This is a report by

Dr Malcolm Coulthard

requested by the *Inquiry into Hyponatraemia-Related Deaths*

in response to the further supplementary brief of 07/02/2012, including a response to the document received from Dr Taylor, dated 01/02/2012

16/02/2012

Remit

I have been sent a copy of Dr Taylor's report of 01/02/2012 by the *Inquiry into Hyponatraemia-Related Deaths* team to comment upon. I will duplicate their questions and present my replies in order.

I have also been sent a composite table of sodium and water balances during the peri-operative period, as completed by myself, Dr Haynes, Prof Gross, and by Dr Taylor on the basis of his present views as presented in his recent report. I have been asked to compare and comment upon these. I have also been asked to consider how Dr Taylor's earlier statements about his fluid management compare to these.

FURTHER SUPPLEMENTAL BRIEF FOR
EXPERT ON PAEDIATRIC NEPHROLOGY

ADAM STRAIN

Introduction

1. Thank you for your previous reports in respect of Adam Strain. The Inquiry Team would be grateful for your comment or opinion on the following issues:

Dr. Robert Taylor's sixth Witness Statement to the Inquiry

2. Please find attached Dr. Robert Taylor's latest witness statement to the Inquiry. The Inquiry team would ask that you reflect on this statement and address the following:

   (1) The extent to which you agree or disagree with the points made by Dr. Taylor

   (2) The significance of Dr. Taylor's statement for the issues to be considered by the Inquiry

   (3) The consistency of Dr Taylor's comments with his previous statements, his deposition to the Coroner and his PSNI interview under caution

I note on reading Dr Taylor's witness statement of 1st February 2012 that, for the first time since 1995, he appears prepared to accept that he made some mistakes in his management of Adam Strain. I have answered the sub-questions 2(1) to 2(3) by responding to each paragraph in Dr Taylor's letter in order, below:

Para 2, beginning "Adam was ..."
Here Dr Taylor sets out his experience of paediatric anaesthesia at the time of treating Adam, and states that he felt that he "had the necessary training and experience to undertake this case".

I agree that his training should have given him sufficient expertise to manage Adam's case appropriately and safely. I will enlarge on this point in response to the question below on the significance of the renal transplant surgery.

Para 3, beginning "I was first ..."
Here Dr Taylor states that although it is (and presumably was in 1995) his usual practice to see patients before their operation, he did not visit Adam prior to him arriving in the anaesthetic room for his transplant operation.

He also states that he does not recall whether he discussed the risks of the anaesthetic with Adam's mother. Since it would neither be appropriate nor possible to do this with her in a meaningful way whilst she was accompanying Adam into the anaesthetic room (as he was about to be anaesthetised for major surgery), Dr Taylor's statement effectively excludes him having provided her with a fully informed or timely description of the risks.

Para 4, beginning "At the time ..."
There are 3 separate issues raised in this paragraph; point (i) relates to the volume of urine that Adam would pass each hour; point (ii) concerns the fixed nature of his urine output; point (iii) concerns whether the fluid regimen that Dr Taylor used would have been appropriate for any of these possible situations.

Para 4, point (i). Dr Taylor now admits that he "made the assumption that [Adam] would pass around 200 mls (sic) per hour of dilute urine". Until now he has attempted to justify his strong assertion that this was Adam's regular hourly urine volume on the basis of (a) his previous experience of anaesthetising Adam, (b) information available in the notes, and (c) information he gained during a telephone call with Dr Savage during the evening before the transplant.

In my previous reports I had concluded that Dr Taylor's assertion that Adam voided 200 ml/hour had no rational basis, and that his genuine urine output would have been approximately 62 ml/hour. Dr Taylor now confirms that it was an unsubstantiated and mistaken assumption, and that he now believes that the correct rate should have been approximately 70-80 ml/hour. 80 ml each hour would total 1,920 ml per day. This is a larger volume than Drs Savage had suggested more than once in Adam's records (about 1,500 ml daily), or than Dr Haynes, Prof Gross or I have estimated from the same evidence since. I am not certain how he has now reached this slightly higher figure, but reviewing his updated assessment of Adam's fluid balance (see comments on tables below), it appears likely that he may have assumed that all of his 'renal' losses are from the kidneys, with no contribution from his dialysis. However, 70-80 ml/hour is clearly *much* closer to my estimated figure of 62 ml/hour than his original value of 200.

Para 4, point (ii). Dr Taylor now admits that Adam had a fixed urine output. This is critically important, and it is the first time that he appears to have accepted this.

It is important to clarify what is meant by a fixed urine output. In health, the kidneys are capable of increasing or decreasing the volume of urine they produce each hour over a very wide range. This allows people to drink large quantities of fluid, and to excrete the excess and to avoid their bodies from becoming water overloaded. They are also able to conserve water very avidly if they lose fluid excessively and are unable to drink to correct their fluid depletion, thereby allowing them to survive much longer without becoming dehydrated.

People with kidney failure lose the ability to increase or decrease their urine volume according to their body's requirements. When they reach the stage that they require dialysis support, there is seldom any responsiveness to changing fluid intakes.

- In most adults and a minority of children on dialysis, the urine output is fixed at zero; they are unable to excrete any water they ingest, and simply retain all of it that is not lost as sweat and in stools, and they cannot alter their degree of water conservation as it is already maximal.
- A few adults and most children on dialysis pass a steady amount of water each hour as dilute urine (in Adam's case at about 62 ml), which does not change as their fluid intake alters. If they drink or are administered more fluid than usual, they retain the extra. If they have no access to water, or cannot drink because they have a vomiting problem, or are otherwise too unwell, or are anaesthetised, or if they become lost in a desert, they will continue voiding urine at the same high rate and thereby will rapidly become dehydrated.

Dr Taylor's previous statements in relation to Adam's urine output made it clear that while he *did* consider it to have a fixed lower volume limit, he *did not* consider him to have any discernable upper limit. Thus, he considered that for every hour that he was left without at least 200 ml of fluid administered he would become dehydrated, and would need for this to be corrected by administering an equal volume of 0.18% saline, but that it would be impossible to produce dilutional hyponatraemia in Adam by giving him extra water because his urine volume would simply increase to deal with the excess. His analogy was of trying to fill a bucket with large holes in it.

Para 4, point (iii). Dr Taylor now states that "The intraoperative fluid [he] administered was based on this incorrect assumption and [he] therefore administered a hypotonic fluid, 0.18NaCl/4% Glucose, at a rate in excess of his ability to excrete it, particularly in the first hour of anaesthesia, 07.00-08.00".

This is a complex statement which consists of 2 logical assertions, one explicit and one implied. Both assertions have to be true for it to be correct, and this is not the case.

- The first (explicit) assertion is that he based his fluid prescription on the incorrect assumptions he had made about Adam urine output. I accept that this was the case.
- The second (implicit) assertion is that the fluid regimen he used would have been appropriate for a child who *did* have the fluid handling characteristics that he had incorrectly ascribed to Adam.

The second point is certainly not true. Dr Taylor's approach to Adam's fluid management was fundamentally flawed. He did not consider the dramatically fast fall in the plasma sodium concentration that would inevitably result from such sudden infusion of dilute fluid, an event that is known to be dangerous. This issue is dealt with in the section below in which the accumulation of free water is calculated and graphed, using both sets of assumptions that Dr Taylor used – the ones at the time, and the ones he now considers to be correct.

In previous reports I have dealt with the principles that underline the correct approaches to managing the fluid requirements of an unconscious child, and I also refer to them later in this report. Here I will merely summarise a fundamental fluid prescription error that he made, which would always be wrong, regardless of a child's kidney function. At the commencement of the anaesthetic Dr Taylor assumed (I believe incorrectly) that Adam was fluid depleted as a result of his prior management, and sought to rectify this by administering an extra volume of intravenous fluid, "at a rate in excess of his ability to excrete it". He chose to do this with a fluid whose sodium concentration was just a fraction of that in his plasma. It is a physical fact that in any circumstances, adding a volume of a weak solution to a stronger one will dilute it. Planning to employ a solution containing just 31 mmol of sodium per litre to increase the volume of plasma which contains sodium at 135-145 mmol/l will inevitably lower this, and will guarantee to produce hyponatraemia. This is true whatever the particulars of that child's renal function.

Para 5. "When I commenced ..."

Dr Taylor now blames his failure to have collected a blood sample for biochemical analysis at the commencement of surgery on being too pre-occupied with other duties, rather than continuing to argue that it was a considered decision based upon the inadequacies of the infrastructure needed to obtain emergency biochemistry in those circumstances. I have previously argued that his earlier stance was unreasonable, so I consider his new position more appropriate.

However, the manoeuvres which Dr Taylor lists as diverting him from the task of sampling blood are the routine procedures required when inducing a general anaesthetic (GA) in any child requiring major surgery. The impression could be gained from Dr Taylor's statement that the tasks on that particular day were especially onerous, perhaps in the light of it being a transplant operation, or being under particular time pressures, etc, but this would be misleading. Such tasks at the start of all GAs for major surgery in children would not normally be considered sufficient to divert the anaesthetist from taking a planned blood sample, nor to prevent him from doing so a little later, or indeed to prevent him from doing so at any point during the operation, as happened here. This is especially so if the consultant had a junior anaesthetic medical colleague assisting, and especially so if he sited a central line during the preparatory phase as that inherently involves drawing a blood sample into a syringe to test the flow through the line.

Furthermore, changing the explanation from infrastructure failures to becoming diverted by the intensity of his other tasks does not explain his failure to send a blood sample in response to the point-of-care sodium concentration result during surgery which indicated an alarmingly low level.

Thus, for me, the reason(s) why Dr Taylor failed to send any blood specimen to the biochemistry laboratory throughout the entire operation remain(s) unexplained. I agree with his current position that the fault lay with him, but do not accept that being too busy to attend to it is a reasonable justification.

Para 6. "The reliability of ..."
This paragraph discusses 2 issues, his responses to Adam's central venous pressure (CVP) recordings, and his responses to the "Arterial Blood Gas (ABG) sodium level". I will deal with them separately.
Sodium I have already referred to Dr Taylor's failure to send a specimen to the laboratory to check the plasma sodium concentration in the hospital laboratory in my response to his paragraph 5. In that section, I have used the generic term 'point-of-care' analysis to indicate a screening assay undertaken by clinical staff close to the operation, so this is synonymous in this case to the ABG level that Dr Taylor is referring to.

In the first sentence of paragraph 6, Dr Taylor states that he had concerns about the reliability of *both* the CVP and the ABG sodium and that he paid less attention to *them* than he should have done. In the last sentence, he goes on to conclude that he recognises "that *this* led to a lower standard of care than [he] would normally provide" (my italics). However, between these 2 sentences he talks about issues which appear to relate only to the CVP, and I am therefore left uncertain whether my italicised *this* refers only to his management of the CVP, or whether he accepts that his decision not to confirm the plasma sodium concentration also led to a lower standard of care. This is an important distinction. I believe that both aspects of management lowered Adam's standard of care.

CVP I refer to a report which I completed very recently (10/02/12) in response to a report by Dr Dyer dated 24/01/12. This concerned the potential impact that Adam's raised CVP may have had on the perfusion of blood in his brain, but also clarified the validity of the measurements made from him. In his report, Dr Dyer points out that Dr Taylor's statement on 011-002-006 (repeated on 011-014-099) included the sentence that "There were both cardiac and respiratory patterns to the waveform confirming correct intravascular placement."

This statement confirms that Adam's CVP reading was indeed a valid measure of the status of his central veins. This is the ultimate test of the direct continuity of a fluid path between the tip of the measuring catheter and the blood in the large veins of the chest. If this was obstructed, or if the catheter had been occluding the vein it was situated in, it would instead have been recording the pressure distal to that, and these dynamic transmissions from the chest would not have been present.

Dr Taylor's decision "that [he] was unable to trust" the CVP reading after he "felt the CVP catheter in Adams (sic) neck and was therefore convinced that it was not in continuity with the great veins draining the heart and therefore could not be relied upon" was therefore definitely wrong. Rather than deciding "to pay them less attention than [he] should have" (that is, ignoring this important warning information), Dr Taylor should have taken quite different actions. Dr Dyer is absolutely correct in his "belief that it is mandatory for the clinician to investigate the cause" of any such apparently abnormal finding.

The correct action for Dr Taylor to have taken would have been to recognise that the presence of the cardiac and respiratory waveforms indicated that the pressure transducer was reflecting genuine pressure changes in the veins within the chest. Given the height of the CVP, he should then have made certain that there was not a technical problem with the recording equipment. I would suggest that this should initially have been to carefully repeat the calibration of the zero pressure (by opening the transducer to air pressure whilst held at the correct vertical level in relation to Adam's chest). If there was still doubt, the electronic recording equipment could be tested at once by switching the leads from the arterial blood pressure transducer and the CVP line to see if they both registered the same values. If these tests confirmed that the reading was indeed genuine, then Dr Taylor should have used this vital information to reassess Adam's cardiovascular and fluid-balance status. This

would have led him to recognise that Adam's circulation was already well filled, an opposite conclusion from the one he guessed and treated.

For completeness, I will give my view about the action that Dr Taylor should have taken if, instead of being patent as it was, Adam's central line had been lodged into a vein and obstructed it. Under such circumstances, which I have occasionally experienced, I would expect the anaesthetist to insert his central line elsewhere, such as into the inferior vena cava through one of his femoral veins (the main vein draining one of his legs). This decision would need to be discussed with the transplant surgeons, as it would be ideal if the femoral vein was used on the opposite side to the planned kidney insertion.

To allow the transplant to proceed in a child without first ensuring a valid CVP measurement that could be relied upon would always be a mistake in my opinion. The trade-off of starting the operation more quickly, but without a valid CVP, would always be strongly disadvantageous.

Para 7. "I recognise that ..."
There are two aspects of this sentence that I have to comment upon, the detail of the physiology, and the potentially misleading significance of capitalising the words Dilutional Hyponatraemia.

The physiology
It is wrong to say "that the administration of excessive volumes of hypotonic fluids ... *can* produce a movement of water into the cells of the body" (my italics). It is a matter of fact that the physical processes that govern the movement of water molecules across cell membranes, including the force of osmosis, mean that the excessive administration of hypotonic fluids *will always* cause water to move into the inside of the body's cells. This is a guaranteed and utterly predictable consequence of the physics of the molecules involved, and is not a matter of 'medical opinion'.

It is also a simple fact that the inevitable event of water moving into cells under these conditions and increasing their volume *will* inevitably increase the volume of the brain because the main bulk of the brain tissue is cells. It is also correct to say that the swelling of the brain *may* be of sufficient degree that it *can* lead to clinical consequences, including its pressure increasing within its restricted housing (the skull), known as cerebral oedema. Finally, the cerebral oedema *may* be sufficiently severe as to diminish the cerebral perfusion pressure, and lead to lack of oxygen supply, brain damage, or even brain-stem death.

I believe that it is vitally important to emphasise that infusing an excess of hypotonic saline is always potentially hazardous because it inevitably leads to brain swelling, and that whether that results in cerebral oedema or not in a particular patient depends on a wider range of other factors, including the rate of infusion.

"Dilutional Hyponatraemia" (as compared to "dilutional hyponatraemia")
It is a matter of fact that if you take a strong solution and add some weak solution to it, it will become diluted. If the solution in question was one of table salt in water, it is also a matter of fact that the concentration of the sodium ions will be diluted to a level lower than its starting value. If the solution was plasma, and the initial sodium concentration was in the normal physiological range (isonatraemic), then the addition of a weaker salt (hypotonic) solution would always cause relative hyponatraemia (a lower sodium concentration) compared to it starting value. The plasma sodium may fall sufficiently far to reach a level not normally seen in healthy patients (hyponatraemia). It is therefore true to say that adding an excess of a hypotonic solution into the blood stream by infusing it will always produce a relative dilutional hyponatraemia, and sufficient infusion may produce an absolute dilutional hyponatraemia.

Note that the above paragraph does not contain any capital 'D's or 'H's; they are not needed to explain the physiological events occurring. When Dilutional Hyponatraemia is used in its capitalised form, it could suggest that it was being used in a way which was meant to mean more than the simple description of the undeniable events described above, such as a specific diagnosis or syndrome. I am aware that both 'Dilutional Hyponatraemia' and 'dilutional hyponatraemia' have been used in some witness statements in exactly that way.

For example, in his police witness statements, Dr Taylor refers to dilutional hyponatraemia as "only a theory" ... "No one's actually proven it", as if it was merely a hypothesis, and one that was "difficult to investigate" and which had never been tested by a "double-blind research trial". He also points out his views there that Arieff's extensive work in this area was so specific that it did not relate to children such as Adam (which is not true).

The attitude of paying little attention to predictable physiological changes, and to deny their relevance or importance unless they have been documented in a particular clinical scientific paper pertaining to a specific subset of patients, is unhelpful. I am not sure whether Dr Taylor's capitalisations in his most recent report were used intentionally to continue to help make that point, or if it was an unintended typographic error. However, it is important that this does not inadvertently give the sentence an inappropriate meaning or more weight than it would otherwise deserve.

Para 8, "Since this case ..."
It is good to know that Dr Taylor no longer uses 0.18% saline to administer fluid boluses. However, I am confused by his claim not to have done this since Adam's case, and cannot understand why this should be the case since he has been defending his management ever since, until this latest report. This is most dramatically seen in the transcripts of his interviews with police under caution over 10 years after Adam's death, where he seemed to be robustly justifying the fluid management he had used. Perhaps he had not come across another child with a fixed urine output since, though this seems unlikely during many years of paediatric anaesthetic practice.

Para 9, "I deeply regret ..."
The use of the term 'calculations' in this sentence deserves some comment. Throughout his testimony, Dr Taylor has referred to his fluid balance 'calculations' for Adam, and has repeatedly indicated that they were complex in nature. I take a completely different view, and one thrust of my teaching over the years has been to try to demystify how simple the principles are when making assessment of fluid requirements and other prescribing issues in paediatric renal failure. What is required is great attention to detail and the ability to estimate, but I would go as far as to say that I do not personally consider the term 'calculations' to be the most appropriate in this context.

To explain (as I have done elsewhere in my witness reports), I will outline how to prescribe fluids for a child undergoing a transplant operation. I am assuming (as was the case for Adam) that the child arrives in theatre in approximate fluid balance:

- Water lost must be replaced by a similar volume of water, containing approximately the same concentration of sodium.
- The effectiveness of the replacement must be assessed continuously by making regular blood sodium measurements (minimum of 4-hourly during periods of change) and recording the volumes of urine samples, and ideally of their sodium concentrations at the same time as the blood is tested, at least initially.
- The body loses water through sweat and exhaled breath (insensible losses), which contains very little sodium. Estimate the volume as 300 ml per day (approx 12 ml per hour) for each m^2 of body surface area (easily estimated from body weight using a table). Replace at that hourly rate with a hypotonic solution, such as glucose without sodium, or 0.18% saline with 4% glucose.
- Estimate the child's hourly urine losses by totting up his usual fluid intake over a typical day, and subtracting his approximate insensible and dialysis losses. This will roughly equal his daily urine output. Divide this by 24 to estimate his typical hourly output. Then infuse that quantity hourly with a solution containing a similar sodium concentration. The ideal way of choosing the best fluid to use is to measure the sodium concentration of a urine sample. In the absence of that, use ½ - normal saline (77 mmol/l) as this is the typical concentration seen in children with renal failure. Monitor the urine flow rate during surgery, especially if the child produces large volumes of urine, and the volumes being replaced are therefore correspondingly large, by catheterising the child and collecting the output in a calibrated 'box' under the operating table. If the urine output falls during surgery (as may happen if the blood pressure falls, for example), reduce the infusion rate accordingly.

- Replace measured blood losses with either blood or plasma or normal saline, depending on the amount of blood lost and the potential for anaemia developing. All of these replacements contain sodium at similar concentrations to plasma, so will not alter the child's levels.
- If any more fluid is needed, for example to correct a perceived prior deficit, or because the CVP is too low, use an isotonic solution, either normal saline or sometimes plasma, in sufficient quantity to correct the problem. Usually begin with boluses of 5 ml/kg unless the clinical situation is very urgent. Continue to monitor the physiological responses (pulse, blood pressure, peripheral perfusion, CVP) very frequently to determine further needs. Since all of this fluid will be retained in the body at least for the next few hours, it is vital that its sodium is similar to the plasma's to prevent it causing dilution.

As stated, this consists of carefully made estimations, attention to detail, and meticulous monitoring, but does not contain any complex mathematics that would really justify it being described as a calculation.

(4) Your assessment of Dr. Taylor's conduct at the time and subsequently from 1995 to 2012 in light of this statement

I believe that Dr Taylor made mistakes whilst managing Adam in 1995 which ultimately lead to his death. It is unfortunate that it has taken him 16 years to recognise these errors. It is very easy for anybody to make misjudgements whilst working in the complex field of medicine; nobody is immune from this, but what is required is to review them openly and honestly with oneself, colleagues and families, to learn from them, and to disseminate that knowledge to reduce the chances of the same errors being repeated. It seems that this opportunity was not taken fully in this case, which will inevitably have delayed learning points for both Dr Taylor and the whole paediatric community. I hope now that Dr Taylor's recognition of mistakes will allow these important lessons to be fully appreciated.

(5) Whether there is anything arising that requires further query or investigation

All the points I wish to make are contained within my answers to questions (1) to (3).

(6) Whether the statement causes any amendment of your previous expert reports to the Inquiry, and if so, what amendment is required and the reason(s) for the amendment(s).

No.

The significance of the renal transplant surgery

3. Please comment on the significance of the fact that Adam was undergoing a renal transplant surgery in terms of:

   (1) The difficulty (or otherwise) in making accurate fluid calculations

In my response to paragraph 9 or Dr Taylors recent report above, I have referred to the principles and practice of estimating and prescribing fluids to a child undergoing a kidney transplant; what Dr Taylor refers to as making fluid calculations. However, what should be emphasised is that the strategies which are needed to guarantee the safe management of children undergoing transplantation are not in any way specific to this group of children, but are generic. The same considerations and care must be given to almost every seriously ill child.

Some of the testimonies for this inquiry (including mine) may not have made this point clear, so that readers who have not had direct experience of paediatric medical care could easily gain the impression that a child receiving a transplant had medical complexities that set them apart from other ill children. Not so.

In health, children regulate their water intakes by drinking in response to their thirst drives, they regulate their calorie intakes by eating in response to hunger (and social pressures), and they regulate their fluid and salt losses by adjusting independently the output of water and sodium by their kidneys. While many of these processes occur without the child's cooperation or awareness, eating and drinking appropriately depend upon complex, sophisticated biological sensory feedback mechanisms such as satiety.

When small children become even mildly ill, loss of appetite and vomiting are very common phenomena, and these interfere with their ability to maintain their nutritional and fluid intakes. In more seriously ill children, such as those recovering from virtually any type of major surgery, many other factors such as the use of pain-killing drugs and sedation further compromise their ability to regulate their own bodily functions, and they therefore require external support.

Acute kidney failure is a very common complication in seriously ill children because the kidneys are very vulnerable in the face of a poorly maintained blood circulation. For example, this is often seen in children with major infections such as meningitis. They are therefore not only unable to regulate their intakes, but are also unable to appropriately control their fluid and sodium losses during this period, when they may not pass any urine at all.

As a consequence, many small children with a wide range of diagnoses looked after in paediatric intensive care units (PICUs) need to have their sodium and water intakes and losses balanced for them by medical staff using the principles I have described above for a child undergoing a kidney transplant. Adam is not a special case; his management parallels that of many other vulnerable children. The child who develops severe meningitis and then temporary kidney failure and who requires an anaesthetic for a procedure presents precisely the same problems, and requires the same management. Caring for acutely ill children is part and parcel of being a paediatrician. Managing them in a PICU setting is a role central to paediatric anaesthetists; it is a key part of their training. Giving resuscitative boluses of fluid is a daily event on PICUs, and doing that with hypotonic saline would be equally dangerous there as it was for Adam.

It is true that there are exceptional circumstances within PICU and anaesthetic settings that I would not expect an 'ordinary' paediatric anaesthetist to be able to manage. These would include, for example, children managed on extra-corporeal membrane oxygenation (ECMO) circuits, or undergoing open-heart surgery on cardio-pulmonary by-pass, which is why such procedures are concentrated in ultra-specialist units, staffed by 'super-specialists'.

However, I would expect any competent paediatric anaesthetist to be able to safely manage the fluid and salt requirements of any child (with or without kidney failure) whilst comatose or during an

ordinary general anaesthetic, and to avoid producing hyponatraemia by the excessive use of hypotonic solutions. Renal transplantation is but one example of this.

I have personally held extensive interactive teaching sessions on salt and water management with all the general paediatric trainees that have worked in my hospital for over 2 decades. I have never heard any junior doctor advocate using one-fifth-normal saline as a rapid infusion or bolus to correct a fluid deficit. I would expect all paediatric registrars (senior trainees) to have a good grasp of the basic fluid management principles that I have outlined. As a paediatric nephrologist, and one particularly interested in salt and water metabolism, I have been asked countless times to assist with the diagnosis and management of children with hyponatraemia throughout hospitals in the English Northern Health Region. Although this is frequently due to less than ideal fluid management, I cannot recall any doctor who has consciously and intentionally infused a child with a hypotonic fluid bolus in the belief that it was an appropriate therapy. I would not expect this of any consultant paediatrician. What happened to Adam is therefore, in my experience, truly exceptional.

(1) The need (or otherwise) for prompt responses to requests for laboratory testing of electrolytes

When children become unwell, they typically become sick much quicker than adults, and similarly they usually 'bounce back' from illnesses much more quickly. Their metabolism is faster,[1] their fluid turnover is greater, their organs typically have less 'reserve capacity', and in many ways they are much more vulnerable than adults. For this reason, the assessment and treatment of ill children nearly always has to be especially prompt. The diagnosis and monitoring of paediatric patients frequently involves the rapid measurement of laboratory tests, including electrolytes.

For these reasons, paediatric services, and PICUs in particular are disproportionately high users of hospital laboratory services, especially of emergency biochemistry assays such as electrolytes. It is not safe to look after very ill children unless electrolyte assays are available promptly 24-hours per day. Paediatric inpatient services in the UK are arranged with a regional and sometimes supra-regional structure, so that all the sickest of children and those undergoing specialist treatment or surgery are concentrated into centres where the expertise is available. Part of the mandate of such centres is to provide rapid laboratory services for sick children. This means ensuring that tests can be performed rapidly on small samples 24/7.

In my experience (from the mid-1970s), it would be very rare to wait more than an hour to obtain electrolyte assay results from specimens sent urgently to a laboratory, and typically they would be completed within about ½ an hour. Ones considered very urgent (with that message telephoned through to the technical staff) are usually ready within approximately 15 minutes. This level of service is a necessity and not a luxury to manage children undergoing renal transplantation.

[1] This is because it varies with body surface area, and the body's surface area to weight ratio changes reciprocally with height – a 2-year old is half the height of an adult and has twice the metabolic rate per kg. A 1 kg baby's metabolic rate per kg is approximately 5-fold that of an adult.

Living donors

4. In her second witness statement to the Inquiry, Debra Slavin, Adam's mother, stated (WS-001-2, Answer to Q25(b), p.5):

"I asked if I could donate [as a living donor] but as a single parent this was not allowed, apart from that there was no other discussion as a living donor."

5. Please comment on the following:

(1) What consideration you think should have been given by Dr. Savage (or anyone else involved in Adam's case) to the possibility of living donation for Adam.

The obvious reason which might be expected to lead to the introduction of a policy of refusing live donation from a single parent is the prospect of that parent dying as a result of the surgery, and leaving the child without any parent. However, the risks of this happening are so small as to make this an unreasonable blanket policy decision, either in 1995 or now. The risk of a donor dying is extremely small. It was of the order of 3,000 to 1 against in 2001,[2] and this had not changed in 15 years. In my experience, this risk is considered so low by relatives considering donation that it hardly enters into the decision making compared to the other issues.

From the perspective of the risks to the child or the physical or psychological morbidity to the parent or the family unit, the situation with regard to living donation of kidneys to small children has evolved steadily during the last decades. This must be remembered when considering in 2012 what should have been done in 1995. There are 2 main reasons for the changes in attitude.

First, live donation has always yielded better success rates than using deceased donors ('cadaveric kidneys'). This is probably mainly due to the closer tissue-matching that is likely to occur when half of the donor kidney's genes have been supplied by the donor. Even among kidneys that on paper are equally closely matched, live donor kidneys do slightly better, and this is probably because of them sharing other features in common that we cannot currently test for.

This difference has been gradually falling over the years because the survival of cadaveric kidneys has improved due to better immunosuppression drugs, but it still exists, and certainly did in 1995.

The second reason for a change in approach to live donation has been the general improvement in paediatric renal transplantation. When I began managing kidney transplants in 1983, a significant proportion of kidneys grafted into small children did not fare well – in particular organs were often lost early on due to them clotting, that is from causes not related to the closeness of match, and which were equally likely to happen to a live-donor kidney. By about 1990 this had improved considerably due to a number of changes to management that were very widely introduced. These included the use of prophylactic heparin (an anti-blood clotting agent), connecting the kidney blood vessels onto the child's aorta and vena cava (much bigger vessels than were previously used).

A child losing a kidney graft and needing to remain on dialysis is traumatic enough; for their parent to have also lost one of their kidneys, and not to be as fit as normal to look after their child at this important time can compound their grief considerably. In addition, the feeling of guilt perceived by a parent engendered by their kidney not working for their child can be highly traumatic. Many doctors were therefore understandably cautious about undertaking live donation at that stage. However, since about 1990 it has been much rarer for kidneys to be lost early for technical reasons, and this has influenced a gradual and now major shift towards live donation from parents or other close relatives. Even more recently, being able to harvest donor kidneys by keyhole surgery has made the procedure less painful for the donor, with a quicker recovery time and less scarring, and this has also influenced the trend.

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[2] Matas AJ, Bartlett ST, Leichtman AB, Delmonico FL. Morbidity and mortality after living kidney donation, 1999–2001: Survey of United States Transplant Centers. American Journal of Transplantation,2003:3;830-4.

Thus, a decade before Adam's transplant there was a significantly better chance of a live-donor kidney working than a cadaveric one, but there was also a definite risk that any kidney might fail in a small child. A decade after his transplant, all small children's kidney transplants are doing much better, so the difference between live-donor and cadaveric survival has become almost negligible, and in our unit the majority of grafts are now from a parent. Other advantages of live donation that have driven this change is that it eliminates the uncertainty and stresses of being on a waiting list for an organ that may come tomorrow, or may not come for a couple of years, and it allows sensible organisation and planning to suit families. The situation in 1995 is difficult to precisely recall, but it would have been changing between these 2 positions.

In my opinion, however, Debra Slavin should have been given all of the facts surrounding the issue, and should have had an opportunity to consider this option in an informed manner. The fact that a parent or other relative brings this subject up themselves is in itself an indication of their commitment, and that deserves to be recognised and responded to.

The message that Debra has taken from Dr Savage's reply is that live donation was not allowed from a single parent. If that is was what was actually said by Dr Savage, I would have been surprised, and I would not be able to understand such an arrangement, but it is possible that he was misunderstood or misinterpreted by Debra.

While live donation has the obvious advantages that I have already outlined, it also has major potential disadvantages and pitfalls. Some parents offer a kidney, and later regret the offer but feel trapped with their initial impulse decision. Donating a kidney which is subsequently lost can be devastating. Providing support and comfort to a child recipient whilst yourself are recovering from giving a kidney is extremely challenging. There can also be specifically increased medical risks to particular donors, such as those with marginally raised blood pressure.

In my view, these negative aspects can sometimes be so important that live donation is not appropriate as it may not be in the overall interests of one or other or both individuals. However, decisions about which donor-recipient pairs would or would not benefit is not simple, and should certainly not be made on the basis of blanket rules, especially ones that rely on stereotyping such as being a single mother.

Instead, each family should be assessed for as many of these aspects as it is possible to do. In 1995 we undertook screening medical assessments for donation by an adult nephrologist (ie, not the doctor looking after the child, as the donor needs their own advocate) and a transplant surgeon, by our team social worker, and by a family psychiatrist. Now there is a more formal structure to the process, but it is essentially the same. If all of these individuals considered that it was appropriate to proceed, then the donor would undergo the more invasive medical testing procedures that are required to ensure safe donation. We would certainly have undertaken these steps for Debra.

Dr Savage and his team must already have known Debra extremely well by 1995, since they had assessed her ability to manage dialysis at home, and had supported her in undertaking and performing that. It is clear from Dr Savage's letters that he held Debra in high regard for her management of Adam's dialysis. It may well be that his detailed awareness of her social circumstances and family support was already so extensive by the time she made the offer of live donation that he could already make a well informed judgement about how it may have affected both her and Adam. If he did use the words that Debra states, it may be that he did so out of a sense of trying to avoid embarking upon an unlikely route or a more potentially painful refusal. That is, it is possible that he was 'fobbing her off' with the best of intentions. Alternatively, it may be that he pointed out to her that she did not have a lot of support as a single mum, and that Adam would not cope well without her by his side all of the time, and that the 'single parent' statement was all that was clearly remembered from a more complex and considered discussion.

(2) If you consider that consideration should have been given to a living donation for Adam, please state

(a) When that should have happened

In 2012, our team would raise the possibility of a live donation as soon as the question of the need for a transplant was raised. In the 1980's it would not have been raised by the professionals, though it would have been responded to as soon as the family asked about it. I cannot remember precisely how it was managed between these extremes in 1995.

(b) Who should have been involved in considering that option
(c) What would have been involved in any such consideration

I have covered these points in the discussion above.

(d) When it should have been raised with Adam's mother and what should have been discussed with her about it

See answer to (a)

(e) What notes and records should have been made of any clinical consideration of a living donation and any information provided to Adam's mother about the process

I would have expected the fact that the issue had been raised, and the discussion or conclusion of that to be documented in the medical records.

(3) Whether the fact that Adam's mother was a single parent was a legitimate factor when considering the viability of living donation in Adam's case

(4) The advantages and disadvantages of living donors.

I have covered these points in the discussion above.

Comparison of fluid balance assessments

6. As you are aware, you recently completed a fluid balance chart showing what you believed to be the fluids received and the fluid lost by Adam both before and during his surgery. The Inquiry also asked other Inquiry expert witnesses and Dr. Robert Taylor to complete a similar chart, and this data has now been included in a comparison table, which is attached.

7. The Inquiry team would ask you to:

(1) Check that your calculations and assumptions have been transcribed correctly onto this comparison table.

Yes

(2) Comment on any differences (if any) that are apparent between your calculations and the other Inquiry expert witnesses.

There are some differences between the figures reached by Dr Haynes and Prof Gross and myself. Most are unimportant to the final quantities and balances, and are due to slight differences in the estimations that we have had to make, but some are consistent and due to methodological differences or errors. I will deal with these below:

i. Prof Gross uses body weight rather than body surface area to estimate the insensible losses of fluid from skin and breath. This is incorrect in children because insensible losses vary with the body surface area. It may be common practice to use a per kg estimate in adult patients where the surface area to weight ratio only varies slightly between different patients, but this cannot be projected to small children. However, this only makes a difference of about 11 ml/hour between his estimates and mine. I note that Dr Haynes uses 400 ml/m^2 hourly instead of the more common figure of 300, and suspect that his figure may be more appropriate for a child in a PICU or theatre (ie warm) environment, and is 4 ml/hour greater than mine.

ii. Dr Haynes and Dr Taylor have both included an extra figure for evaporative water losses of about 80 ml hourly from the open wound during the 3½ hours of surgery. This reflects their anaesthetic expertise, and Prof Gross and I both omitted to do this.

iii. Drs Haynes and Taylor and Prof Gross use 40 mmol/l as an estimate of Adam's urinary sodium concentration, whereas I use a value of 75 mmol/l. I have explained the reasons for this difference previously. In summary, Adam's urinary sodium concentration was not measured around the time of his transplant, so the value has to be an educated guess. The other 3 doctors have used urinary sodium levels recorded for Adam from when his kidneys were working better, and keeping him essentially fit and well. I did not do this because I recognise that whatever the urinary sodium may be when a child has mild or moderate renal failure, it always tends to approach a value of about 75 mmol/l once the kidneys fail enough to require dialysis (ie, that is how end-stage kidneys behave).

iv. Prof Gross has miscalculated the quantity of sodium present in 0.18% saline. For example, in the 07:00 to 08:00 period, he calculates that the 650 ml administered contained just 16 mmol, implying a concentration of about 25 mmol/l. His other figures are not fully explained, but I think that this is a consistent error rather than a single arithmetical mistake. For the record, both Dr Haynes and I rounded the concentration to 30 mmol/l, whereas the true figure, given the molecular weight of sodium chloride of 58.5 is 1800/58.5 = 30.8 mmol/l. In the recalculations I present below, I have used the closest integer of 31 mmol/l.

v. In his section on administered fluid between 08:00 and 10:00, Prof Gross provided a figure of 1,750 ml, which is 550 ml less than either Dr Haynes or I find. It may be that 150 ml of the difference is that Prof Gross has assumed that only 600 ml of the 750 ml of 0.18% saline which was given after 08:00 was administered before 10:00, as he indicates that 150 ml was given a little later, but I cannot even speculate about the rest of the difference as Prof Gross has not supplied details of how he reaches a total of 1,750 ml.

(3) Comment on the assumptions and calculations used by Dr. Robert Taylor, particularly in the light of:

(a) His previous arguments and
(b) His most recent statement

I will deal with point (b) first, and comment on Dr Taylor's figures from his recent statement first, and compare what this would mean for Adam's salt and water balance with the conclusions that the 3 expert witnesses have drawn. After that I will calculate the serial balances that would have been achieved if Dr Taylor's initial assumptions had been true.

(b) His most recent statement

The figures that Dr Taylor had entered into the composite table that I have been supplied with are broadly consistent with the ones in his statement of 01/02/2012. However, there are some important points to make:

i. Dr Taylor does not ascribe any fluid losses to Adam's dialysis. I do not know why he still does not do this in his recent reassessment, as these data are available in the bundles (from Debra Slavin's diaries), and have been discussed in detail in previous expert witness submissions. This alters the balance estimations for 2 reasons:

   a. It alters the assessment of his normal daily urine volume, which is then used to predict his hourly urine output during surgery. When Drs Savage and Haynes, and Prof Gross and I have estimated his daily urine output to be between 1,350 and 1,500 ml, we made an allowance for the fact that his dialysis removes water every day. If we had not done so, we would have come up with higher hourly urine volume estimates. This may explain why Dr Taylor uses the higher figure of 78 ml/hour.

   b. It alters the estimates of sodium loss. His urine sodium concentration will have been much lower than his dialysis fluid sodium concentration, so counting dialysis losses as urine losses exaggerates the loss of hypotonic fluid, and would tend to overestimate the amount of free water Adam would need to be given.

ii. Dr Taylor estimates that between 08:00 and 10:00 (the first 2 hours of surgery)

   a. The blood losses were 800 ml, at least 200 ml greater than the other 3 assessors' values.

   b. Only 400 ml of the 750 ml of 0.18% saline administered after 08:00 was given during this 2-hour period (he indicates in the table that the rest was given over the next 2¼ hours, which is not consistent with the evidence in the medical records). Although the total quantities match, this makes a significant difference because a fundamental issue is how quickly the hypotonic fluid was administered; how much it was a bolus, and how much it was a slow infusion.

Recalculation of the sodium and water balances from the tables provided.

Water and sodium balances

I have produced an Excel file which has allowed me to calculate the estimated water losses and replacements for each of the time periods, and then to calculate the balances for each period, and to serially sum them to produce cumulative balances figures. The same spreadsheet has allowed me to calculate the parallel values for sodium, including cumulative sodium balances.

I have provided an electronic copy of the Excel file to the inquiry which shows precisely how the calculations have been undertaken, and allows others to easily produce any other modified scenarios that they may consider helpful.

Using these figures, it has been possible to calculate the mean (average) sodium concentration of the accumulated fluid for each period, and on a cumulative basis:

mean sodium concentration of retained fluid = sodium balance / water balance (mmol/l)

Infused free water volumes

This figure has allowed me to calculate the total free water volume that has been accumulated during each period, and cumulatively. This is deduced by treating the retained water and salt as if it was made up of 2 volumes, a quantity of physiological saline with a sodium concentration of 140 mmol/l, and a quantity of water without any sodium (free water). The calculation is as follows:

free water = ((water balance x 140) – sodium balance) / 140

This is the crux of the issue. This is the volume of water without any salt that has been added into Adam's circulation, and therefore the amount which will cause the plasma water to become diluted.

Notes about the tables (which are presented on pages 23 – 28)

a) The time periods are as follows:

1	22:00 – 05:00	7 hours	Whilst on the ward, not fasted
2	05:00 – 07:00	2 hours	Period of fasting before anaesthetic induction
3	07:00 – 08:00	1 hour	Anaesthetic induction, before surgery began
4	08:00 – 10:00	2 hours	From start of surgery to vascular clamps going on
5	10:00 – 10:30	0.5 hour	While vascular clamps are applied
6	10:30 – 11:30	1 hour	From release of clamps to end of surgery
7	11:30 – 12:15	0.75 hour	Between end of surgery and arrival in PICU

b) I have used a sodium concentration of 31 mmol/l for 0.18% saline, including in Prof Gross' table.

c) I have used a concentration of urinary sodium of 75 mmol/l in my table, and 40 for the others.

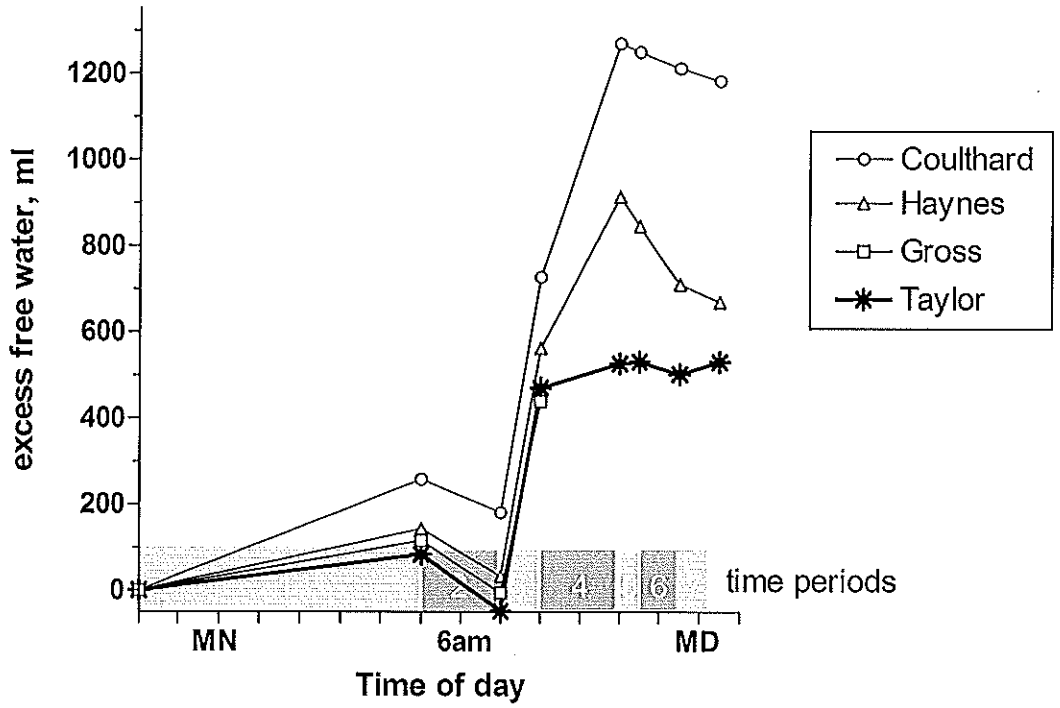
Results of the calculations

The tables for Dr Haynes, Prof Gross and myself using the data from the integrated table supplied by the inquiry team are on pages 23 – 25, and the table for Dr Taylor using the same source or figures is on page 26.

In Figure 1, I have graphed the cumulative free water volumes from these 4 tables. Although there are some clear points of difference between the different assessors' values, there are some obvious and important similarities.

First, all 4 plots indicate that the amount of free water that Adam accumulated between his admission to the ward and his anaesthetic induction increased slowly to between about 100 and about 250 over 7 hours, and then fell back again by around 80-120 ml while he fasted. All assessors therefore consider that he was close to neutral by the time he was anaesthetised, somewhere between being about 50 depleted of free water, and being 180 ml overloaded. At this stage I consider that he was less depleted of free water the other 3 assessors do, and this is mostly because I have assumed that his urinary sodium would have been 75 mmol/l, as is seen in other children with end-stage renal failure, rather than using Adam's own historic data when his kidneys were not end-stage.

Figure 1 Graph of Adam Strain's peri-operative free water accumulation, divided into 7 time periods, according to estimated made by 3 expert witnesses (open symbols) and by Dr Taylor, according to his recent testimony.



The second point of close agreement is that all 4 graphs show that Adam accumulated about 500 ml (445 – 545 ml) during the next hour, from the induction of the anaesthetic and the commencement of surgery. This would have inevitably diluted his plasma sodium concentration rapidly.

It is not possible to plot the free water accumulation during the next 2-hour period from Prof Gross' data because the breakdown of the infused fluids is not provided in the table provided. However, both Dr Haynes and I calculate a further accumulation of about another 450 ml of free water during this time (350 – 542 ml). This would sum to a total of just under 1 litre of water during a 3 hour period (878 – 1087 ml).

By contrast, Dr Taylor's assumptions indicate that only another 56 ml would have accumulated during the 2 hours of period 4. However, I have already noted that he has interpreted that Adam did not have all of the 0.18% saline that is indicated in the medical records during this time, and even now his assessment of Adam's urine losses fail to take his dialysis into account.

Conclusion about Dr Taylor's management in relation to his recent reassessments

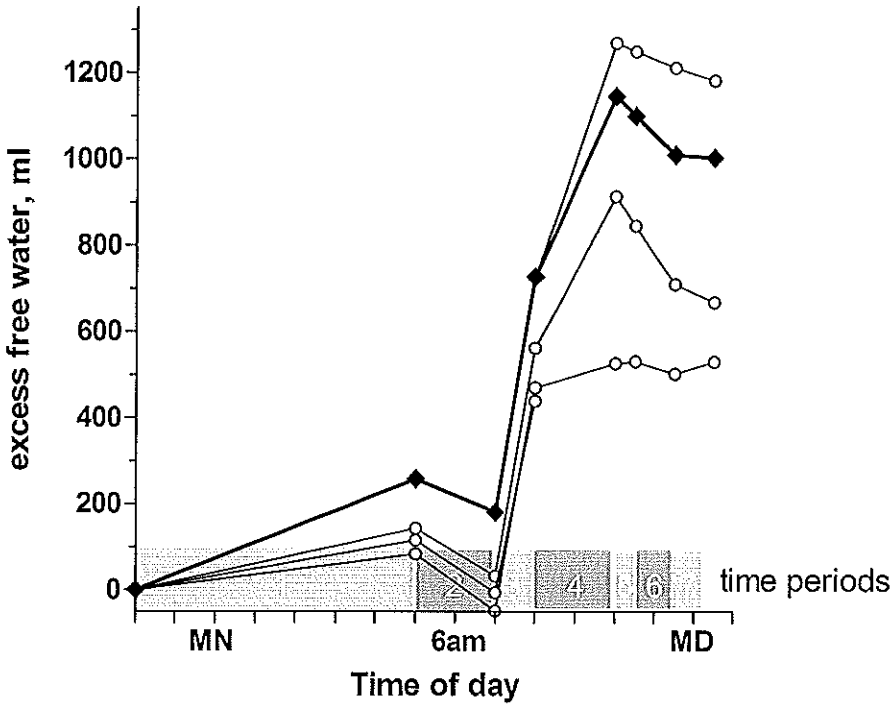
As they stand, Dr Taylor's present assessment of Adam's balances during the first 3 hours of anaesthesia agrees with that of the 3 expert witnesses in that he would have accumulated 0.5 litres of free water, over and above any physiologically correct fluids during that time. It appears to the 2 experts who have supplied sufficient detail to analyse this time period that Adam then accumulated about another 450 ml over the next 2 hours, and I cannot explain how Dr Taylor has interpreted the medical records in such a way that he considers that this quantity would only have been about 50 ml.

My new position

In reanalysing these data, I have realised that in my original assumptions I had not included a figure for the extra water that would have evaporated from his open wound. I also realise that I have until now been including a calculated urine output as if it continued throughout the whole of the period of surgery, even though the notes make it clear that this was not the case. Instead, Adam was noted to have produced merely 49 ml between arriving in the operating theatre and arriving in PICU. This makes sense if he was unstable during his anaesthetic, and that this had an impact on his residual kidney function. This is common in end-stage renal failure, where any slight pathophysiological change can produce a fall in kidney perfusion sufficient to prevent renal filtration.

I have therefore recalculated what I personally now believe to be the most reasonable set of assumptions, and present these in the table on page 27, and in Figure 2. I have also plotted the 4 lines from Figure 1 onto that figure. According to that model, Adam would have accumulated 962 ml of water during the first 3 hours of his anaesthetic.

Figure 2 Graph of Adam Strain's peri-operative free water accumulation, divided into 7 time periods. In this version, the 3 expert witness and Dr Taylor's graphs from Fig 1 are shown with open symbols, and the curve that Dr Coulthard now considers to be the most likely to be valid is shown with solid diamonds.



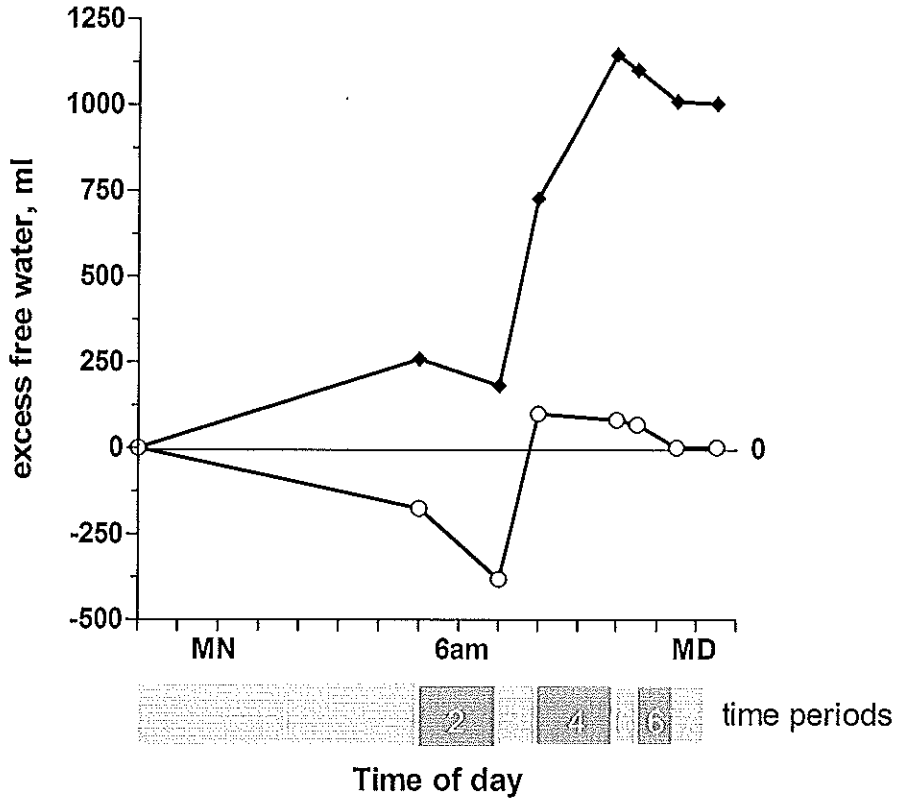
(a) His previous arguments

The table on page 28 shows the fluid balances that would have been achieved by Dr Taylor if his assumptions at the time about Adam's abilities to manage sodium and water had been correct. Figure 3 shows this graph plotted alongside the line (already shown in Figure 1) using the assumptions that I now consider most likely to be correct.

This shows that by assuming the urine output was unable to fall below 200 ml/hour, by assuming its sodium concentration was likely to be 40 rather than about 75 mmol/l, and by ignoring the effect of dialysis on stabilising his fluid volume and sodium concentrations overnight, Dr Taylor's predictions would have been that Adam would have become water depleted by almost 400 ml by the time he came to induce his anaesthetic.

He then infused 0.18% saline into him in order to correct this water deficiency. It can be seen that this is what happened, and the balance was then maintained close to correct (0) right until the end of the operation.

Figure 3 Graph of Adam Strain's peri-operative free water accumulation, divided into 7 time periods. In this version, the curve that Dr Coulthard considers to be the most likely to be valid is shown with solid diamonds, and the curve produced by using Dr Taylor's assumptions at the time are shown using open circles.



If Dr Taylor had been correct that Adam had been water depleted at the start of the anaesthetic (instead of being about 200 ml in surfeit, as he was), he should not have dealt with it by simply infusing in 0.18% saline quickly in this way because this will inevitably suddenly lower the sodium concentration. Falling from a high level to a more normal one is just as damaging as going from normal to very low – it is the rate of fall of plasma sodium that must be prevented in all circumstances.

Thus, it is fair to say that his fluid management did indeed match *overall* the fluid requirements that he had wrongly imagined Adam to have, but that it would have achieved that by inducing an acute fall in plasma sodium.

Conclusion about Dr Taylor's management in relation to his original assessments

By (a) considering that Adam had a fluid output of at least 200 ml/hour, and with no upper limit, (b) accepting that his urine concentration would be unaltered from his pre-end-stage days, and (c) by failing to consider the stabilising impact that peritoneal dialysis has, Dr Taylor imagined that he was dealing with a clinical challenge that was very different from its reality. By deciding to ignore the CVP trace which contradicted his notion that Adam was fluid depleted, he missed an opportunity to correct his position, and gave up a vital tool for the rest of the procedure. With this background, he would have managed to maintain what would have been an overall relatively steady status quo (ending up in balance) for Adam if his physiology did indeed behave as he had thought it would, but at the price of a sudden increase in free water infusion and a predictably too-sharp fall in his plasma sodium concentration.

By not catheterising Adam he relinquished an opportunity to check that the urine output was indeed as high as he had imagined, or that it was unaffected by the anaesthetic, given how vulnerable such poorly functioning kidneys were likely to be.

By not checking the plasma sodium after discovering that the near-patient reading indicated a very low reading, and to disregard it instead, he opted to simply continue on blindly administering hypotonic fluids without the benefit of checking their impact upon Adam's blood levels.

Thus, while Dr Taylor's primary error was to fail to properly estimate Adam's normal losses (and thus his required replacement fluids), this would not have led to any mishap if (a) he had not used rapid boluses of hypotonic saline instead of isotonic or near-isotonic solutions and aiming for gentle biochemical changes, and if (b) he had ensured that he monitored his CVP, his urine output and his biochemistry, because the trend to induce hyponatraemia would have been detected earlier, and severe changes could have been avoided.

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 _____ 16/02/2012

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

Dr Haynes

Time periods			1	2	3	4	5	6	7		totals
duration of period (hours)			7.0	2.0	1.0	2.0	0.5	1.0	0.75		**14.25**
LOSSES											
General insensible	(ml/h rate)	14	98	28	14	28	7	14	11		**200**
Wound insensible	(ml/h rate)	80				160	40	80			**280**
urine output	(ml/h rate)	57.5	403	115	58	115	29	58	43		**819**
dialysis			213								**213**
blood						600	200	328			**1128**
all fluid losses			714	143	72	903	276	480	54		**2640**
Cumulative fluid losses			**714**	**857**	**928**	**1831**	**2107**	**2586**	**2640**		
FLUIDS GIVEN											
Dioralyte			952								**952**
0.18% saline					750	750					**1500**
Hartmann's						500					**500**
Plasma/HPPF						800	200				**1000**
Blood						250		250			**500**
All fluids in			952	0	750	2300	200	250	0		**4452**
Cumulative fluid intake			**952**	**952**	**1702**	**4002**	**4202**	**4452**	**4452**		
Fluid balance			239	-143	679	1397	-76	-230	-54		**1812**
Cumulative fluid balance			**239**	**96**	**774**	**2171**	**2095**	**1866**	**1812**		
SODIUM LOSSES (urine Na 40)			44	5	2	89	29	48	2	0	218
SODIUM GAINS			57	0	23	235	28	35	0	0	379
Sodium balance			13	-5	21	147	-1	-13	-2		160
Cumulative sodium balance			**13**	**9**	**30**	**176**	**175**	**162**	**160**		
Mean Na of extra water (mmol/l)			**56**	**91**	**38**	**81**	**84**	**87**	**88**		
Excess free water gained	**(ml)**		**143**	**33**	**562**	**912**	**844**	**709**	**668**		

 Signed .....

Prof Gross

Time periods			1	2	3	4	5	6	7	totals
duration of period (hours)			7.0	2.0	1.0	2.0	0.5	1.0	0.75	**14.25**
LOSSES										
General insensible	(ml/h rate)	21	147	42	21	42	11	21	16	**299**
Wound insensible	(ml/h rate)									
urine output	(ml/h rate)	56	392	112	56	112	28	56	42	**798**
dialysis			154							**154**
blood						518	130	259		**907**
all fluid losses			693	154	77	672	169	336	58	**2158**
Cumulative fluid losses			**693**	**847**	**924**	**1596**	**1765**	**2101**	**2158**	
FLUIDS GIVEN										
Dioralyte			970							**970**
0.18% saline					650	?		150	25	**825**
Hartmann's						?				**0**
Plasma/HPPF						?	200			**200**
Blood						?		250		**250**
All fluids in			970	0	650	1750	200	400	25	**2245**
Cumulative fluid intake			**970**	**970**	**1620**	**3370**	**3570**	**3970**	**3995**	
Fluid balance			277	-154	573	1078	32	64	-33	**1837**
Cumulative fluid balance			**277**	**123**	**696**	**1774**	**1806**	**1870**	**1837**	
SODIUM LOSSES (urine Na 40)			36	4	2	77	19	39	2	207
SODIUM GAINS			58	0	20	?	28	40	1	?
Sodium balance			23	-4	18	?	9	1	-1	?
Cumulative sodium balance			**23**	**18**	**36**	**?**	**?**	**?**	**?**	**?**
Mean Na of extra water (mmol/l)			**81**	**147**	**52**	**?**	**?**	**?**	**?**	
Excess free water gained	**(ml)**		**116**	**-6**	**439**	**?**	**?**	**?**	**?**	

Dr Coulthard

Time periods			1	2	3	4	5	6	7		totals
duration of period (hours)			7.0	2.0	1.0	2.0	0.5	1.0	0.75		**14.25**
LOSSES											
General insensible	(ml/h rate)	10	70	20	10	20	5	10	8		**143**
Wound insensible	(ml/h rate)										
urine output	(ml/h rate)	62	434	124	62	124	31	62	47		**884**
dialysis			200								**200**
blood						600	200	328			**1128**
all fluid losses			704	144	72	744	236	400	54		**2354**
Cumulative fluid losses			**704**	**848**	**920**	**1664**	**1900**	**2300**	**2354**		
FLUIDS GIVEN											
Dioralyte			952								**952**
0.18% saline					750	750					**1500**
Hartmann's						500					**500**
Plasma/HPPF						800	200				**1000**
Blood						250		250			**500**
All fluids in			952	0	750	2300	200	250	0		**4452**
Cumulative fluid intake			**952**	**952**	**1702**	**4002**	**4202**	**4452**	**4452**		
Fluid balance			248	-144	678	1556	-36	-150	-54		**2098**
Cumulative fluid balance			**248**	**104**	**782**	**2338**	**2302**	**2152**	**2098**		
SODIUM LOSSES (urine Na 75)			59	9	5	93	30	51	3		250
SODIUM GAINS			57	0	23	235	28	35	0	0	379
Sodium balance			-1	-9	19	142	-2	-16	-3		128
Cumulative sodium balance			**-1**	**-11**	**8**	**150**	**147**	**132**	**128**		
Mean Na of extra water (mmol/l)			**-6**	**-103**	**10**	**64**	**64**	**61**	**61**		
Excess free water gained	**(ml)**		**258**	**181**	**726**	**1268**	**1248**	**1210**	**1181**		

Dr Taylor, on basis of his statement of 01/02/2012

Time periods			1	2	3	4	5	6	7	totals
duration of period (hours)			7.0	2.0	1.0	2.0	0.5	1.0	0.75	**14.25**
LOSSES										
General insensible	(ml/h rate)	10	70	20	10	20	5	10	8	**143**
Wound insensible	(ml/h rate)	80				160	40	80		**280**
urine output	(ml/h rate)	78.1	547	156	78	156	39	78	59	**1113**
dialysis			0							**0**
blood						800	200	211		**1211**
all fluid losses			617	176	88	1136	284	379	66	**2746**
Cumulative fluid losses			**617**	**793**	**881**	**2017**	**2301**	**2680**	**2746**	
FLUIDS GIVEN										
Dioralyte			952							**952**
0.18% saline					750	400	100	150	100	**1500**
Hartmann's						500				**500**
Plasma/HPPF						800				**800**
Blood						250		250		**500**
All fluids in			952	0	750	1950	100	400	100	**4252**
Cumulative fluid intake			**952**	**952**	**1702**	**3652**	**3752**	**4152**	**4252**	
Fluid balance			335	-176	662	814	-184	21	34	**1506**
Cumulative fluid balance			**335**	**159**	**821**	**1635**	**1451**	**1472**	**1506**	
SODIUM LOSSES (urine Na 40)			22	6	3	118	30	33	2	253
SODIUM GAINS			57	0	23	224	3	40	3	0 351
Sodium balance			35	-6	20	106	-26	7	1	98
Cumulative sodium balance			**35**	**29**	**49**	**155**	**129**	**136**	**137**	
Mean Na of extra water (mmol/l)			**105**	**182**	**60**	**95**	**89**	**92**	**91**	
Excess free water gained	**(ml)**		**84**	**-48**	**470**	**526**	**531**	**502**	**530**	

Dr Coulthard's current 'final view'

Time periods			1	2	3	4	5	6	7		totals
duration of period (hours)			7.0	2.0	1.0	2.0	0.5	1.0	0.75		14.25
LOSSES											
General insensible	(ml/h rate)	10	70	20	10	20	5	10	8		143
Wound insensible	(ml/h rate)	80				160	40	80			280
urine output	(ml/h rate)	62	434	124	62	46					666
dialysis			200								200
blood						600	200	328			1128
all fluid losses			704	144	72	826	245	418	8		2417
Cumulative fluid losses			**704**	**848**	**920**	**1746**	**1991**	**2409**	**2417**		
FLUIDS GIVEN											
Dioralyte			952								952
0.18% saline					750	750					1500
Hartmann's						500					500
Plasma/HPPF						800	200				1000
Blood						250		250			500
All fluids in			952	0	750	2300	200	250	0		4452
Cumulative fluid intake			**952**	**952**	**1702**	**4002**	**4202**	**4452**	**4452**		
Fluid balance			248	-144	678	1474	-45	-168	-8		2036
Cumulative fluid balance			**248**	**104**	**782**	**2256**	**2211**	**2043**	**2036**		
SODIUM LOSSES (urine Na 75)			59	9	5	87	28	46	0		234
SODIUM GAINS			57	0	23	235	28	35	0	0	379
Sodium balance			-1	-9	19	148	0	-11	0		145
Cumulative sodium balance			**-1**	**-11**	**8**	**156**	**156**	**145**	**145**		
Mean Na of extra water (mmol/l)			**-6**	**-103**	**10**	**69**	**70**	**71**	**71**		
Excess free water gained	**(ml)**		**258**	**181**	**726**	**1144**	**1099**	**1009**	**1002**		

 Signed .....

Dr Taylor on the basis of his original assumptions

Time periods			1	2	3	4	5	6	7		totals
duration of period (hours)			7.0	2.0	1.0	2.0	0.5	1.0	0.75		14.25
LOSSES											
General insensible	(ml/h rate)	10	70	20	10	20	5	10	8		143
Wound insensible	(ml/h rate)	80				160	40	80			280
urine output	(ml/h rate)	200	1400	400	200	400	100	200	150		2850
dialysis			0								0
blood						800	200	211			1211
all fluid losses			1470	420	210	1380	345	501	158		4484
Cumulative fluid losses			**1470**	**1890**	**2100**	**3480**	**3825**	**4326**	**4484**		
FLUIDS GIVEN											
Dioralyte			952								952
0.18% saline					750	400	100	150	100		1500
Hartmann's						500					500
Plasma/HPPF						800					800
Blood						250		250			500
All fluids in			952	0	750	1950	100	400	100		4252
Cumulative fluid intake			**952**	**952**	**1702**	**3652**	**3752**	**4152**	**4252**		
Fluid balance			-518	-420	540	570	-245	-101	-58		-232
Cumulative fluid balance			**-518**	**-938**	**-398**	**172**	**-73**	**-174**	**-232**		
SODIUM LOSSES (urine Na 75)			105	30	15	142	36	45	11		383
SODIUM GAINS			57	0	23	224	3	40	3	0	351
Sodium balance			-48	-30	8	82	-32	-5	-8		-33
Cumulative sodium balance			**-48**	**-78**	**-70**	**13**	**-20**	**-25**	**-33**		
Mean Na of extra water (mmol/l)			**92**	**83**	**175**	**74**	**269**	**141**	**141**		
Excess free water gained	**(ml)**		**-176**	**-382**	**99**	**81**	**67**	**1**	**2**		

Signed .....