sodium concentrations were known over the years.

Dr Taylor's statement that "the salt content of his urine [had been] looked at over a number of years" is not true. Neither was his statement that "All his life he's passed" urine with a known low sodium concentration. Adam's urine sodium concentrations were only ever measured between November and December of 1991, when he was 4 months old. At that stage his kidneys were still functioning relatively well.

He did not die until 4 years later, during which time his kidney function had declined to reach end-stage, and he had needed to be treated with dialysis. It would be typical for a child's urine sodium concentration to gradually increase during this process. However, it was not measured at any point during this time. It was simply unknown.

Despite this, he claims that he relies upon Adam's background urine sodium concentration information to determine which fluids to provide ... "of similar nature to his renal losses."

Point (3) was that Dr Taylor claims that he reviewed and regulated Adam's fluid intake to keep up with the losses from his kidneys.

This is not true, according to the evidence in Adam's case records, and this is perhaps the most crucial point of all.

Throughout his interview, Dr Taylor stressed the role of the anaesthetist in continuously monitoring aspects of the patient's condition. He mentioned checking various vital signs on the monitor and responding to them as required. However, for somebody undertaking an anaesthetic on a child who they believe to have an extra-ordinarily high output of excessively dilute urine, whose fluid administration strategy was based on these assumptions being true, it appears that he did not monitor the obvious and simple parameter of the urine output (let alone confirm that its sodium concentration was as low as he presumed). During the whole anaesthetic there was no record of his urine output, against which all the fluid was apparently being titrated, being measured or checked in any way at all. How were the regular re-assessments undertaken of his fluid status throughout the procedure, as stated by Dr Taylor, if he did not have any information about the biggest and most important variable, the urine output?

The first record in Adam's notes of his urine output on the day of his transplant surgery was made at the end of the procedure, when he had already developed cerebral oedema and could not be woken up, when a total urine volume for the entire time was measured and recorded as 49 ml.

If Adam had voided at Dr Taylor's predicted minimum rate of 200 ml each hour, then during 4½ hours of surgery he would be expected to have voided more than 900 ml, that is nearly 20-times higher than he has continued to insist must have been produced in that time, and which he felt certain needed to be replaced. 900 ml is a very large volume in a lad of Adam's size, and would have been expected to hugely dilate his bladder, such that it would have bulged up into his abdomen and created an obvious swelling. This would have been an impediment to undertaking transplant surgery, which involves exposing the large arteries and veins in the lower abdomen, behind the bladder, making space for the kidney to be bedded into adjacent to the bladder, and incising the bladder wall itself to join the donor's ureter (urine drainage tube) to.

For both of these reasons; that is to prevent it becoming dilated and to allow the critical value of the urine output to be monitored, it would have been appropriate to catheterise Adam's bladder prior to commencing surgery (at the same time as setting up all the other vital anaesthetic monitoring arrangements), or at the very least for it to have been catheterised by the surgeons as they began their laparotomy. Bladder catheterisation is not a major procedure in an anaesthetised child, much less difficult and invasive than other procedures he underwent, such as placement of a central venous catheter, or an arterial line.

Point (4) was that Dr Taylor states that Adam's CVP only rose moderately by 3 mmHg from 17 to 20 during the operation.

My reading of the CVP trace is quite different from this. On the copy of the record that I have seen, it started at the relatively high level of about 17, and then approximately doubled to the extremely high values of 30 and greater during the procedure. I cannot understand how the trace I have seen could be interpreted in the way that Dr Taylor states.

Point (5) was that Dr Taylor considers pulmonary oedema to accompany most kidney transplant operations.

This would not be my experience, and I do not believe this view would be shared widely at all. This is dealt with in some depth on page 14 of my second report. In summary, it is certainly true

that there is a well recognised risk to establishing the blood flow to a newly transplanted kidney if the patient is allowed to become at all dehydrated, and as a consequence there is a rational policy for administering generous volumes of fluid rather than withholding it. However, the risk of pulmonary oedema occurring with fluid overload is so well known that the balance of fluid administration and measured losses, especially of urine, is usually monitored with great precision. As a result, the clinical diagnosis of pulmonary oedema is rare under these circumstances.

My personal experience is of seeing 2 children with this complication out of a total of approximately 200 transplant operations. Both children made uneventful recoveries, but in both cases we undertook a case analysis afterwards to try to identify the specific reasons for this problem occurring, in an attempt to learn from it. To consider it almost acceptable, a reasonably likely outcome of surgery, is in my view unacceptably lacking in critical appraisal.

Point (6) was that Dr Taylor identified 0.18% saline (1/5 Normal saline with 4% dextrose) as being the fluid recommended for treating dehydration by the BNF.

Dr Taylor claims that the page he supplied in his testimony (BNF number 29, March 2005) indicates that 0.18% saline + 4% dextrose "mixture is recommended for dehydration", but this is not true. Under replacement therapy (that means replacing a fluid deficit or level of dehydration) it says that this "may be given as NaCl 0.9% for initial fluid bolus in acute fluid loss." To clarify, Dr Taylor alleges that the BNF recommends using one-fifth normal saline, whereas in reality it recommends using normal saline.

The second recommendation on that page is about maintenance fluid therapy. There, it states that "subsequent fluid and electrolyte requirements are determined by clinical assessment of fluid balance, including *measurement of ongoing renal and extra renal losses*, and measurement of plasma electrolytes ... where appropriate." (my italics). What this is saying is that an important way to determine the right volume and strength of fluid to use to maintain a patient in a stable state is to measure the volume and concentration of the urine (and sometimes other fluids), and choose the nearest match to it. In routine more minor surgery on patients with normal kidney function, it would not be necessary to collect and measure urine volumes and strength, but in transplant surgery in a child thought to have a bizarre fluid handling problem, it would clearly be essential to address this issue.

It has already been established in points 1, 2 and 3 that Dr Taylor did not either establish the correct historic information about Adam's urine output (about 1.5 litres daily), nor did he measure its volume at all during the surgery while administering huge volumes of fluid, nor did he measure Adam's urine sodium concentration during surgery, and nor did he know its usual value because it had not been measured since Adam had been a little baby.

Despite misquoting the BNF page that he supplied, he does appear to understand the basic concept that it is appropriate to replace each fluid loss with its closest equivalent fluid. As evidence of this, he states that "We also recognised that there would be the need to replace the type of fluid lost by the body by that type of fluid which it most closely resembled ie replace water with water, salt with salt, and blood with blood." Unfortunately he appears to have considered it sufficiently accurate to guess rather than measure the volume and salt concentrations of urine which Adam produced during the operation.

I note that on interview tape T0175882A, end of page 7, beginning of page 8, that his statement gives the impression that he was monitoring the urine output (though he was not). He says "We were reasonably satisfied towards the end of the second hour of surgery that the renal losses were now adequately replaced and therefore erected a third bag (500 mls) of 0.18% nacl/4% glucose to be given at a much reduced rate over the next 2 hours 20 minutes to maintain the loss of dilute urine by Adam's native kidneys." It would appear that he was referring to the losses he imagined Adam might have had, based on his false assumption that he always voided at least 200 ml each hour.

Point (7) was that Dr Taylor repeatedly states that he knows Adam's medical history, problems, and unique responses to fluids particularly very well.

Despite claiming to know Adam's medical details particularly well, Dr Taylor refers to him throughout as having an entirely wrong diagnosis.

Adam had dysplastic kidneys, while Dr Taylor consistently describes him as having had congenital nephrotic syndrome. These 2 conditions are not only completely distinct and very different in many ways, but they also present quite different fluid balance problems at transplantation. This is because congenital nephrotic syndrome is associated with a low plasma albumin which makes hypovolaemia a particular risk during transplantation. Because of this, such children are typically not considered for a transplant until they have been on dialysis for a sufficiently long period for their urine output to virtually cease. Only then does the nephrotic element of their problem resolve.

Thus, children with dysplastic kidneys can have variable urine outputs by the time they require a kidney transplant, but children with congenital nephrotic syndrome almost exclusively only produce very small volumes of urine.

Point (8) was that Dr Taylor explains prior knowledge of Adam tolerating 300 ml of 0.18% saline over 1 hour allowed him to conclude that he would also tolerate 500 ml over ½ hour.

Dr Taylor reached the above conclusion by arguing that "normal children shouldn't cope with 300 mls over an hour", and thus making the point that Adam was different from normal children. Even if the first 300 ml infusion had marked out Adam as being different from other children, it is a non-sequitur to effectively conclude that he could receive fluid at more than 3-times the rate and still be safe.

Simple calculations, of the same nature as I have employed elsewhere in my reports, show that giving 300 ml of 0.18% NaCl fluid over 1 hour to a 20 kg child would be expected to lower the plasma sodium concentration by approximately 2 mmol/l, and therefore at a rate of 2 mmol/l per hour, while giving 500 ml over ½ hour would be expected to reduce it by 6 mmol/l, and thus at a rate of 12 mmol/l per hour. That is, during the first half-hour, it would disturb the plasma sodium concentration six-times more rapidly. It is clear that no safety conclusions can be drawn from the prior experience described of infusing 0.18% saline at a lower rate and volume.

Point (9) was that Dr Taylor argues that the common practice of resuscitating ill children with 1 or 2 boluses of 20 ml/kg fluid is evidence that his fluid management was not extreme or unusual.

The practice of resuscitating ill children with 1 or 2 boluses of 20 ml/kg of fluid (which indeed is common) is always with normal saline, or with fluids with a similar sodium concentration, such as plasma or Hartmann's solution. This is absolutely standard practice – please refer again to the recommendations in the BNF in point 6, which confirms this. Delivering a bolus of 0.18% saline is absolutely counter to all normal practice and recommendations, and is both extreme and unusual (and predictably dangerous).

Point (10) was that Dr Taylor explained that there was no other strength fluids are available that allowed the simultaneous administration of both sodium and sugar.

This is not true. A number of standardly manufactured fluid bags contain both sodium and glucose (sugar). These include half-normal saline (0.45% saline + 4% glucose) and normal saline (0.9% saline + 5% glucose). All of these are routinely available on our general paediatric wards and on our children's kidney wards, and have been throughout my whole clinical career.

Point (11) was that Dr Taylor claimed that 0.18% saline is effectively like an isotonic solution in a child with a low sugar metabolism.

There are 2 points to be made here. The first is to refute the statement that 0.18% saline is ever effectively like an isotonic solution when infused. I have dealt with this already on page 34 of my first report; in summary, as soon as the fluid mixes with blood, the glucose is diluted, and then either used by the body for energy or stored, and the solution almost instantaneously behaves as if it was a pure solution of salt and water, but with a sodium concentration only one-fifth of the value of physiological saline.

The second is that when he was justifying using a fluid which contained glucose (and implying incorrectly that this limited him to just one concentration of sodium), he argued that Adam had a particularly *high* requirement for sugar, and had to have this high infusion rate partly to satisfy that need. Here, he seems to be arguing that Adam had a particularly *low* rate of sugar metabolism, as if that would make 0.18% saline solution behave more like an isotonic one (which is also false).

Point (12) was that Dr Taylor explains that osmosis is a process in which salt passes from areas of high concentration to areas of low concentration

Dr Taylor states that "the [body's] organs contain salt and therefore the salt passes from the high concentrations to a low, the basic theory of osmosis."

This is simply not true. It is almost the opposite of the truth. Osmosis is a process which does not involve the movement of salt at all. Rather, it involves the movement of water *without* salt across a semi-permeable membrane from an overall weaker to an overall stronger solution. In the body, the strength of the solutions inside and outside cells and organs are made up by dissolved chemicals. Salt is prominent as a chemical which contributes greatly to the overall osmotic strength of the fluid outside of cells, and is at much lower concentrations inside cells, where other chemicals predominate. However, the basic mechanism of osmosis is the movement of water without its dissolved chemicals.

Point (13) was that Dr Taylor explains the delays and difficulties of getting laboratory tests done, along with it being a lower priority for him than his many other tasks, blaming lack of theatre staff, difficulty in getting porters, and getting lab workers to come in and do a blood test.

These statements are completely exaggerated and unreasonable.

Any hospital which contains a paediatric intensive care facility and undertakes paediatric transplantation will always require and have the infrastructure to provide laboratory electrolyte measurement at any time of day or night, with minimal delay. While all doctors will have experienced irritations with some of the processes at times, these are never an insurmountable block to obtaining important laboratory results. The operation took place during the routine working day – to suggest that there would have been difficulties in getting laboratory staff to undertake these routine tasks is without any justification. The same has applied throughout my entire career undertaking similar work.

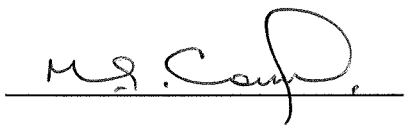
The sole reason why Dr Taylor had no laboratory electrolytes available to guide him with his ongoing fluid and electrolyte therapy by was because he did not take any blood for this purpose. It should be appreciated that one of the purposes of the central line which he placed in Adam was to enable easy blood sampling. Children's doctors describe them to children as being like a tap, and means that they do not need to have any needles while it remains in. Indeed, during the siting of the central line, blood will have been drawn into a syringe to test its patency – this could have been sent to the lab instead of simply being pushed back in.

Expert Witness Declaration

I Malcolm Coulthard DECLARE THAT:
1) I understand that my duty in providing written reports and giving evidence is to help the Court, and that this duty overrides any obligation to the party by whom I am engaged or the person who has paid or is liable to pay me. I confirm that I have complied and will continue to comply with my duty.
2) I confirm that I have not entered into any arrangement where the amount or payment of my fees is in any way dependent on the outcome of the case.
3) I know of no conflict of interest of any kind, other than any which I have disclosed in my report.
4) I do not consider that any interest which I have disclosed affects my suitability as an expert witness on any issues on which I have given evidence.
5) I will advise the party by whom I am instructed if, between the date of my report and the trial, there is any change in circumstances which affect my answers to points 3 and 4 above.
6) I have shown the sources of all information I have used.
7) I have exercised reasonable care and skill in order to be accurate and complete in preparing this report.
8) I have endeavoured to include in my report those matt ers, of which I have knowledge or of which I have been made aware, that might adversely affect the validity of my opinion. I have clearly stated any qualifications to my opinion.
9) I have not, without forming an independent view, included or excluded anything which has been suggested to me by others, including my instructing lawyers.
10) I will notify those instructing me immediately and confirm in writing if, for any reason, my existing report requires any correction or qualification.
11) I understand that;
    11.1) my report will form the evidence to be given under oath or affirmation;
    11.2) questions may be put to me in writing for the purposes of clarifying my report and that my answers shall be treated as part of my report and covered by my statement of truth;
    11.3) the court may at any stage direct a discussion to take place between experts for the purpose of identifying and discussing the expert issues in the proceedings, where possible reaching an agreed opinion on those issues and identifying what action, if any, may be taken to resolve any of the outstanding issues between the parties;
    11.4) the court may direct that following a discussion between the experts that a statement should be prepared showing those issues which are agreed, and those issues which are not agreed, together with a summary of the reasons for disagreeing;
    11.5) I may be required to attend court to be cross-examined on my report by a cross-examiner assisted by an expert;
    11.6) I am likely to be the subject of public adverse criticism by the judge if the Court concludes that I have not taken reasonable care in trying to meet the standards set out above.
12) I have read Part 35 of the Civil Procedure Rules and the accompanying practice direction including the "Protocol for Instruction of Experts to give Evidence in Civil Claims" and I have complied with their requirements.
13) I am aware of the practice direction on pre-action conduct. I have acted in accordance with the Code of Practice for Experts.

Statement of Truth

I confirm that I have made clear which facts and matters referred to in this report are within my own knowledge and which are not. Those that are within my own knowledge I confirm to be true. The opinions I have expressed represent my true and complete professional opinions on the matters to which they refer.

Signed  Dr Malcolm Coulthard

Dated 15/3/11 15/03/2011

Dr Malcolm Coulthard, BSc, MB BS, DCH, FRCP, FRCPCH, PhD