Wednesday, 9 May 2012

(10.00 am)

(Delay in proceedings)

(10.16 am)

DR MALCOLM COULTHARD (continued)

Questions from MS ANYADIKE-DANES (continued)

THE CHAIRMAN: Good morning.

MS ANYADIKE-DANES: Good morning, Mr Chairman.

Good morning, Dr Coulthard. There are a few things to revisit with you and maybe a couple of substantive points. One of the things I wonder if I could revisit with you is the whole issue of re-zeroing.

Just so that we have it clear, what actually is involved in re-zeroing? What physically do you do?

A. When you have a venous pressure device, essentially there is a central line, ie a tube, going into the child's vein, which is connected, and the fluid in that is in direct continuity with the transducer, a small pressure-reading device.

That will register a pressure trace, but in order to zero it, what you're actually doing is determining at what height on the child's body you're recording the value of zero. The important point, just for people to understand why we're doing that, is that what you're wanting to do is to measure the pressure in relation to

the pressure in the child's atrium, that is to say in the heart, as it's returning to the body.

So what one does is to use an external marker from the child's body to act as a determinant for that. So a particular point on the child's side with the child lying in a particular position is taken as the point that you want the zero pressure to be recorded: in practice, in physical terms, what you do about that is that you -- in a very simple situation where the device is immediately next to the child, you simply hold the device, the transducer, at the level, vertical level that you want it to be in relation to the child's heart, and then do your zeroing process, which is actually a process whereby you open the pressure transducer to atmospheric pressure and then close it again. At that point, the transducer has been set to record zero at that level.

It's a simple enough manoeuvre to do if you have, as you do for example in intensive care very often, the actual transducer may be physically attached to the child or on the child's bed a few centimetres away.

The big practical problem in many clinical situations is that the child is in one place and the transducer is some considerable way away, for example if the child is on an operating theatre table under drapes

and so forth, and the anaesthetist is at the head of the table and he may have his instruments behind him, the transducer may well be actually physically something like a metre or two away from the child.

So what's necessary is to ensure that the opening and closing of the transducer to actually zero the instrument is done whilst the transducer is exactly the same vertical height from the ground as the child's heart.

Obviously, doing that two metres away is a technical problem. It's like putting up a shelf: if you're going to put up a shelf and you want it to be level that end and that end (indicating), you have to use some device to ensure that works.

Q. You actually produced a picture, a photograph, in a report which might help you illustrate the thing that you're now explaining. I think it's 200-019-228. There; is that it?

A. Yes, that's right, yes. So in that example, did the --

Q. Can you just blow up the picture? Thank you.

A. In that example, this is an old-fashioned central venous pressure monitoring device where the vertical column on the left is actually a fluid-filled column. It will be -- there will be a fluid-filled continuity between that and the central line which goes into the patient's

neck. And you can see that the point that is being taken as zero on the instrument is the point nearly at the bottom where the horizontal bar is. That horizontal bar in this case is a bar which has a spirit level in it. So in this very, very simple model, effectively you literally have a pressure column of fluid which is being adjusted at the physical level of the patient. Imagine if that was, say, attached to the wall or at a stand and you jacked the patient's bed up by 10 centimetres, you'd have to move the device up by 10 centimetres as well, otherwise it would read a different level.

Q. That's exactly what I was coming to.

A. Just to be absolutely clear, this is an illustration of it. What in reality we're dealing with isn't a column like this. What in reality we're dealing with is an electronic pressure device. So at the point where that vertical column meets the horizontal bar, you have a small device. But the principle is precisely the same, but that small device may be a couple of metres away from the patient and when you zero it, you have to ensure it's at the same level as the patient and it's the technical difficulties of doing that which I refer to as the problems of re-zeroing --

Q. I wonder if you can help with what actually happened --

MR UBEROI: I simply wish to establish with the witness that this is his invention, as it were, a solution to this problem, but not something that was in general use in 1995.

A. No.

MS ANYADIKE-DANES: Thank you, Mr Uberoi. The reason why I pulled up the picture is that I simply wanted to have an illustration of what he was saying. We actually know what Dr Taylor says happened and I'm just going to take you to that and I'm going to ask you how that would have worked, if I can put it that way. It's at 011-002-006.

It's the bottom paragraph starting, "The haemodynamics". He's doing it in relation to explaining some of the movement you see in that compressed trace, if I can put it that way, from the CVP monitor. What he says is that:

"The sudden increase in CVP to 28 occurred when the table was raised 5 to 6 inches for surgical reasons, but the transducer was attached to a drip stand and thus an artefact occurred. When the transducer was re-zeroed to take account of the differences in levels, the pressure returned to the previous stable range, 20 to 22, consistent with no net increase in fluid load or circulating blood volume."

And just what he's talking about is that printout

that we've all seen. There's a very clear one I found in the PSNI files. I'm just going to take you to that now. It's 094-037-211. So that's the CVP, the bottom one, and its range is 0 to 60.

A. Sorry --

Q. Its range is 0 to 40. And you can see the CVP across there. So we have understood those occasions when the trace goes down to zero to be instances of zeroing.

A. Mm.

Q. Or re-zeroing, I suppose. And what Dr Taylor was explaining is he was seeking to explain why it went up to 28, which looks like roughly around 10 am or just before 10 am, and saying that that coincided with raising the table. They then re-zeroed it and that presumably is the fall to zero round about 10 am, and then he says it comes back to, roughly, its 20 to 22 range. Well, that's his contention anyway.

So what I wanted to ask you is: if that had happened, if the surgeons for whatever reason had wanted the table raised at any given point during the procedure and that had had an effect because of where the transducer is attached, what physically happens in the operating theatre to re-zero the machine? What could be seen to be happening?

A. There are actually a number of ways that it could be

done, actually physically, and I am telling you what the potential errors are. I don't know physically precisely how they did it. Okay? Essentially, what you need to do is very simple in principle. You need a spirit level, a long enough spirit level to be able to have one end of the spirit level approximated to the marker on the child's chest, the relevant height. And that needs to be held precisely level by observing the spirit level bubble while somebody at the other end of that -- so one person, probably the anaesthetist or an assistant, would actually go under the drapes to align that to the child's chest and someone would be monitoring very carefully that the spirit level was exactly level. And at the other end, another nurse or technician or anaesthetist or somebody would then move the transducer to the same level. They would then, at that point, re-zero it. That is to say, having got it at the same level they would go through the process of opening it to the atmosphere and closing it so that was the zero marker again.

There are two reasons why I'm speculating and, I have to say, speculation is what might have happened here. There are two reasons that made me think that this is a very strong possibility. One is comparing the pressure traces on here to the pressure trace after the

child was taken to PICU. But if I could come back to that.

The second reason is that I have always been personally very unhappy about this, what I consider to be a very crude method of zeroing transducers because it's so critical. It's a sort of problem. I have seen it personally go wrong so many times in clinical practice that I've actually introduced a new way of doing it, which I've personally used, and I wish I'd published it now -- I could easily publish because I think it's an useful advance.

Personally what I have seen is just an ordinary builder's spirit level that you would use to level a shelf or something like that, itself maybe being 10 or 12 centimetres wide, being placed at one end very, very carefully by somebody at one end of the child's chest using the bottom marker, the bottom edge of the spirit level. That's fine. Then somebody at the other end is concentrating on putting the transducer at the right level and maybe puts it at the -- this is something I've seen happen -- top edge of the device, and then automatically the readings will be positive by a value equal to the height in centimetres of the actual instrument.

So if you have a big builder's instrument -- you

can't use a little one because the child is some distance away so you get something like a metre-long spirit level, which is 10 or 15 ... My own one at home -- I happened to measure it when I was thinking about this -- is 12 centimetres high. So if you have the person at this end, assuming that we're levelling it at the bottom edge, and the person this end, assuming you're levelling it at the top edge, then you immediately add 12 centimetres of value to the reading. So that a reading, a genuine reading of 12, say, would come out as 24. If it was a 15-centimetre width one, it would come out that much higher. So that is how it's commonly done. I have seen that error potentially happen in my own patients.

If people are interested, it'll take a second to explain. What I personally do is I get an oxygen tubing, just a long piece of tubing, and fill it virtually with water so that it's a long loop. If you imagine holding a skipping rope with this long loop, but water to the same level in both -- obviously the same level in both arms, and then I would get one person to hold that fluid level at the child's chest and the other person to hold it and then you can do it as far away as you like. I could know that if I put the level here (indicating) and I gave the other end to the chairman,

if it was as long as that, he would know it would be precisely the same. It seems to me in an operating theatre where you're doing it under drapes inevitably and all sort of activity going on, there's a huge possibility of that error. That would give you -- and my speculation is it may have happened. It would consistently give you a consistent error so that even if you re-zeroed and re-zeroed and re-zeroed it, if you're doing it the same way, it would always be wrong by a value of 15 or 20 depending on the instrument.

Q. One thing you've discussed is the possibility that re-zeroing it introduces errors just because of the way in which you do it. The other thing that you have clearly demonstrated is that it seems that it's not possible to carry out that exercise without it being obvious that you are carrying out that exercise. I must say, before you described it I thought you sort of pressed a reset button.

A. No.

Q. It seems to be something far more physical than that.

A. It is, yes.

Q. If you're doing that three or four times during a procedure, is that something that people are likely to notice?

A. Well, although it is physical in the sense that I've

described, it would be done quite subtly. For example, if you're wondering who else in the theatre would be aware of that, it could be done very -- I mean, you would perhaps -- if there was quiet in the theatre and people were dealing with a complex bit of surgery, a nurse could kind of creep in and slip ...

Q. But somebody's getting under the drapes, is that not something --

A. Yes, they are, but the drapes are over the -- the surgeon's operating --

THE CHAIRMAN: But there's also somebody with a spirit level and, of all the equipment of which I've heard about so far in the operating theatre, nobody has mentioned that there was a spirit level.

A. Well, there has to be in order to -- there are two options. Three options. One is that you actually have the instrument stuck on the child's chest. That we know isn't the case from -- I mean, that would be an unusual thing to do, but we know actually from the document which you just read us that it was attached to a drip stand. One option is that the drip stand is taken so proximate to the child that it's more or less touching. That would be far more disruptive in theatre than just slipping in under the drapes with a spirit level. Physically, it has to happen like that.

The third option is to use the device that we use, which again you have to slip in there, but it's less intrusive because you don't need a big metre-long instrument.

Do you want me to explain the part of the other reasoning?

MS ANYADIKE-DANES: Yes. You said there were two and then you said you'd go back to the first.

A. Right. I don't know if it's helpful to -- whether you can easily draw up the graph that I produced as part of the document from which you got that earlier picture. But what I essentially did was to --

Q. I can. If I just call it out. 200-019-229. Is that it?

A. Yes, thank you. Okay. What I've done here is simply to clean the graph that -- I have just taken individual values from the readouts and I have plotted them so that this is just a simplified graph of the graph that you saw earlier. The top one shows the readings that were taken as being true values or that were taken as being the values during the theatre. The closed -- the black dot on the right is the value when he was taken to PICU afterwards.

In PICU, the whole process of zeroing is considerably easier because the child isn't under drapes

and generally it's much simpler. You can usually bring the drips down, which has the transducer very close to the child and the whole process of making errors with zeroing are much, much less likely to happen because it's all within one person's view instead of two separate people. So the second graph is -- I just have to explain that I've talked about centimetres of the instrument ...

The instrument is ... If the instrument was 12 centimetres tall, that would make a difference of 12 centimetres of water pressure.

Q. Not mercury?

A. And the pressure transducer is actually calibrated -- you could do it in centimetres of water, but it is actually calibrated in millimetres of mercury, and the conversion factor of that because of the density of mercury related to water is 0.144, which means that 12 centimetres of water converts to 9 millimetres of mercury. So what I've then done is say: if I'd done it with my spirit level at home, the one I happen to have at home when I did this graph and took 9 millimetres off all the values in theatre, you then achieve the trace at the bottom, which would then indicate that when the first reading was taken at something like quarter to 8 in the morning, after which time he'd arrived in theatre

and had a certain volume of fluid, that the CVP was around 8 or 9, which might be exactly what you would expect if you took a child to theatre who was in normal balance and then gave them that volume of fluid.

In other words, it produces a graph, which, if that was the explanation, would make clinical sense and it would make sense as well when you look at the right-hand end of the graph, the closed circle, which is the pressure measurement in PICU and is not significantly different from the previous three or four values.

Q. Sorry, can I just be clear? What you're saying is if they had used a spirit level in the way that you described it --

A. In an erroneous way, yes.

Q. And if, therefore, they had failed to allow for the height of the spirit level or at least allowed an error to enter into it because of the different ways they treated the height -- one was going from the bottom end, the other was going from the top end, to take an example -- you have produced a graph that takes off that error and replotted those figures taking off that error, and if you do that you achieve the graph you have at the bottom --

A. Exactly.

Q. -- which is far more as you might expect if you were actually getting accurate readings?
A. Exactly.
Q. And is therefore, towards the end of surgery, far more consistent with the readings that they were getting in paediatric intensive care; is that what you're saying?
A. That is exactly what I'm saying.
Q. Of course, nobody knows if they did actually introduce that error. So far as I understand, all you are saying is: if they did, that is what it would look like.
A. If they did and they would the same size spirit level that I have at home, yes. Is it okay to just make one other point, which I think is very important in relation to why I made this speculation?
Q. Yes.
A. We talked yesterday -- and there has been a lot of discussion from other experts -- about the possibility that this monitor was actually -- the two possibilities that it was either in continuity with the blood by a fluid pathway by the blood in the right atrium or it wasn't and it was in some way stuck in a neck vessel or something like that.

The evidence, as I said yesterday, that would determine that, one way or the other, would be whether or not there was a dynamic trace showing the arterial

and venous pressure and respiratory pressure. My reading of the evidence on that -- and I just draw this to your attention -- is that the person who was responsible for looking after that, which was Dr Taylor, has said -- and I think it's the same quote, but I've seen it in two places in the evidence that I've read -- that such a trace did exist.

Q. Yes.

A. So if one accepts that the trace did exist, then it completely, in my view, removes any point in discussing where it might or might not have been jammed into a neck vein. Because if it had been in some way fixed into a neck vein, it would not reflect the central venous pressure, it would not have an arterial and a respiratory trace. If it did have that trace, which Dr Taylor tells us it did, then by definition it was reading a central venous pressure reflection. So that then leads you on the troubleshooting system to then deciding: well, if that's the case, it must be something to do with the re-zeroing.

The way that one would test that electronically -- one other possibility would be that the actual device which interprets the pressure transducer and turns it into a trace on the screen may be wrong. What one would do there -- and I think this was also referred to in the

evidence -- is because the child also has an arterial pressure trace using an identical bit of electronics -- which you know is working because you have no doubt that the reflection of that trace is clinically sensible and appropriate -- you would simply swap the leads and put the venous pressure lead -- the transducers are identical -- into the electronic device and check that.

My reading of it is that that was done. So we now know that if you accept that evidence, that the trace must have been central venous because it had a wave. The electronics must have been working properly because they checked that in that way and, therefore, to my logical conclusion, either the values were right or they were wrong because of a zeroing error, and that's what led me to look at this and get my spirit level out and check that possibility.

Q. Thank you. Could I just ask you, if your hypothesis is correct and if Adam's actual central venous pressure would be as reflected in your bottom graph, what is the significance of the level that you see there at 10 o'clock, which is just below 25? What's the significance of that?

A. That would be a very high central venous pressure. I mean --

THE CHAIRMAN: Is that effectively a fatal central venous

pressure?

A. No, no. No. Could I ... It would help me to talk you from the beginning of the graph and then I could explain that, if that's helpful.

The central venous pressure that you would expect to find in a child taken to theatre who was either in fluid balance or a bit replete, or even a tiny bit deplete of fluid, would be of the order of 3, 4 or 5.

MS ANYADIKE-DANES: Okay.

A. What you would anticipate to happen in a normally controlled transplant situation would be for it to start off at about 3, 4 or 5 and for the anaesthetist to infuse normal saline through the procedure so that it would gradually creep up to the target value of about 8 to 10 or 12 -- around 10 -- and that it would reach that by the time that you got to the point where you can open the clamps. That's what you would expect to happen.

What's clearly different from that here is it starts earlier than the peak. That is why I wanted to start talking about it before then. The value at 7.45, when he's only been asleep for -- and we're around the time of knife to skin or whatever -- is considerably higher than you would have in a child who just arrives in theatre at that stage. That would be highly compatible

with a child who had been given fluid in the preceding period. So if you came to the theatre at 7 o'clock -- we don't have a reading at 7 o'clock because, obviously, the instrument wasn't set up then, but one would have predicted his CVP would be about 3 to 5. The fact that it's about 8 to 10, if you look at the values before and after 8 o'clock, they're around 8 to 12, that sort of range, 8 to 11, 8 to 12. Those would be the values you would expect to achieve having already filled him to the appropriate volume.

What then clearly happens on this trace is there is a continuing creep up and then a sudden or an exacerbation of that, so if you were to draw a smooth line through the 8 o'clock until 10 o'clock, there's an upward curve. That would be compatible with a child being given too much fluid. So I think that around the 10 o'clock time, it would be compatible with a child who had too much fluid.

The chairman asked about the clinical implications of that. The clinical implications of that would be very, very different if the child was got into that state by being given saline or being got into that state by being given fifth normal saline. If that child had reached that state getting saline, then you have one problem, which is fluid overload, but not a perturbation

of his sodium concentration. At, let's say, 10 o'clock you'd then have a child with too much fluid on board, but his sodium was all right and there would be no fluid shift in and out of his cells and we wouldn't be discussing anything to do with cerebral oedema.

What we would then be discussing as a clinical problem, and the reason why you would want to avoid this situation, would be that when you have just too much fluid in your bloodstream, rather thanking it moving it into the cells it moves to the spaces in between the cells. You have fluid in the plasma, fluid in the cells and then fluid -- 15 per cent of your body weight is fluid between cells. The easiest place in the body for fluid to move to between cells is actually to move into the air spaces in the lungs. So the air spaces in the lungs would actually become soggy and water filled and then the lungs can't work properly. That is pulmonary oedema.

It's unfortunate that the term "oedema" is attached to cerebral oedema, pulmonary oedema and peripheral oedema, which is swelling of the -- as they each have a different physiological mechanism and it's very important because I'm talking about pulmonary oedema. Cerebral oedema has different mechanisms. If we had reached that just by fluid volume, that would be

a serious risk of pulmonary oedema. If I was in a clinical situation like that and it had happened like that, what we would then be discussing is avoiding pulmonary oedema, which would be altering ventilation and dialysing and getting fluid off. It would be a process to do with altering fluid volume and managing his lungs.

However, if this situation had been achieved by giving effectively a small amount of normal saline and a very large amount of water, which is exactly what happens if you give a large volume of fifth normal saline and the 0.18 solution, then partly you would have a problem of the pulmonary oedema, but in addition to that, much more importantly, you'd have the fluid shift that we've talked about into the cells themselves, and that could -- it would produce cell swelling, it would produce brain swelling and if it produces enough brain swelling, it will produce high pressure in the head.

It's important to understand that at this point because what happens after 10 o'clock -- I apologise, if this is kind of a complex clinical scenario, but it is a complex clinical scenario. If I go back to what would happen if this had all been saline, that pressure would have maintained at 20 to 25 and that would have continued at that level until the volume had been

dispersed from the bloodstream and we dealt with the pulmonary oedema.

The reason it falls -- and again I'm giving a clinical opinion here. My explanation of why it would fall at this point is two things. First, the water would be moving into cells and out of the interstitial space, so there would be less propensity for it to be causing pulmonary oedema and a much greater propensity for it to be cause swelling of all the organs, including the brain. That would then take fluid out of the vascular compartment and would gradually reduce the pressure.

The second thing is that if it did cause cerebral oedema, it would also be likely to cause or could reach the point where it would cause brain death. When we refer to brain death what we're talking about is the death or the cessation of function in the most primary part of the brain, the brainstem.

The most primary part of your brain, the brainstem, contains the neurological control mechanisms for basic functions such as breathing, maintaining your blood pressure and so on and so forth. At that point, your whole physiology is likely to change and really dealing with -- there is a science, it sounds macabre, but there is a science of dealing with the medical management of

people who are brain-dead. That science has been developed in order that people who are presumed to be possibly brain-dead are managed in the hope that you can make them recover. People that have brain death and that are potentially going to -- whose relatives want them to become donors may be kept alive on a ventilator while those preparations occur.

So people now are quite skilled at managing people who are brain-dead. We have a lot of experience of that. During that time, all sorts of physiological changes occur and the normal responses of maintaining the CVP and so forth will all change. So I think what happens after 10 o'clock or after, you know, around that time, is very complex in a child who not only has an expanded blood volume because of fluid, but also has a low sodium, will have expanded volume of organs, including the brain, and whose brainstem is no longer driving their physiology in the normal way.

So I think that after that, it becomes a highly complex -- but that trace to me is best explained by ... It would fit with what I believe I understand about Adam's process.

Q. What would have been your response if you'd seen a trace like that? I'm talking about the bottom graph.

A. If I'd seen a trace like that at -- it depends what

time, obviously.

Q. Of course it wouldn't appear exactly like that because it's sort of -- there's a continuous display and then there's a spooling out of the paperwork.

A. Yes. Not only that, but it would depend on what time I was asked to see it because I could only -- I couldn't see the -- at the moment I can see the whole graph until midday.

Q. Exactly, yes.

A. If I was asked, for example, to see that at let's say 9 o'clock or 9.30 and could look at the trace up to 9.30, I would be surprised that it started off at high as it did and I would be questioning why. I would be suggesting this child did not need any more excess fluid. The child has obviously, by 8.30, got a CVP as high as you would already want it for opening the clamps, and you certainly wouldn't want to continue giving fluid.

Q. Let's go to 10 o'clock. The reason I say that is because, yesterday, the chairman was asking for when it might have been that Dr O'Connor noticed the 30 and then went to speak to Dr Taylor and got the explanation that he gave her. And what I didn't have to hand then was actually the compressed printout. When I went back to her evidence I realised that we had put to her that

compressed printout. If she saw the 30, given that she was looking at the display -- looking at the number, sorry, rather than -- so she saw that number, then she would have to be there at round about 10 o'clock and I think they accepted that.

A. Yes.

Q. If you're looking at it at 10 o'clock, you have all those previous values and you see how, on any graph, how it's going up and it reaches that point, whether it be the just below the 25 or it's just below the 35. So what is your response when you see that as the nephrologist?

A. It's a bit of a complicated question because if you're asking me what would my response be to see a graph like that and believe that it was valid, I could tell you that. But I think the situation that Dr O'Connor was faced with was that there was a value like this and we don't believe it.

Q. In fairness to Dr O'Connor, she was not presented with a value of -- as it appears on the screen it's just over 30, I think. She wasn't presented with that as being an accurate value. What she saw was that figure, she asked Dr Taylor about it, he gave her an explanation saying it had started at 17 and it wasn't accurate for the reasons that he gave, which you have been explaining to us and

that he was using it as a marker for relative change, effectively.

So if you have received that explanation, do you go through the consideration that you just explained to the chairman about whether it could, in fact, be jammed up against the wall of a vessel in the same way as Dr Taylor has told you? How serious is it that, whatever its reason, you have a figure that comes up at that level? What do you do in the operating theatre?

THE CHAIRMAN: Does this not take Dr Coulthard back to what he said yesterday afternoon, which is that he didn't ... Correct me if I'm wrong, but what I understood you to say yesterday was that you don't accept that Dr O'Connor should have listened to what Dr Taylor had told her and then said, "That's all right then", in terms, and move on.

A. That's correct. So to summarise what I would do and to agree with that, if I had been confronted with an apparent reading like that and been told that it wasn't valid, I would first of all look at the trace. So the simple answer is I'd troubleshoot it and try and get the true value. And in fact, if there was a trace -- the only evidence about that that we have is that there was a trace -- I would then have wanted to re-zero it and so forth. Obviously, if one had then re-zeroed it and

looked for those errors, we would have found the true value.

MS ANYADIKE-DANES: The reason for asking you is that yesterday you said that whenever you discovered it, you would need to do something about it --

A. Absolutely, yes.

Q. -- but for slightly different reasons, depending on when it was. And I think where some people wanted some guidance is if it is happening at 10 o'clock, what are your options?

A. Right --

Q. I mean you have to try and find out why it is happening, but what are your practical options?

A. Okay. Let's say that we go through that, we troubleshoot it, we find that actually it was zeroed wrongly, we look back at the trace and find it's now 25, ie it's way over where it should be and they're about to open the clamps, my thought process is then -- because obviously, at this point, I'm presuming Dr O'Connor was not aware of the potential hyponatraemic component. But at that point I would be saying to the anaesthetist: why is this CVP 25 or 22 or whatever it is? What is happening? What are the options? And then the options would be: how much fluid have you given and why are you giving any fluid when the CVP was already high enough

and you're still giving fluids? Why are you doing that? And that would lead to a discussion about what fluids they were giving and hopefully one would then move back to realising that the wrong fluids were given and one would address that.

So it would initiate a whole process of logical thinking, but each step is kind of dependent on what the response was to the next question.

Q. I understand. As you just put it, it's roughly 10 o'clock, the clamps may be being opened or released at some point soon.

A. What would I have advised? Well, the first thing is that if we leave aside the knowledge about the nature of the fluid and we're just talking about the CVP and the clamps being opened, the risk of a high CVP, forgetting the sodium element, is pulmonary oedema. The risk is not a problem to the kidney. What the -- the reason that we're worried about the CVP with a kidney is if the CVP is too low, that would indicate there's not enough blood circulating and there's a risk that the kidney's going to clot. There would be no reason not to continue with the surgery. You'd say: you wanted pressure, you got plenty of pressure, the kidney will be perfused nicely, you carry on with that, but meanwhile let's think about how we're going to manage his lungs.

And the management of pulmonary oedema is -- you don't want to get into that situation, but it's completely manageable. It would be reversible. So in that situation, if the only thing that had happened was that he had reached a CVP of 20-odd because he had been given too much fluid but he didn't have a perturbation of his sodium, then (a) you'd carry on with the surgery, (b) you'd discuss with the anaesthetist how we were then going to manage his chest, which would need a change of tack in management, but it would be completely reversible --

Q. I understand?

A. -- and leave no permanent damage.

Q. If it leads to a discussion as to the fluids that have being given and that introduces the issue of low sodium fluids, you're facing another prospect. It's not that we're potentially dealing with pulmonary oedema here, we're dealing with something else; what then do you do? I can see what you say about the first instance. Press on with the surgery --

A. Press on with the surgery, make sure he doesn't get pulmonary oedema, ventilate him appropriately and so on and --

Q. Understood. Second option?

A. Well, what's his sodium? What is the plasma sodium?

That would be my next question.
Q. Right.
A. "What was your last measurement of his plasma sodium?", would be my next question. If you're giving that much fluid, hyponatraemic fluid, you are going to make him hyponatraemic. "What is his sodium?", would be my next question.
Q. Okay.
THE CHAIRMAN: There has been a debate between you and the other experts, doctor, about at what point Adam's condition was irreversible.
A. Yes.
THE CHAIRMAN: When I asked you a few minutes ago about whether that reading at about 10 o'clock of about 30 was fatal, you said: no, if it was normal saline, it would be reversible.
A. If it was due to -- that's right.
THE CHAIRMAN: In this scenario, you have a reading at 10 o'clock. It appears to be around 30.
A. Mm-hm.
THE CHAIRMAN: And you then find it's one-fifth normal saline. Is that reversible?
A. That would depend. If you separate the medical issues, which you can do quite clearly, compartmentalise them. You have dealt with the surgery. The pulmonary side of

it is an irreversible problem. My question to him would be: what's the plasma sodium? If it was very low, it could ... Then I'd be very concerned. Let me just talk about what's certain and what isn't certain.

What is absolutely certain is that if a child's plasma sodium falls and falls fairly rapidly, what is absolutely certain is that it will mean that fluid will move into the inside of the cells of the body and that it will cause the brain to swell.

Knowing the plasma sodium at that point this time will not tell you whether that has happened. What it would tell you is whether there is a propensity for it to happen. In other words, at that point, it could have been, in theory, that you could have found that the sodium was low, that therefore the brain will have swollen, but it could have been that it had swollen to a degree which had not raised the pressure sufficiently to cause brain death. So at that point, what you would do -- I mean, the question is what I would do then as opposed to what I would have done if I know everything that I know now. If I knew everything I know now, I would say this child must be brain-dead. But in that situation I would be saying: this child is at serious risk of cerebral oedema, what is the sodium? Let's deal with it. And in fact, in the meantime, if you knew

he had been given fifth normal saline while you're waiting for a plasma sodium to be taken, you would immediately stop the volume going in and, secondly, give twice normal saline, ie much stronger than -- not just change from fifth normal saline to completely normal, as it were, but you'd give a higher sodium concentration.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Okay. Thank you.

A. I mean, just to make it absolutely clear, obviously at that point in time it's luck whether or not you have reached the point where it's still reversible or not because, you know, you prospectively have to make that assumption. I think at 10 o'clock, had Dr O'Connor done all that, it would have made no difference because I think that the clinical scenario that we've read indicates to me that he will have been brain-dead by then.

Q. Thank you. Can I just pull up 058-003-005? This is an anaesthetic record sheet that we've all seen so often. Can you see, two-thirds of the way down, there's a line that says "CVP"?

A. Mm-hm.

Q. Perhaps just highlight that.

A. Yes, I can see it.

Q. That's completely unfilled.

A. Sure.
Q. Is that something that you would expect should be filled, just as all the other measurements are?
A. Yes. I mean, you would expect it -- conventionally it would be filled. What matters is what's in the doctor's head rather than what's on the bit of paper. And if somebody is watching the trace and it's electronically available afterwards, then you could argue whether that matters or not. But what matters is that that is known. Yes, you would expect it to be written down.
MR UBEROI: I'm happy for the question to be asked and the answer given, but it is the anaesthetic record and I do wonder really whether that was more a question for the consultant anaesthetist.
MS ANYADIKE-DANES: It may be, but Dr Coulthard has been dealing with a number of matters to do with CVP.
A. Perhaps from that perspective, I understand the observation made. From the perspective of what was happening at the time, if I arrived and I knew and I could have a conversation with somebody who could tell me what the CVP had been, I'd be happy at that. I would not be happy that this would be the permanent end trace because I would not then be able to go back to this information later and see what had happened. Either it

has to be filled in afterwards from the trace or it has to be filled in at the time. It has to be filled in.

Q. At some point?

A. At some point, it has to be filled in so that that information is available to me when I look at the child the next day and the CVP is doing whatever it's doing. I'm just trying to say that as long as I could be given that information at the time and it would be available permanently in a convenient form, ie they either had to write it at the time or write it in afterwards, that wouldn't bother me. What would bother me is that it is known and it is ultimately recorded.

Q. Ultimately recorded, okay. I also need to ask you, just while we're on recording things, to do with the timings of fluids. I wonder if we can go to Professor Savage's statement at 002/2, page 19. I think it's the answer to 1(c), which is up at the top. On the third line:

"Intravenous fluids at 25 ml per hour for 6 hours."

That's what he was indicating should be given. And then:

"When tube feeds were finished, 2 hours of intravenous fluids at 75 ml per hour ..."

So he's actually calculating it there, but --

A. Sorry, could I clarify? When this was in relation to the preoperative --

Q. Yes. It's the evening of the 26th, 26 November. So that's a reference to intravenous fluids at 25 ml per hour over six hours. Then if we go and look at the medical notes and records, let's start with 058-035-144.

So this is the evening, you can see the date of 26th. The note in the middle, which starts at 9.30, that's a note that's made by Dr Cartmill, the SHO. There you see:

"To have IV fluid at 75 ml per hour (maintenance)."

So if you bear that in mind, it doesn't say when it starts, it doesn't say for how long. That's just her note in the medical notes and records. Then if one goes to the IV sheet, 057-010-014, if you go to the top there, you can see one lot of 500 ml. It tells you the type, the rate. Start and finish not recorded, prescribed by Dr Cartmill.

Item 2, same thing, start, finish not recorded.

Then if we go to 057-010-013. Then you can see if you look at 2300 hours, you can see 20 ml an hour. Okay? "Fifth normal at 20 ml an hour."

Then finally, if you look at 057-014-019, and flip that around, this is the nursing record. You can see under the 26th November at 10 pm:

"Clear fluid via gastrostomy, 180 ml an hour. IV fluids at 20 ml per hour."

Then his normal peritoneal dialysis until 6 am. Then, of course, you have the information about the cannula tissuing. The point I'm going to ask you -- because this is your territory as a nephrologist, getting the child ready, fit and in the best possible condition -- slightly replete, I think you called it -- for his surgery. Do you expect these fluid prescriptions and administrations to have attached to them the times when they should start and the times when they should finish? Should that be recorded anywhere?

A. Yes. I'm just kind of -- give me a minute just to get to grips with the ... Sorry, this last one is a nursing sheet, yeah? That's a --

Q. Yes, this is a nursing sheet. It's the only other place you see a reference to 20 ml an hour, so far as I can tell.

A. I think in clinical practice, what you would expect -- there's obviously a discussion, the junior doctor is instructed that we're going to give 75 ml an hour of intravenous fluids and then, reading into it, there's then a discussion that actually we'll give that as 25 ml an hour and some oral fluid until 6 o'clock, and then ...

Q. Yes. The only issue is, do you record the time?

A. That sort of discussion would be done in this kind of

way. You'd have, literally, a debate about it --

Q. Yes.

A. -- and that would be clearly made to the nurses. Actually, the timing -- the practice is that, in reality, at this time of night, 11 o'clock at night, whenever it was that you actually did it, the doctor probably would just write a 500 ml bag of the appropriate fluid and give it to the nurse and say: can you start that? So although that's not a written time and maybe, in theory, you should, what's happening here is probably common practice and probably quite acceptable practice in the sense that it's safe in that somebody is giving an instruction to act immediately. They may not say, "I want you to give this now and it's 11 o'clock and I'm going to write it down", they say," I want you to give this now". Because the timing of that, the recording of that, is assumed by the doctor to be then made in the child's records by the nurse.

So what you would expect is another piece of paper --

Q. That's what I'm getting at.

A. -- which would be the nurse then says on the fluid chart: started at this time giving this amount. Then each hour, he or she would written down the actual

amount administered. That's appropriate practice. So insofar as interpreting what's happening, I don't have a problem with this.

Q. You have actually articulated it. What I am asking you is -- and I think you have articulated this -- is whether you would expect the actual time at which you started to administer to be recorded somewhere.?

A. Yes, but not on the -- the first sheet you gave me was a doctor's prescription sheet. I'm happy that it's not on there. In practice -- was there a date? It would be nice if there was a date. I can't remember whether there was or not. I don't think that putting the time on there is essential within the process. What is essential is that it is recorded by the nurse on the fluid chart.

The one that's up at the moment --

Q. What I have given you, Dr Coulthard, is all the records that we have as to the actual administration of those fluids.

A. Okay.

Q. That's why I'm asking you: should there be some record somewhere --

A. Is there not a fluid chart which gives -- can you take me back one?

Q. There is. I've just pulled it up. [OVERSPEAKING].

A. If you give me a second, could I just look at the last document?
Q. Of course.
A. That is the fluid chart where it will be recorded. I can't see the whole thing. That's better. Right. So we've got 2300 hours, the fluid type and ... I can't see the ... To my reading of this, it looks to me -- I would interpret this -- yeah, that's better, thank you -- as if it ... I don't ... It's not very clear, is it? What one would hope to see is for each hour, so for the hour, 23 to 24, in the horizontal row labelled 24 on the left would be -- that would be what the nurse would write at midnight and it would reflect what happened between 11 o'clock and midnight. So the hour that -- the row labelled "24" really means "23 to 24". What you would expect to be there on the left-hand side on that -- in ... It depends how it's -- quite how it's delivered. But you'd expect at least somewhere the volume of fluid that was given that hour in a column, which was clearly labelled "intravenous fluid".
Q. Yes. And what you can't see is any reference to the 75 ml an hour.
A. No, I can't see that at all.
Q. That's the point.
A. No.

Q. Thank you.
A. It's not clear what's happened there to me.
Q. Thank you. So my question to you was: since that had been prescribed by the doctor, there should be some reference to when it was administered and, presumably, if it wasn't administered for some reason, that should be recorded in the notes?
A. Yes.
Q. Thank you.
MR FORTUNE: Before we leave that document, could we ask Dr Coulthard whether the document he's looking at is a document he would expect to be completed by a junior doctor or a nurse?
A. The document that's up at the moment, I would expect to be completed by a nurse.
MS ANYADIKE-DANES: Thank you.
A. I know I haven't been asked another specific question, but I don't want to leave it in a confused state. It does seem to me that the information that we have here suggests that the junior doctor was originally considering -- or a discussion had led to considering giving 75 ml an hour and then there's a change to giving the fluid, from what we hear from Maurice Savage, to giving it intravenously at 25 ml an hour and the rest orally or nasogastrically. That's how I'm interpreting

that previous statement. I would have liked to have seen that written as an instruction by the doctors.

Q. Yes. I think originally the 75 ml was to start after the feeds, which would have been in that 5 am to 7 am period.

A. Okay, so what I would like to see written -- I don't know. I'm left confused at the moment what the nurses are expected to deliver from 11 o'clock when the drip is erected. What we have on the prescription chart ... I'm trying to remember the prescription chart, but I can't remember what that said.

Q. We can go back to the prescription chart, I think. There we are.

A. Okay. So the preparation chart written by a doctor and signed by a doctor --

Q. Yes.

A. -- indicates 75 ml an hour. I can understand that that may have then been changed verbally to 25 ml an hour or a lower volume and that that may have been what the nurses were trying to achieve. But from the paper trail that we have here, the instruction, the written instruction, signed written instruction, is to deliver it at 75 ml an hour. So if in fact that was an initial thought and then it was decided to change to 25 while he was having gastric feeds, then that wasn't conveyed in

writing.

Q. It's really a recording point and I'm just trying to clarify from you your views. It seems to me you're thinking it's not very clear.

A. It's not very clear.

Q. The other thing to point out, of course, is that at the time -- I think it was 9.30 when this was being prescribed by Dr Cartmill -- at that stage, they had no idea whether the operation would actually be going ahead and, if it were going to be going ahead, when. In any event, all I was trying to ask you since you're in the position of the nephrologist dealing with this run up period and I think your view is that it is not very clear --

A. If I could just say that if I were to, for example, come in on this to take over as the consultant taking over this information, I would not really understand what's going on and I would want it written clearer than this. I wouldn't know who was prescribing what volume for how long --

Q. Yes.

A. -- on the information I have.

Q. That's actually exactly what happened. Not that a consultant came on, but a different SHO came on. Dr O'Neill came on at roughly 10 pm. So not only

do you have the records in this state, if I can put it that way, but you actually have a change between whoever had originally prescribed, which is Dr Cartmill, to Dr O'Neill, who's going to be there during the late night into the early morning shift.

A. This would be very confusing.

Q. Thank you.

MR FORTUNE: To assist Dr Coulthard further, having referred to Dr O'Neill, could Dr Coulthard first be shown 059-006-009? There's also some writing on the back of the page, 010. That's Dr O'Neill's note at 11.30.

MS ANYADIKE-DANES: I think the back of the page is coming up, Mr Fortune, next to it. There we are. It doesn't take us much further.

MR FORTUNE: Then we move on in time to Professor Savage's note, 059-006 --

A. Would you like me to read -- before it moves on, I was listening to you and I haven't -- could I just have a minute to read that? Thank you. (Pause).

Okay, thank you.

MR FORTUNE: Could we move on in time to Professor Savage's note, 059-006-011? So we've moved on to some time around 1 am to 2 am because, by then, the cross-match has been effected. Would you like to read that to yourself?

A. Thank you. (Pause).

Okay, thank you.

MS ANYADIKE-DANES: Yes?

MR FORTUNE: Dr Coulthard, would that assist you in understanding what the fluid regime overnight was to be?

A. No.

MS ANYADIKE-DANES: Thank you.

A. What this note is is a very clear note and plan about the situation regarding going to theatre and various important studies, the viral studies and so on, that are necessary to get clear a plan of repeating his blood tests and then a list of drugs to be administered in theatre, which central line he would like given and a discussion about which drugs to use, including mannitol, his height, weight and surface area to aid drug prescribing, and a post-operative drug. There's no reference here to fluid management.

Q. Thank you. I wonder if I could now ask you another question in relation to what you might have expected Dr Savage to have known. Dr Savage was asked directly whether he appreciated that it was possible during major surgery, or during surgery of this sort in particular, for the native kidneys to just simply shut down, which is something which you had first raised in the experts' discussions in Newcastle -- I think during the 9 March

discussions. The reference for that is 307-008-193 at lines 1 to 3. Just so that you have it, to remind you, it says:

"The kidneys are functioning on a real knife edge and almost anything that happens to that child is capable of just switching their kidneys off because they are so dependent and just not robust at all."

That's what you were saying. That was put to Dr Taylor as to whether he appreciated that that might --

THE CHAIRMAN: Sorry, I think you started with Dr Savage.

MR FORTUNE: Are we talking Dr Savage or Dr Taylor?

MS ANYADIKE-DANES: I'm so sorry. I meant Dr Taylor. It wasn't put to Dr Savage; I meant Dr Taylor.

MR UBEROI: [Inaudible: no microphone] I think a question about the expectation of the level of knowledge of the consultant anaesthetist was one that was properly for your expert consultant anaesthetist and Dr Haynes, in fact, was asked this and, in my submission, it wouldn't be helpful for Dr Coulthard to be asked the same question.

MS ANYADIKE-DANES: I'm not sure that Dr Haynes was asked that, but in any event, even if he was --

MR UBEROI: He was.

MS ANYADIKE-DANES: Even if he was, the reason why I would

be asking the consultant nephrologist that is because Dr Coulthard has given evidence as to the discussions that he would have with the paediatric consultant anaesthetist and what level of understanding he would expect that person to have. Because of the way the question was framed is: is that something you were expecting to know about and potentially factor into his fluid calculations? That when he's calculating maintaining his fluid balance, he should bear in mind it is possible that the native kidneys might not actually produce any urine at all during that period. So I think it is an appropriate thing to ask the nephrologist as to whether he would expect the person he's talking to to know that.

Dr Coulthard?

A. Thank you. The answer is that I would expect it, but I would like to just expand that, if I may.

Q. Yes.

A. There's an important general principle here, which is that any patient of any age with normal kidneys, if exposed to stresses and trauma, such as might occur during an anaesthetic, one of the most vulnerable organs in your body in response to a fall in blood pressure, which is a very common consequence of the use of anaesthetic drugs and anaesthesia in general, is that

anybody's kidneys may function less well. If you use a colloquial term, they may slow down or even, in extreme circumstances, stop.

You take any clinical situation, a woman having a Caesarean section who loses a lot of blood if her blood pressure falls, her kidneys will be very vulnerable, and I would expect any medical student to know that. I would certainly expect all anaesthetists to be totally aware of that at all times because it's a working day-to-day concern that they will always have.

It would seem almost too obvious to say that if you are dealing with somebody whose kidneys barely work to the point that they have required dialysis for some time, that they would be at least as vulnerable and almost certainly much more vulnerable than somebody with normal kidneys. I would take that as being a basic piece of understanding if I spoke to an anaesthetist. For example, when discussing fluid management with an anaesthetist, I would not remind them that a child whose kidneys barely work like anybody else is vulnerable to them slowing down, but even more so because they are so vulnerable or, as I put it in that phrase, on a knife edge.

Q. Thank you. If I could just continue that theme as to what you would have expected Dr Taylor to know or

appreciate. Dr Taylor, as you know, has made a statement on 1 February when he conceded certain errors. In fairness to him, he has frankly conceded errors in the course of his evidence. You, yesterday, were explaining how illogical you thought his approach had been, given certain of his assumptions, if you like, as to the fluid deficit. You were therefore going on to say if that's what you thought was happening, that would not be an appropriate way to deal with it, irrespective as to whether you thought the urine output was 200 ml per hour or not.

A. Yes.

Q. What I am going to ask you, though, is even though Dr Taylor has conceded that he made certain of those errors and he doesn't know why he made them, do you understand the explanations that he gave over the intervening period from when he first wrote down what he did and why he did it until that ... I saw that out of the corner of my eye.

MR UBEROI: [Inaudible: no microphone] I was grateful of the explanation and the slight re-framing of the question which I objected to before in terms of moving to a stage where the question was being asked from the point of view of a nephrologist handing over to an anaesthetist in theatre, "What would you assume to be known?", "What

would you not assume to be known?". This, in my submission, really is now a question of what was going on in Dr Taylor's mind, which is not a question for expert evidence; it is a question of fact for you, sir. Not only is it a question about what is going on in Dr Taylor's mind, even moving beyond that objection, it is one that was more appropriately put to the anaesthetist, Dr Haynes. Indeed, I didn't object when it was put to Dr Haynes.

THE CHAIRMAN: But is there not another point, Mr Uberoi, that Dr Taylor doesn't understand his own explanations in the intervening period? He doesn't try to stand over them. I understand his evidence to be that, subsequent to Adam's death, he made a statement to the coroner, he made a police statement, he made various statements to the inquiry. Effectively, he doesn't stand over them and he doesn't -- he says that his statements contain things which himself described as outrageous. So asking this witness if he understand the explanations which were given by someone who now doesn't stand over them and who accepts they were wrong doesn't seem to me to be advancing the inquiry, unless Dr Taylor is standing over the explanations that he gave, which I do not understand him to be.

MR UBEROI: No, sir, he's not.

THE CHAIRMAN: Then if he doesn't stand over them, they don't make any sense. Sorry, I understand that he himself has conceded that his explanations do not make any sense.

MR UBEROI: The basis of the concession, I'm sure, or one of the important pieces of evidence, in my submission, that you heard was that the comment he did make on them was that his thought processes were clearly disturbed, which is why I'd bring it back to the point where that is a question point as to Dr Taylor's thought processes and his mind and not a point for expert evidence, but a question of fact for you.

MS ANYADIKE-DANES: That I understand and I would like to deal with matters in this way. I understand your objection.

Leaving aside Dr Taylor and his disturbed or not thought processes and the errors which he made, which are there in writing over all that period and which he is not standing over, then the question is this: if you are the consultant paediatric nephrologist and you are aware of those sorts of errors having been made over that period, then what is your response to that?

THE CHAIRMAN: After the event?

MS ANYADIKE-DANES: Yes.

THE CHAIRMAN: We're moving on to something which is rather

different.

MS ANYADIKE-DANES: I took the objection so I'm moving on to a different point.

MR FORTUNE: I'm now getting to my feet. Over what period and when are we now talking about?

MS ANYADIKE-DANES: That was the general area that I was moving into, and then I was going to put some specific documents.

MR FORTUNE: Well, could we take it in stages?

MS ANYADIKE-DANES: Yes.

MR FORTUNE: Otherwise, this is a very large question that's not going to help Dr Coulthard and most probably will not help the chairman.

MS ANYADIKE-DANES: Well, I hope the chairman will tell me if I'm not assisting him.

THE CHAIRMAN: Let's see where we're going.

MS ANYADIKE-DANES: The beginning of it is the statement that Dr Taylor provided to Dr Murnaghan for the purposes of complying with the coroner's request that all the clinicians involved produce statements. That can be found at 011-002-003. That's the start of the letter. In fact, you can see it says, "Statement". And he describes what he was doing.

MR UBEROI: Sorry, I'm slightly concerned we're moving back into territory that I objected to and which I understood

both the chairman and my learned friend agreed with. I thought we were moving on to a question for the nephrologist of what he, as a nephrologist, would have done on a letter from his point of view. So if this is a letter from Dr Taylor, and I can't yet see the second page, but the way it's been introduced by my learned friend is that this is a letter from Dr Taylor then I think we are creeping back in to the same territory --

MS ANYADIKE-DANES: Let's go to the second page, 004. There we are, "cc Dr Savage, Dr Gaston, clinical director".

MR FORTUNE: I rise again because I'm concerned as to exactly what Dr Coulthard is being asked to say about this letter. This is Dr Taylor's recollection of events. In what circumstances can Dr Coulthard, as the consultant nephrologist, properly be asked to comment on Dr Taylor's recollection of what took place in that theatre that morning?

MS ANYADIKE-DANES: What I'm going to ask him to comment on is what he would have done if he had appreciated this is what a consultant paediatric anaesthetist who works in the Children's Hospital and is carrying on working and will, if the opportunity presents itself, carrying on working in paediatric renal transplants, what his response would be if this is what the paediatric anaesthetist is saying. This goes to the knowledge that

he is demonstrating to Dr Savage about actually what happened. This is his knowledge about the medical processes and so forth. And just as in the same way as it has now been conceded that what he did was illogical and made no sense, to the extent that these documents reflect that lack of logic and lack of sense, the question is: what does the consultant paediatric nephrologist do?

THE CHAIRMAN: Well, this is in terms of the continued work by Dr Taylor in the Children's Hospital.

MS ANYADIKE-DANES: Yes.

THE CHAIRMAN: And in particular, his subsequent re-involvement in paediatric renal transplants.

MS ANYADIKE-DANES: Yes.

MR FORTUNE: Is that an appropriate matter for Professor Savage in the circumstances of this inquiry?

MS ANYADIKE-DANES: Well, it was put to Professor Savage in terms when the letter that Dr Murnaghan sent to all the clinicians involved and referred to differences of view or at least potential, I think it was, differences of view between the clinicians. Professor Savage was then being asked about differences of view and I'm developing that as to: if you know there are, what should you be expected to do about them, particularly if the person carries on working in that field?

If you were to tell me that that's a bit of a governance point, I would accept that, and it is a bit of a governance point, but we have covered that kind of governance area with other witnesses and I'm simply taking the opportunity to advance it here rather than call somebody back in the governance hearings.

MR FORTUNE: Sir, this is essentially a matter for governance. Mr Murnaghan represented the trust, he was pulling together statements from the clinicians involved with a view to their submission to Her Majesty's Coroner. It was up to the trust to take action against any of the clinicians. It would not be appropriate, in our submission, for Dr Coulthard to be asked what he would do if he was presented with the letter written by Dr Taylor. After all, Dr Taylor is answerable to the Director of the Anaesthesia Directorate, if there was such a person in those days, and certainly answerable to the Medical Director of the trust and, no doubt, to those responsible for employing him.

THE CHAIRMAN: Well, my own view at this stage is that it goes further than that. Professor Savage was developing the nephrology service. After Adam's death, he accepted the inquest verdict. Whether we have to wait until the inquest for Professor Savage to form a view about why Adam died is a different question. But at the very

least, he accepted the inquest verdict. Mr Keane accepted the inquest verdict. And so far as I can see from all the papers we've gathered, the Royal allowed Dr Taylor to continue to operate as an anaesthetist and, in particular, it allowed him to continue to operate as a paediatric anaesthetist in renal transplants. Your client must surely have been involved in that. And if your client had concerns, as he surely did if he accepted the inquest verdict, what I will hear either in this hearing or in the governance hearing is why nothing was done.

MR FORTUNE: Sir, even if Professor Savage had concerns, and in particular that he knew that Dr Taylor did not accept initially the medical cause of death and then, in time, the verdict of Her Majesty's Coroner, and that is the evidence, it would not be up to Professor Savage to stop Dr Taylor carrying out anaesthesia for any child, a patient of Professor Savage. That would be up to the hospital, and strictly speaking, that is governance, sir.

THE CHAIRMAN: Well, we can get into that at the governance stage. The idea that Professor Savage would not raise a concern --

MR FORTUNE: Well, even if he raised a concern, it's not ultimately up to him for action to be taken.

THE CHAIRMAN: Well, you're quite right. He's not the employer, but it is up to him to raise a concern if he has a belief or a view that a child has died primarily because of inadequate anaesthesia provided by Dr Taylor. To those of us outside the Health Service, it would be astonishing if he did not have an obligation to raise that.

MR FORTUNE: Well, you will recall and will have read Professor Savage's account of what happened. Ultimately, those letters, submitted by Professor Savage, Dr Taylor and Mr Keane, went to the hospital authorities represented here by Mr Murnaghan. It is our submission that it was up to the hospital authorities to take action. It would appear that no action was taken.

THE CHAIRMAN: Well, yes. I have seen letters which went to Dr Murnaghan. These are for the purposes of the coroner.

MR FORTUNE: But also, that information -- and let's be realistic -- would have been shared with the medical director or should have been --

THE CHAIRMAN: It should have been, yes.

MR FORTUNE: -- and those responsible for employing any of the clinicians.

THE CHAIRMAN: Yes.

MR FORTUNE: And it would be reasonable --
THE CHAIRMAN: That's the end of Dr Savage's potential involvement in this, that he's written a letter?
MR FORTUNE: I'm not saying it's the actual end, but the ultimate decisions are taken either by the board or indeed by the medical director because it would have been open to the medical director to have suspended Dr Taylor or, indeed, if he had any real concerns, to have referred him to the General Medical Council for investigation.
THE CHAIRMAN: I think I have already got Dr Haynes' evidence on this, which is to the effect that he would have had an informal meeting after the event with -- as a medical director or clinical director, he would have had an informal meeting with Dr Taylor and, if Dr Taylor had appeared not to have accepted what had gone wrong or not to have recognised what had gone wrong, he would immediately have brought an end to the informal process and would immediately have started a formal process.
MR FORTUNE: I wouldn't disagree with that, sir, but here is Dr Coulthard, who is not wearing the hat, as Dr Haynes was, of being a clinical director.
THE CHAIRMAN: Yes. Look, my immediate concern is I'm not sure that I need really to hear much evidence on this, if any evidence, from Dr Coulthard, because I -- this,

in a sense, is already teed up for the governance hearing, the state of the evidence that we have, and I'm not sure that Dr Coulthard can really add very much to that.

MS ANYADIKE-DANES: Of course, it's entirely a matter for you, sir, and I do understand what you say about the governance hearing. But in a sense, what you have heard is you have heard Dr Haynes viewing it from the perspective of what he would have done as the anaesthetist. What I am inviting Dr Coulthard to express a view on is what he would have done as the nephrologist.

When Dr Coulthard started his evidence -- and in fact, in large part, because it was thought that it was helpful, there were parallels between Dr Coulthard starting up right from scratch almost the paediatric renal transplant service in Newcastle and there seemed to be parallels between that and the efforts and the work that Professor Savage meant to start up a similar thing. So in a sense, there's Professor Savage, who really is it in terms of that paediatric renal transplant unit, and Dr Coulthard is in a rather similar position. All I'm saying is, you've had the benefit of Haynes on Taylor, if you like, and I think it might be helpful for you to have somebody who's very closely

in the position of Dr Savage and what he would have done about somebody who is providing those services as part and parcel of him delivering paediatric renal transplant service.

THE CHAIRMAN: Okay. I don't think Haynes on Taylor is quite the analogy because when Dr Haynes was giving that evidence, he wasn't giving that evidence as an anaesthetist.

MS ANYADIKE-DANES: No, no.

THE CHAIRMAN: He was giving evidence wearing a different hat.

MS ANYADIKE-DANES: Yes.

THE CHAIRMAN: So the Haynes/Taylor analogy is not comparable, if I may drop the titles, to Coulthard/Savage. It is of interest to me to know, Dr Coulthard, in this scenario where you had an anaesthetist who appeared to you to have made significant mistakes which had contributed, at least, to the death of a child, what steps, if any, would you have taken to raise concerns within the trust which employed you?

A. You want me to answer that? I'm sorry.

THE CHAIRMAN: Yes.

A. Okay, thank you. There are three stages to this. The first stage is that I would have spoken informally and

privately with the anaesthetist to try to understand his thinking and try to come to a common agreement and hopefully -- it sounds pompous, but to kind of re-educate him, as it were, to try to, in some way, get him to understand the error of his ways so that such a thing would not happen again.

If that was not successful, then I would initiate a formal concern by contacting his clinical director and I would expect that to then involve the medical director of the trust. If that was not -- if that did not result in Dr Taylor then giving ... If the end result of that was that I was still, as a paediatric nephrologist, in a trust where there was a chance that my patients would be anaesthetised by somebody who I didn't have confidence, understood the appropriate management, I would have taken that to the GMC without any doubt. There is a duty in my view. My duty as a paediatric nephrologist is to look after my patients. My duty to the trust and the management system is obviously an important part of my employment contractual arrangements, but my primary duty is the Hippocratic duty and it's my duty to my patients, and it is actually now very clear from the GMC that I have a duty as a doctor to raise with the GMC concerns if I feel patients are being put at risk.

I'm not sure whether that was so clearly stated at that time, but I would have behaved in that way.

THE CHAIRMAN: This is going to be explored in some more depth at the governance segment, as I think you're aware.

A. And I have to say that this is based on actions that I have -- you know, this is -- I have had to deal with parallel issues, not as severe as this, and this is what I have actually always believed and that's what I would have done because I've taken similar action. I have had to take similar action in relation to other professional colleagues.

THE CHAIRMAN: Mr Fortune?

MR FORTUNE: Sir, just looking at those three steps, if Dr Coulthard, in place of Professor Savage, had spoken to Dr Taylor as here, and Dr Taylor had maintained his position, then clearly there would have been a less than satisfactory outcome to this private or informal discussion. Dr Coulthard would then have gone to the clinical director or the medical director. At that point, the matter would be firmly in the hands of the hospital management and it would be for the trust to take action. The third step can be --

THE CHAIRMAN: I agree. Your client has no disciplinary power.

MR FORTUNE: The third step is whether, in 1995, it was common for doctors to report other doctors to the General Medical Council. It was --

THE CHAIRMAN: Let me pause there. There are two separate questions in that. One is whether it was common to do it, okay? Let me ask you the first point. Was it common for doctors to report other doctors to the GMC in 1995?

A. Not as common as it is now.

THE CHAIRMAN: Secondly, why was that? Why was it not as common in 1995 as now?

A. I presume it's an element of culture, but it would have been my view at that time -- and in fact I did consider such action at the time in relation ... I have done that prior to the change in the general atmosphere and the directions from the GMC. I have had to threaten to do that in my own clinical situation with the chairman of our trust.

THE CHAIRMAN: Is this a change in culture or a change in a specific duty which doctors are under?

A. If you're asking my personal position, my personal position is that my duty as a doctor has always been to look after my patients first and foremost and whether I perceive there to be a problem in doing that at the level of an individual professional colleague or

a management professional colleague is really neither here nor there. At the bottom line, if I'm being pushed in a position -- it's not quite parallel, but a similar position. If I was pushed into the position of being forced to accept by the trust I worked in a substandard of care for my patients, my children -- and that's the way paediatric nephrologists and certainly the way I think -- then I would have taken it to the GMC and indeed those discussions of that possibility have been raised by me in the past prior to the current atmosphere. I didn't have to do so, but it would have been my position and I believe it ought always to have been the position of all doctors.

THE CHAIRMAN: One of Mr Fortune's points on behalf of Professor Savage is that he doesn't have any disciplinary power or control over Dr Taylor and I presume that you would be in an equivalent position.

A. Absolutely, which is why you go through the mechanisms where the power is enshrined. But if you are thwarted -- I mean, the assumption that you go as far as the management of your hospital and no further is enshrined in the -- it makes the assumption that you can always rely on the management of your hospital reaching a conclusion which will always be the best for your patients. And if you disagree with them, then it's your

duty as a doctor to go to a higher authority.

THE CHAIRMAN: Okay. Mr Fortune?

MR FORTUNE: It's that culture that has changed in that period since 1995, is it not, Dr Coulthard?

A. You're asking my personal position? I'm not in a position to give expert testimony on what the culture is within the rest of the doctors or within the GMC. I'm telling you what my position was from the moment I qualified as a doctor and continues to be.

THE CHAIRMAN: Okay.

MR FORTUNE: Thank you, sir.

THE CHAIRMAN: Is there anything more for this witness?

MS ANYADIKE-DANES: I don't think so, no.

THE CHAIRMAN: Is there anything arising from this morning's exchanges or have the questions been covered?

MR FORTUNE: Sir, I asked you last night for Dr Coulthard to remain so that we could reflect on the evidence that he had given. On reflection, we have no questions and we are sorry to have detained Dr Coulthard for our purposes.

THE CHAIRMAN: It's lucky we didn't sit from 4.30 to 5.00 last night to finish since it has taken us an hour and a half this morning. There is no apology required. Thank you very much, Mr Fortune. We'll take a break now and we should start Professor Gross at about 12.10.

MS ANYADIKE-DANES: I wonder if we could have a slightly longer break than that. I'm conscious that ...

THE CHAIRMAN: If we take a longer break than that, are we talking about an early lunch and starting at 1 o'clock?

MS ANYADIKE-DANES: That might actually be preferable. Otherwise we'll get ourselves out of kilter.

THE CHAIRMAN: That's on the basis of your private promise to me that I'm now about to make public that he is a half-day witness.

MR FORTUNE: Is that correct?

MS ANYADIKE-DANES: There are no guarantees. One does one's best, but you go where the evidence takes you.

THE CHAIRMAN: But if we start at 1 o'clock --

MS ANYADIKE-DANES: I've got a better chance.

THE CHAIRMAN: Okay.

Dr Coulthard, thank you for your time. We'll break until 1 o'clock and start Professor Gross at 1 o'clock.

(The witness withdrew)

Timetabling Discussion

THE CHAIRMAN: Just one moment, I just want to tell you one thing. We've been looking at the timetable for the next few days and I'd like you to consider something.

If we finish Professor Gross this afternoon, we will have no witnesses tomorrow. We will not sit tomorrow. I'm afraid, Mr Fortune, that we've made enquiries.

Dr Montague, who I have already inconvenienced once, is only available on Friday so I have to take him on Friday. If we finish Professor Gross today, we will not sit tomorrow, we'll resume on Friday.

MR FORTUNE: Sir, is Dr McCallion still on Friday?

THE CHAIRMAN: Yes, but I am told Dr McCallion is a very short witness.

MR FORTUNE: We've heard promises like that before from leading counsel.

THE CHAIRMAN: I will explore that a little bit more. The short position is that we have to sit on Friday. On Monday, Ms Ramsay and Dr Webb are to give evidence. I explained yesterday that Dr Webb unfortunately can't and won't be available in all likelihood during this hearing at all. That leaves the question about Ms Ramsay. Now, Ms Ramsay had a number of comments, which were to a degree critical of nursing and nursing records in particular, Mr McAlinden. I think she's really primarily a witness who you may have issues with and also the representatives of Staff Nurse Murphy, who I think are not here today, so we'll have to contact them.

So far as Staff Nurse Murphy is concerned, Ms Ramsay did her reports, Miss Murphy replied in writing, Miss Ramsay replied in writing and Miss Murphy has given

oral evidence. And so far as Miss Murphy is concerned, for my own part, I do not believe I require Ms Ramsay to give oral evidence and I'm wondering whether she needs to give oral evidence at all. Would you like to think about that over lunch?

MR McALINDEN: I can address that this issue at this stage. I don't think it would be necessary for her to give oral evidence because I think the issues she raised have been dealt with by the nursing evidence and, in any event, the issues she has raised, in my submission, have no direct bearing on the substantive issues that you have to decide.

THE CHAIRMAN: Okay. Let's look at them. She is, to a degree, critical of the nursing records.

MR McALINDEN: Yes.

THE CHAIRMAN: One of the problems is, of course, we do not know if the records are complete, and it's been suggested to us -- for instance, by Staff Nurse Murphy -- that there are at least one or two records which she played a part in compiling, which cannot be traced. So I have to take that into account. But on the general issue of how fundamental any failures in nursing are to the issue which we're investigating, my view at this stage is they're not fundamental and Ms Ramsay doesn't suggest they are fundamental and none of the other

experts suggest they are fundamental. I am also influenced in this -- but it obviously doesn't bind me -- by the fact that, on behalf of Adam's family, a statement has been read to the inquiry to say that they acknowledge the work done by the nurses and they thank the nurses for all that they have done.

MR McALINDEN: [Inaudible: no microphone] for the care provided by the nurses.

THE CHAIRMAN: I have to be careful about that, Mr McAlinden. That doesn't mean that the nurses did everything right, but it directs me away from them as any significant --

MR McALINDEN: Certainly in terms of who is in the best position to assess the quality of nursing care, it would be my submission that it would be the mother of the deceased child who would be best in a position to discuss that from her hands-on experience.

THE CHAIRMAN: I'm not sure that's necessarily entirely right, but it's a factor I bear in mind.

Is there any other party who has a view about whether Ms Ramsay needs to be called? Let me make it clear: if she's not called I will take her report as it stands along with the other evidence.

MS ANYADIKE-DANES: Before anyone does, I'm not sure that Miss Murphy's representatives are here.

THE CHAIRMAN: I have noticed that and they will have to be contacted about that. If they have a position on this, I will report back to you. So I won't conclude a position on this until the issue has been raised with Miss Murphy's legal representatives. If they do have an issue, then she will have to give her evidence on Monday, even though we anticipate it being comparatively brief. I'm afraid it looks at the moment that whether we sit on Monday or not, we're going to have to sit from Tuesday to Friday. Mr Koffman will give evidence on Wednesday, I think. And there are some other witnesses, some of whom are likely to be quite short, who will have to be fitted in.

The one other issue, I should say, Mr Uberoi, is about some evidence has been given about Dr Taylor subsequent to him giving his evidence. In particular, evidence has been given by Mr Keane about the CVP. I know that you have already alerted me to the fact that's going to be part of a submission which you make about Mr Keane's reliability. I'm wondering, in fact, might it not be better to consider recalling Dr Taylor if he can be made available for a short time?

There is a factual issue which wasn't raised with Dr Taylor because we didn't know about it because it didn't feature in Mr Keane's statement. That is about

him calling out specifically and asking what the CVP number was on a number of occasions.

MR UBEROI: Yes, sir. I appreciate your indication that, even if he were to be recalled, it would be for a very short time. I would resist that. Firstly, on Dr Keane's evidence the submissions I would make would be internal and intrinsic to the evidence he gave and they would focus on reliability.

On your second point on whether Dr Taylor needs to be recalled for it. In my submission, if potentially unreliable evidence has been given subsequent to evidence being given by Dr Taylor, then it's not particularly fair to Dr Taylor to recall him to deal with that orally, particularly given the ... It was quite a tough couple of days for Dr Taylor and I'm sure for Debbie Slavin as well.

In my submission, the better course would be simply to offer Dr Taylor the opportunity to comment in a written witness statement on those questions that would be put to him if he were simply recalled and questioned orally because otherwise you have a situation where he is, in some ways, paying a penalty for the fact that a subsequent witness has, for the first time in oral evidence, mentioned something which is he's never mentioned in three, four, five witness statements

before. And in my submission, that would be really quite unfair and unjust on Dr Taylor and I think, in light of the way it has arisen, the better approach is the halfway house of Dr Taylor simply being offered the opportunity to comment in a written witness statement on that particular issue so the questions that would be put to him orally can be put in writing, and if there's anything that needs querying after that, he would be recalled thereafter.

THE CHAIRMAN: If we put it in writing in the first place?

MR UBEROI: Yes, sir.

MS ANYADIKE-DANES: Before you make a decision on that, I would like to address you on that. Maybe this is not the time or the place to do it, but I would like to address you on whether it is entirely satisfactory in a public inquiry where everybody has recognised the importance of the CVP, such an important piece of evidence was given by Mr Keane, that that should be then met with a statement in writing in answer to certain questions by Dr Taylor. I think that is, subject to anything else -- and I'm not formulating the submission now -- but I certainly think that is precisely the sort of thing which would warrant recalling Dr Taylor.

I recognise it that was a difficult couple of days. Frankly, I think it's been very difficult for all the

witnesses and I don't seek to isolate him in that way. But it is an extremely important point and we are here to try, so far as I can do it, to put before you the evidence for you to make your findings. I don't think it would be satisfactory for those findings in relation to what did or did not happen in relation to a discussion on CVP and the circumstances and how people conducted themselves in the light of that information to be left with Mr Keane's evidence which was tested by questioning and Dr Taylor's evidence, which is simply in the form of a written statement.

We have everybody's written statements about lots of things ands it has not led us to the position that we don't nonetheless want to hear those people in oral evidence.

THE CHAIRMAN: It has in some cases. The question is how important the issue is.

MS ANYADIKE-DANES: Yes. Well, I think --

THE CHAIRMAN: And the CVP line is clearly important, Mr Uberoi.

MR UBEROI: It is, sir. You have my submission and I'm happy to build on it in this way. It is an important issue, but the point which hasn't been addressed there is the principle of a witness being recalled simply because a subsequent witness has given

potentially unreliable evidence. This is not a situation where a new document has arisen, new evidence has come to light which couldn't be foreseen, and you have the principle of a witness suffering arguable unfairness or recall simply as a result of matters outside his control: a subsequent witness going, frankly, a long way off message, as it were, from his witness statements and offering potentially unreliable evidence. So you have the principle of the unfairness of recalling someone simply because of subsequent evidence which was, at face value, unreliable.

THE CHAIRMAN: Well, whether at face value it's unreliable is not yet a position that I have reached. But it may actually be entirely fair to your client to have him put his position on this. I'm inclined to the route of asking him in writing first for a response. If he took the view that, "Yes, Mr Keane's right, he did ask me a number of times for the CVP number and I gave him a number", then that, to a degree, corroborates Mr Keane's evidence.

MR UBEROI: Yes. If I may say, that would be an entirely sensible course.

THE CHAIRMAN: If on the other hand, he says that's absolutely not true -- and I think we discussed the possible scenarios of this a few days ago -- and if he

says, "I never called out a CVP number to Mr Keane, a specific number beyond saying things were okay" -- Mr Keane's evidence was really in two parts, as I recall it. One is he would check if everything was okay, but specifically, on a considerable number of occasions, he asked what the CVP number was. If Dr Taylor's position is that is simply not correct, then whatever else Dr Taylor may be criticised for, it leaves open a major issue about whether Mr Keane did what he hadn't said he had done in statements, did what he said he did for the first time in his oral evidence and that's negated. But we'll start with a written statement and get that out to you by close of business tomorrow.

MR UBEROI: I am grateful, sir.

THE CHAIRMAN: Mr McBrien.

MR McBRIEN: Sir, I think my learned friend indicated that the family would prefer written statements to oral evidence.

MR UBEROI: No.

MR McBRIEN: My instructions are that Debra's family would prefer Dr Taylor to be recalled.

THE CHAIRMAN: I don't think that's quite what Mr Uberoi said, to be fair to him.

MR McBRIEN: It is their position that they would like to have him recalled.

MR McALINDEN: Mr Chairman, there's one other issue which I would like to address at this stage in relation to any further statement that Dr Taylor would give. It's the evidence of Dr Coulthard in relation to re-zeroing and the use of a spirit level. Obviously, that is new evidence in this inquiry and I think Dr Taylor should be given the opportunity to address how precisely he would re-zero the machine.

THE CHAIRMAN: Or how he did re-zero in 1995.

MR McALINDEN: Yes.

MR FORTUNE: Sir, can I come back to the week as a whole? Because you started with Monday and, hopefully, Sally Ramsay will not be required. If you look at the remaining four days, the heaviest day is likely to be Wednesday and Professor Koffman. I see my learned friend raises her eyes in agreement. But in reality, we have got six other witnesses who should be taken quite shortly. In the circumstances, would it not be possible for the two junior doctors to be brought forward, one on to Tuesday, one on a Thursday with a bit of arm twisting?

THE CHAIRMAN: There have been efforts. I agree with you that if we can compact these because I agree that the fundamental points is that we don't want to be sitting here, say, on Tuesday for two hours and then come back

on Thursday for an hour-and-a-half or on Friday for two hours. We're trying to avoid that.

I think it's Dr Cartmill who is only available on Friday. Let me go and double-check that over lunch. Let me go and double-check this information.

Miss Conlon is making the best efforts she can over the last 24 hours and, in fact, since Friday, when this began to emerge, and I think she's having great difficulty in condensing the evidence into Tuesday, Wednesday, Thursday. I will report back to you on that after lunch.

MR FORTUNE: Because otherwise we could be, as you say, left with half afternoons.

THE CHAIRMAN: Yes. That's not productive.

MR UBEROI: May I make one final point in response to my learned friend's point about the re-zeroing of the CVP.

I'm happy for that to go in the witness statement and Dr Taylor will try and address it, I'm sure. I'm not sure it's quite right that it hadn't arisen before because Dr Coulthard's view, his invention and his suggestions on the re-zeroing were obviously present in his report. So I'm not sure it's quite right to say that it hadn't arisen before Dr Taylor gave evidence, but I'm sure he'll be happy to address it in the written witness statement.

THE CHAIRMAN: Okay, thank you very much. 1 o'clock?

(12.12 pm)

(The Short Adjournment)

(1.00 pm)

(Delay in proceedings)

(1.15 pm)

PROFESSOR PETER GROSS (called)

Questions from MS ANYADIKE-DANES

MS ANYADIKE-DANES: Good afternoon, Professor Gross.

Professor, do you have there your CV?

A. Do I have it?

Q. Yes, with you. I will just provide with you a copy now.

(Handed).

A. When it was requested again, I was already travelling and I had left Dresden, so I think what was sent to you by my secretary was sort of a short form.

Q. That is it there, is it?

A. Yes.

Q. That's your short form without all your publications, as I understand it.

A. It doesn't list my 180 publications.

THE CHAIRMAN: Good.

A. Too bad.

MS ANYADIKE-DANES: I just wonder if you could explain your qualifications. I see that in 1977 you were certified

by the American Board of Internal Medicine. Just so that we all understand the terminology, what does "internal medicine" mean?

A. It is a designation to distinguish surgical medicine from medical medicine that works with drugs rather than with a knife and cutting. That is being performed in adults as opposed to paediatrics, which is internal medicine in children. It is now oftentimes split up in several sub-divisions such as cardiology and pulmonary diseases and endocrinology, gastroenterology, and nephrology -- which is my specialty -- is one of them, but they all belong under the umbrella of internal medicine and in your education you usually train for a few years in general internal medicine first and then you go on after three or four years to specialise in -- I specialised in nephrology by doing this for another two or three years. You take another exam.

Q. I understand. As I look down through your CV you received training in the UK in Glasgow and London, also training in the US. And then you, ultimately in 1983, received your German certification as a specialist in internal medicine and as a specialist in nephrology. And those are the specialisms that you have today.

A. That's correct. I specialised in the States in internal medicine/nephrology as well.

Q. Thank you. You were also part of the university. You started, as I understand it, as an associate professor of the Department of Internal Medicine and Nephrology in Berlin and then Professor of Nephrology and chief of the Division of Nephrology in Dresden and you held that position from 1994 to 2009, as I understand it. And you continue to be involved in research.

A. After my requirement in 2009, the university asked me to stay on and, a year ago, I was made what they called -- termed a senior professor to the medical faculty in Dresden. Actually, I'm the first person to hold this designation, being a senior professor, and I continue doing research work there.

Q. Just at the last part, which is 306-048-002, the second part, we see that you are presently on the editorial board of a number of journals, one of them, the first listed there is Nephrology, Dialysis and Transplantation.

A. Mm-hm.

Q. So not only are you involved in your own research, but you are seeing, I presume, the product of the research of others in those areas?

A. Yes, you could say that. Yes.

Q. Could I just ask, in terms of that first journal, for which the nephrologists will be providing their papers,

is that continental Europe, is it worldwide?

A. Number 1, number 3 and number -- numbers 1 and 3 are European-wide journals that are also read to some degree in Japan, but very little in the United States.

Q. When you say "European", you are including the United Kingdom in that?

A. Included in there.

Q. Thank you.

A. Actually, the first, Nephrology, Dialysis and Transplantation, was inaugurated in 1971 from the United Kingdom. It originated here.

Q. I understand.

A. It's still being widely read here too. Number two is a German nephrology journal. Number five is a Polish nephrology journal. Number four is a small European nephrology journal that does not have a very large circulation.

Q. And number three?

A. And number three, oh yes. How could I overlook that? That's a very important one. That's the clinical arm of the American Society of Nephrology journal that is being read and accepted worldwide. That's quite an important journal.

Q. Thank you very much indeed. Professor Gross, we have heard from Dr Haynes, who you know from the experts'

meetings in Newcastle. We have also heard from Dr Coulthard and you will know that Dr Taylor, who was the anaesthetist in this case, provided a statement where he accepted many things that he had not previously accepted. As a result of that, there are a number of questions which we would otherwise have been asking you, but we don't need to ask you because he has accepted many things and, to some extent, those who covered his particular area of expertise, anaesthesia, in the case of Dr Haynes, he has given his evidence on that. So there are some other things that I would like to ask you so that we're not simply asking you to duplicate the evidence that others have given.

Also, you have provided expert reports, which we all have, and I take it that you stand by those expert reports. So we have the benefit of those views. Then finally, I should say that you will know that there were debates amongst you all, including Professor Kirkham, about certain views that she had expressed. Her reports, preliminary report and final report, are being peer reviewed, so on that basis we're not really asking any questions that touch upon that work, even though all of you provided reports that relate to that, but we're not addressing that.

So that has refined the questions that I would like

to put to you this afternoon. But before I do that, and as you were going through your CV and the journals with me, I wondered if you could say something about your experience in a particular element of the nephrology, which is hyponatraemia. Your experience and expertise in that would be helpful.

A. My involvement with this electrolyte disorder started when I was working in Denver, Colorado, with the University Medical Centre there from 1974 through 1980, first as an intern and then as a resident in internal medicine, and then the last three years as a nephrologist.

I might say that in those days, the nephrology division in Denver was called the Mecca of hyponatraemia. Denver had the most interest in the world in this particular electrolyte disturbance. It published the most, it arranged congresses on it, and there were many fellows -- I was not the only one -- studying there and studying hyponatraemia.

I used this time to do experimental work in hyponatraemia in rats where I induced hyponatraemia by infusing dextrose in water and antidiuretic hormone and I studied the effects on the body and on the kidney -- not on the brain, though. That led to several publications in accepted journals.

When I returned to Germany, I spent the next seven years in Heidelberg in Germany as a nephrologist and a research -- what we called a Heisenberg scholar.

During this time, I performed clinical research in hyponatraemia and this has been published in three publications where I studied the causes of hyponatraemia in 200 patients and documented that, and I did some -- during that same time I was able to do basic science research in the cells of the kidney that mediate or generate this hyponatraemia. It was something new in those days that you could sort of harvest these kinds of collecting duct cells from a kidney, grow them in culture and then study them.

When I was in Berlin later, I did not work particularly focused on hyponatraemia; I had other things to do. But since I came to Dresden, which is in 1994, for the last 15 years I think I have performed six more clinical published studies on hyponatraemia, how it comes about, which patients get it, what the consequences are and, most recently, in the last eight years, how we can treat it. There is a new group of substances, drugs, that have been developed for the first time that enable doctors to treat hyponatraemia specifically, something we never had to our disposal ever before. And I have been involved in testing the

various compounds and there were studies being done, licensing studies, those kind of things, where we were heavily involved in this.

I'm sort of known in my hospital for being the hyponatraemia professor. So I oftentimes get calls from the entire hospital -- hyponatraemia happens everywhere: it happens in OBGYN, it happens in psychiatry, it happens a lot in neurology, it happens in orthopaedics, in areas where you wouldn't expect it. And they ask me two or three times a week to come by and help them to diagnose it better, make the differential diagnosis, and recommend treatment. And I usually have a telephone with me and that number is fairly well-known and people call me from other places for these same reasons too. They know from congresses and so on and so forth that this is my area of interest and they sometimes request help.

Q. Thank you. I'm going to return to that bank of expertise later on towards the end, but there are some specific questions in relation to this case that I would like to ask you.

The first relates to the fluid calculations and administration. The clinicians -- that is certainly Professor Savage and Dr Taylor and yourself as expert and Dr Haynes and Dr Coulthard as experts -- were all

asked to prepare a table where you could see what your assumptions were in a standard format to permit comparisons to be made. You will probably remember that. We have looked at it many times in the course of these hearings. So I'm not going to put that to you, but just so that you recognise or recall the calculations that were being made.

One of the issues I would like to put to you is the question of the extent to which, when the anaesthetist is in the operating theatre itself, the observable features of the patient can assist in determining whether the patient is being fluid overloaded. I will give you an example, which may help. If we call up 011-002-007.

There is a reference there to "skin colour, skin mottling". There we are. It's in the final paragraph and if we stake the second sentence:

"The calculation --

THE CHAIRMAN: This is a statement by?

MS ANYADIKE-DANES: I'm so sorry. This is a statement by Dr Taylor, which was ultimately to be turned into his -- in fact, if we go to the first page of this -- so we'll come back to that place -- to assist with its origins. I believe it starts at 011-002-003. Yes.

This is a letter that Dr Taylor wrote to

Dr Murnaghan. You'll see that the date of that is 30 November, so it's extremely close to the date when Adam died. This is part of his, as you see there, statement of Dr Taylor. That's going to turn itself into a statement for the coroner, but Adam died on the 28th; this is being written on the 30th.

If we go back to 007, if we go back to that second sentence:

"The calculation was complicated and included many subjective factors not easily measured [this is the calculation in relation to his fluid management]."

Then in parentheses he says:

"(Skin colour, skin mottling, peripheral perfusion, pulse volume, pulse response to fluid bolus, et cetera), which become 'natural' for an anaesthetist."

The question I'm putting to you is the extent to which those sorts of observable features assist in determining or assessing whether a patient is becoming fluid overloaded.

A. If you permit that I answer your question in three steps.

Q. Yes, of course.

A. First, I would like to say, just for clarification hyponatraemia and fluid overload, I guess to most nephrologists, are two separate pairs of shoes.

Q. I understand.
A. So we're not now talking about hyponatraemia, we're talking about fluid overload, I understand.
Q. Let's first start with fluid overload, then we'll see if it assists with hyponatraemia.
A. Your question then was: what could the anaesthetist in the operating room have -- what kind of information could he have used, if I understand you correctly, to judge whether fluid overload was present or not; correct?
Q. Yes. The observable features. So these descriptions he gives.
A. And Dr Taylor, if I understand this sentence here correctly, is saying that into this judgment, it has to be fed into this judgment what the skin colour of the patient is, whether the skin looks mottled, irregularly coloured or not. What he can find out about the peripheral perfusion, by which I understand whether the hands are warm, the feet are warm and well perfused. What the pulse volume -- the strength of the palpable pulse -- might be, whether it is very flat or whether it's strongly pulsating and perhaps what the pulse response to fluids is.
MR UBEROI: May I rise? Sorry to interrupt, but perhaps we could slightly rephrase that, if we may. Rather than

the question being along the lines -- to be fair, I don't think it was asked like this, but it was potentially interpreted by the witness along the lines of what should Dr Taylor have seen. Rather, I believe my learned friend is getting more at: to what extent would these things have been present --

MS ANYADIKE-DANES: Yes, it is the latter that I was getting at.

MR UBEROI: Thank you.

A. So to mention those criteria or to use those criteria to my mind is most helpful. That part of the statement is, to my mind, justifiable and clinically very correct and should be used. The beginning of the sentence is "the calculation was complicated". Well, I would say that the fluid management would be facilitated by these criteria mentioned here and the calculation that he performed, as I recall it, was indeed complicated, as it says here, but by other things: it was complicated by his assumptions about what Adam's urinary volume would be, 200cc an hour; how much he thought that Adam was behind on fluids at 7 am when anaesthesia and the operation began; the concept was maybe 500 to 700cc, as I recall it.

But what is mentioned here doesn't really complicate the calculation of the fluid management; it would have

helped it.

THE CHAIRMAN: Sorry, professor, was the calculation complicated at all? Sorry. Should it have been complicated at all?

A. If your question is, "Is it complicated to know how I should replenish fluids in a patient who is making so much urine with this sodium concentration in it, who has not had IVs or unable to swallow fluid for five hours?", in Adam's case two hours, that to calculate is not really complicated, I don't think.

THE CHAIRMAN: And that's what I understood. I understood you to be saying that this calculation became complicated because Dr Taylor unfortunately made some wrong assumptions; is that right?

A. Mm-hm.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Thank you. You answered that in terms of these observable features that would help you in managing his fluid, I think you were going on to say, and understanding whether he was becoming fluid overloaded. To take the second part of it: do they help you in understanding whether he is becoming hyponatraemic?

A. Not normally. Hyponatraemia is a disorder that causes primarily mental changes, but it is not usually

diagnosable physically by examining whether the feet are warm or by examining whether the tongue is well moistened or by examining the blood pressure. Even severe acute hyponatraemia, like happened in Adam, is not diagnosable by there being oedema. That was a theme that came up in the testimonies later. Oedema like in the lungs or swelling in the face, those are signs of fluid overload.

Hyponatraemia, per se having too much water in the body, distributes over the entire water space of the body, which is about 60 per cent of body weight. In other words, it doesn't lead to much increased lymph formation, therefore it doesn't leads to oedema of the face, oedema of the legs, oedema in the lungs, you cannot see it. Normally, hyponatraemic patients show mental abnormalities that you have to be aware of to be able to suspect that something like hyponatraemia might be the cause and search for it, but there is no pathognomic sign of oedema that you can pick up by a physical examination of the body from the outside.

Q. When you say "the mental ones", what do you mean? Do you mean being disorientated? What do you mean?

A. When hyponatraemia progresses from mild to severe, usually, first of all, they become some apathetic, lethargic, slow, adynamic. When this progresses

further, they may complain of headache. When it progresses even further there may be confusion, they start to stagger or even fall, they can't walk on a straight line. They may develop seizures, grand mal seizures, and vomiting. This sort of being slow and apathetic may progress to somnolence. This is followed by coma and then it may be followed by even worse.

Q. If that happened whilst a patient was anaesthetised, you wouldn't have the benefit of those signs.

A. That's exactly the problem.

Q. That is hyponatraemia. Is it possible to distinguish between that and dilutional hyponatraemia, which is a means by which your sodium is reduced, if I can put it that way?

A. Is it possible to distinguish between dilutional hyponatraemia?

Q. And the hyponatraemia that happens because you have a reduction of sodium. In either case, I assume you have a reduction of sodium. One may be you have a reduction of sodium because you have an infusion of, effectively, free water and that reduces your sodium. And, I presume, on the other side you may not have fluid infused like that, but your sodium is reduced. Is there a difference in how the patients appear if, on the one hand, they have dilutional hyponatraemia and on the

other hand, they have hyponatraemia which is simply caused by another means by which their sodium is reduced?

A. Almost all hyponatraemia is dilutional. I just say this as a side remark. But to answer your question, in terms of these neurological -- mostly brain signs -- neurological signs we call them -- you basically could not distinguish between those two forms. What causes these neurological symptoms is the hyponatraemia. That's what the brain cares about or what the brain is affected by. It's not much affected by the cause for it. But of course, there are other subtle differences between these two categories of diagnosis. Most patients with dilutional hyponatraemia have a fairly well-balanced circulation, normal blood pressure, normal pulse rate. When you raise them up, the blood pressure doesn't change.

If it is what you call salt-losing hyponatraemia -- I'll just use your term -- if that is the case because a patient has a salt-losing nephropathy and is losing more salt from his kidney than he can eat or replenish, those are usually patients that have a reduced state of their circulation. They tend to have low blood pressure, they tend to have even lower blood pressure when they rise from a supine position, so they may

complain about being dizzy and they may complain more about falling to the ground. They may complain more about being weak, they may complain about having salt appetite and they may show salt-seeking behaviour. They tell you crazy things sometimes that they have to eat ... Not a teaspoon -- the next bigger spoon, is that a tablespoon?

Q. Yes.

A. There are patients like this that I had to see that tell you that they have to eat a tablespoon of salt just in the morning to be able to feel well throughout the morning or the day. So they may say these kinds of things and you can distinguish the two categories sometimes by these aspects.

Q. Thank you. I want to move on now to some specific points that Dr Taylor has said, just to see if you can help us so we can understand.

He had a witness statement of 008/2, at page 46, I believe. If you bear with me a moment. If you see right up at the top -- could we perhaps go to the previous page because that's where I think it starts?

If you start with 124. If you highlight that. That is an extract from the statement he made to the PSNI. It's really an exchange between the interviewer and Dr Taylor. He starts with saying:

"You said to him he chose one-fifth normal saline because it's isotonic. Yes. My understanding is that that is technically true as it sits in a bottle. It is. But the minute it's infused, its effect is hypotonic. It can become hypotonic, but not in every patient; it depends on their metabolic condition."

This is the response of Dr Taylor:

"How quickly they burn the glucose, basically. Well, under anaesthetic you burn less glucose because your muscles aren't active. You don't have to breathe, you don't have to take as much sugar out of your fluid. If you're awake and active ..."

And he gives two other cases that the inquiry is examining:

"... then you're going to burn more of the sugar under anaesthetic with the body at rest. Apart from the brain, which contains some activity, the rest of the body is at rest. The glucose metabolism is much reduced so its ability to remain isotonic is enhanced; it shouldn't become hypotonic to the same degree. That's another reason why the isotonic [and if we go over the page] dilutional hyponatraemia theory does not hold for Adam's case."

And then he is asked -- this is by the inquiry now:

"Explain in detail your assertion that the

administration of the 0.18 per cent solution to Adam was isotonic in nature rather than hypotonic."

And he says:

"Glucose is iso-osmolar with respect to plasma and not isotonic as I stated. It has an osmolarity of 284 compared with that of plasma, 290."

This is his second statement to the inquiry, which is where he accepts that what he had previously described as isotonic was not isotonic. I wonder if you can help by just explaining that.

Firstly, explain the difference and explain the argument that was being put forward that it was effectively the way the body's metabolism worked because his original argument is what -- actually presented what would otherwise appear to be a hypotonic solution as actually isotonic. I wonder if you could help us with understanding that.

A. I think it was fairly well expressed here, but I can say something about it in addition. When you count the osmotic particles being present per litre of 0.18 per cent saline with 4.3 per cent dextrose or glucose, you find that it has very similar osmolarity of that -- of the osmotic particles that would be present in one litre of blood serum. In that sense, it is isotonic or iso-osmolar. I think for today's discussion, isotonic

and iso-osmolar can be considered the same. In scientific terms, there are minuscule differences between the two terms, but I think that's not relevant here.

What has been said about the glucose component in this isotonic fluid is very correct. If this fluid is given to a normal organism that has normal glucose metabolism and handling, then there is insulin in the circulation from the pancreas, and this insulin makes sure that the glucose or the dextrose that you have infused into this patient is removed from the blood circulation or the extracellular space and taken up into cells in muscle and liver within a few minutes after the infusion.

I suppose that this would have been so in Adam during the time when he received these infusions from 7 am to 11 am on 27 November. It would have been similar because I saw at least one measurement of glucose that was obtained in him in the paediatric ICU later on. It was 7-something, close to normal. He was not a diabetic being under lack of insulin and being unable to take up the glucose into his cells and leaving it outside.

So once the glucose is removed and taken up in the cells, what remains outside the cells is the water. And

that then generates or makes this seemingly isotonic 0.18 per cent NaCl with 4.3 per cent dextrose or glucose, what seems to make it isotonic in chemical terms turns into hypotonic 0.18, one fifth normal saline biologically in an organism that has glucose around.

This is why -- and I think Dr Taylor in his most recent statement explained that he accepts that 0.18 per cent NaCl with this dextrose is a hypotonic fluid. He said that in writing. And I think he said he accepts that this led to dilutional hyponatraemia in Adam and so on.

Q. He does, but what I wanted to ask you is that if it has that effect, so it's metabolised in that way so long as you're not dealing with a diabetic whose system doesn't allow that to happen, assuming you're not dealing with someone like that, does it really matter whether the person is active or not? Will it metabolise in that way just as a function of the way the body works?

A. As far as I can tell, it doesn't matter much. It may have a tiny effect somewhere that I am not aware of, but in my eyes, this is not clinically relevant, how active you would be with this.

Q. Just so that we're clear: that argument that he put forward, which he now has appreciated was incorrect, that could, so far as you're saying, never be right. It

was always, so long as he wasn't a diabetic, his body was always going to metabolise it in that way and you were always going to be left with residual free water?

A. I think at the time this was made, could not be said to have been a significant, a biologically relevant argument.

Q. Thank you.

A. Would you perhaps give me a second to add something to what I said before?

Q. Yes.

A. There is a tiny point I forgot to mention. I wanted to mention it. I also wish to point out that there is literature, specifically from the United Kingdom -- and this is in the documents I sent to you -- that appeared between 1985 and 2006. There are five or six articles that speak about this particular nature of the 0.18 per cent saline with 4.3 per cent dextrose, which is apparently a solution that was used in this country preferentially by anaesthetists. There are several publications that speak about the risks to patients in terms of causing water retention in the post-operative state and, with it, hyponatraemia, and warn against it. There was even an alert, I think, that came out in -- I forget, maybe 2004 or so -- warning anaesthetists against using this particular 0.18 per cent saline with

4.3 per cent dextrose and replacing it with half normal saline was the proposal.

I only wanted to indicate that what I explained, tried to explain before, for why, biologically, it has to be seen as hypotonic is also in the literature and I think I supplied five articles to support that to you.

Q. Thank you. I wonder if we could move on to something that you had briefly raised before, which is the difference between acute and chronic hyponatraemia. I wonder if you could explain that.

A. That's a very important point, if you allow me to say that because, in terms of physical consequences from hyponatraemia, there's a big difference between the two.

Acute hyponatraemia, although it is defined as having lasted between 1 and 36 to 48 hours -- and chronic is when it lasts longer than 48 hours -- although it seems short, acute hyponatraemia is the one causing all the bad symptoms and the bad problems. Chronic hyponatraemia usually leads to mild consequences in terms of symptoms and so on.

It's like fever. If you have a sudden fever, a acute fever, you have shaking, chills and rigors and you feel very sick. If someone has chronic fever for months, which sometimes happens, they oftentimes don't feel it and they have fewer consequences from this. But

this is only a comparison.

Acute hyponatraemia -- let's say of 125 millimoles per litre -- can make an adult patient very sick. It can be -- this patient may become forgetful, confused, have severe headaches, be vomiting and, in extreme cases, might even be developing an epileptic seizure. Chronic hyponatraemia, you sometimes meet patients that have a sodium concentration as low as 105 millimoles per litre, and they seem to have very few symptoms. You can talk to them almost normally. They memorise poorly, they're a little more tired. So there's a big difference.

This has perplexed physicians for time and there is research that has been conducted to try and explain this difference. Consensus in the literature on explaining why these two states are so different is that acute hyponatraemia causes all cells of the body to swell. But this swelling is a transient phenomenon. The body, the cells, have no mechanisms to defend themselves apparently from this volume expansion, from this swelling of the cell, and bring the cell volume almost back down to normal. That is believed to be -- that's a process that starts one to two hours after the onset of acute hyponatraemia and is all complete by between 24 and 48 hours.

That is usually quoted in the literature or also shown in an article I provided to the IHRD by Dr Joseph Verbalis where he shows that after -- in his experiment, I think it was 12 days. Of course, it was in rats. All this research is on experimental animals and we have to assume that humans behave similarly to them. He shows in them how this volume compensation, bringing the volume of the swollen cells back down to normal, how this happens, and he shows how cells have mechanisms to pump the osmotic particles inside the cell, the sugars and sorbitol and what it all is, out of the cell to unload cells of osmotic particles and thereby water leaves cells again and cells go back to their normal cell volume. And with this, the symptoms that those patients suffer from are sort of mitigated or disappear.

This is an aspect that I believe is of particular relevance in today's subject.

Q. Why do you say that?

A. Well, because we know from the CT that was taking the computerised tomogram that was obtained in Adam at 14.15 of the 27th, they saw diffuse -- I think Dr Anslow said the brain is very swollen. And we know that the cerebellum descended into the foramen magnum as a sign of the swelling. We know that the autopsy describes

severe brain swelling. So here we see swelling and we know that Adam was made hyponatraemic over a relatively short period of time. So it seems like these two factors were closely related.

Q. Does that mean you think that the rate of fall of the serum sodium is a significant factor?

A. Yes, I do. As I tried to explain before, when the serum sodium is lowered over -- as happened in Adam from approximately 134, 132 to 123, if it was lowered to that degree over three or four days, I expect that the CT would have found no brain swelling and there would have been no major symptoms in him from the hyponatraemia. The shorter the period, the time that is available to induce this hyponatraemia, the more severe the swelling can be expected to be.

I want to say something more about this, but before I go to that, I just want to mention that we calculated Adam's rate of fall of the serum sodium to have been, if I recall it correctly --

Q. I think it's reference 201-015-225.

A. -- 3.6 or 6.6 millimoles per litre per hour between 7.00 and 9.32 on 27 November. Whereas in all previous episodes of hyponatraemia that he had undergone, I believe there were seven, but only six in which the rate of fall could be calculated, it was between

something like 0.005 and 0.75, so it was a lot faster Obviously, there may be a limit to this. I am not attempting to say that you could take a patient from 132 to 123 by giving hypotonic fluid in a minute and make it even worse than you do when you give it over two-and-a-half hours. I guess I've described this kind of poorly. I'll try again.

I'm trying to say that the water has to make it from the blood vessel into the tissues and, in this particular case, into the brain. And there are barriers between the blood column and the tissue. So the water cannot flow from one compartment to the other as if there was an open pipe between them.

This is sort of a weak point in acute hyponatraemia that we do not know precisely how long it would take if you induced hyponatraemia of 123 millimoles per litre in the blood for the appropriate amount, 7 to 8 per cent of water, to make it into the brain and cause brain swelling there.

I pointed this out in a comment that I sent to the IHRD about two-and-a-half weeks ago. There is some point where or some period of time that is necessary for the water that has to elapse for the water to be able to leave the blood vessel and go to the brain. And there are no precise measurements available to this. The

article by Arieff in the British Medical Journal --

Q. Sorry, if we just stick with that concept. It's not an instantaneous transfer of water from the blood into the brain, there is a period of time when it crosses that blood-brain barrier, if I can put it that way. Just so I understand the significance of that, does that give you a window to reverse the effects?

A. Does it give me a?

Q. Does it give you an opportunity to reverse the effects?

A. If you ... I'll answer that, then I want to say one final sentence to the previous discussion, if you'll allow me to do that.

Q. Yes, of course.

A. The brain swelling itself, if it's due to osmotic reasons, is, of course, always reversible, in theory. If you apply some osmotic agent to the bloodstream like mannitol or hypotonics, 3 or 5 per cent saline, the descent of the cerebellum and brainstem into the foramen magnum is not reversible. And, of course, if you pick up a hyponatraemia that is rapidly progressing early enough -- at one hour or so -- and you do something against it, it supposedly would be reversible.

Q. So before the coning process has actually started, if you appreciated what was happening, you could reverse it?

A. I would say that, yes.
Q. Okay, thank you.
A. If you would allow me to finish the earlier question. I was sort of worried that there are no direct measurements on how fast brain oedema can start to be there. I therefore obtained additional literature. I sent you a comment that quotes ten articles discussing patients with acute hyponatraemia, many of them died, not all of them. And it quotes, I think, seven articles from experimental literature. And they do show that within two to four hours from the induction of acute hyponatraemia of the degree that was present here -- 120 millimoles per litre plus or minus 2 millimoles per litre -- that, indeed, when those animals then were -- all those animals developed epileptic seizures, coma or died within two to four hours. When their brains were sectioned, gross cerebral oedema was found. And when they determined in these articles -- I'm now talking about the experimental animals -- brain water content, they could find that was increased 8 to 15 per cent. These articles say -- I have them all with me. If someone cares to see, we can go through that.

So I came to the conclusion that even though water doesn't make it from the bloodstream into the brain instantaneously, it must take some time, probably more

than half an hour. A time frame of 2 to 4 hours in the experimental literature is clearly established as being sufficient of a serum sodium of 120, plus or minus 2, to produce significant brain oedema with severe symptoms and a measurable major increase in the brain water content in the range of 8 to 14 per cent. So I sort of -- and the reports on patients in these ten publications sort of parallel this, although most reports have no measurement of the brain water content and these kind of things that you can do only in experimental animals. With this evidence, I satisfied myself that the sequence of events in terms of the hyponatraemia that had happened in Adam would go along with this literature and would be able to explain his brain oedema probably primarily on an osmotic water shift basis alone, primarily.

Q. And therefore, if it had been possible to identify that process, then it was susceptible to being reversed?

A. And as I said before, that's a reversible process, expect for the coning.

Q. Thank you. I'd like to take you to just a small part of one of your reports, just so that you can give us your observation or expand on your observation. It's 201-004-124. It's in the middle of, roughly, that long first paragraph. You're talking about -- I think it

starts "one watches". It's just after the stitching of the anastomosis:

"One watches if the transplant turns red (is being perfused) and perhaps even begins to make urine appearing from the ureter after a few minutes. Such is frequently not the case. In a case like Adam's, it would not be very surprising to see that the kidney turns red at first and appears to be (more or less well) perfused, only to show signs of reduced perfusion later on."

And then you say, and this is what I wanted some assistance with:

"Usually, perfusion is usually judged macroscopically by eyesight and not by precise measurements, like those theoretically possible with duplex ultrasound. In this way, different observers may get a slightly different impression of the perfusion of the kidney, depending on how they visualise and interpret its colour."

Apart from being an observation deduced by -- if it's subjective, then almost necessarily that means that people can differ about it, but is that your actual experience, that that happens?

A. Yes.

Q. That people looking at the precisely the same event and

describe it slightly differently in terms of the quality or nature of its colour?

A. Describing or remembering it differently.

Q. Yes.

A. Maybe not so much in Adam because he was smaller, but usually the operative field is not very large, it's not, frankly, open so that everybody could see from a distance. Only the people very close to it have a good look. There is bleeding around the kidneys, covered by blood. There are other things around. I think if you asked three surgeons about what the kidney looked like in the first five minutes and in the second ten, in the third ten, it could well be that you get two different opinions even though they all saw the same thing.

Q. I understand.

A. Of course, everybody in the operating room is happy to see that the kidney pinks up. They connect it and it turns reddish and they say the anastomosis seems to be working, the kidney is well perfused. But the experiences is that this is not always the case. Parts of the kidney pink up well, other parts remain greyish. And usually, the experience is that the operation still goes on and we do know, like I think Professor or Dr O'Connor -- I seem to remember she has, in my

opinion, pointed out quite correctly ... We do know that oftentimes kidneys look sort of a bit unusual when they're put in and the clamps are removed and they do well a week later or two weeks later and start to function fine. We have to give these patients a week of dialysis to get them through and then the kidney kicks in, makes urine, functions fine.

So it's only that if after an operation in the next few days there is more evidence that this kidney is not working, it's not making any urine whatsoever, the patient maybe develops swelling, we do ultrasound or perfusional studies using ultrasound, we see perfusion is decreasing every day, that we then start thinking something may be wrong and open the patient up and take a second look or do something about it.

Q. In any event, if I understand you, these descriptions of the kidney are not necessarily an accurate way to judge what might be the ultimate success of the graft? Although in Adam's case, of course, the kidney was examined microscopically so there are expert views as to the condition of the kidney, which they then try and back time, if I can put it that way, to when they think the kidney had started to fail. But anyway, these descriptions, you're saying might be a little misleading.

A. Absolutely. I would agree with what you said. These are not like a report from pathology where he says, "I saw this and this means that", "I saw that and that means that", or so. It's just an impression, not more than that.

MR MILLAR: Sir, just while we're on this point, there is a useful section on this point at 201-002-049 where Professor Gross is asked to comment on the very different descriptions of the kidney. At 050, he has a comment that you might find helpful, sir, just on this point.

MS ANYADIKE-DANES: Well, I wasn't necessarily going -- I think he's essentially encapsulated the point. I think maybe the chairman has the point. But it's good to have the reference though that we can continue --

THE CHAIRMAN: Is that 049 going into 050 where he says in bold:

"All the people are saying more or less the same thing and, to me, there are no important discrepancies"?

MS ANYADIKE-DANES: Sorry, I wasn't meaning to decry the value of it. I just thought the witness had given a particularly graphic oral testimony on that point.

The next point that I want to take you to is, I think, something that nobody else has commented on and, in your report, it's at 201-004-142. I think what

you're dealing with here are the possible implications of the fact that the transplant malfunctioned, the kidney ultimately did not work. I wonder if you could explain this. For the benefit of those who may not see it, let's highlight it. You say:

"The malfunctioning transplant in itself did not contribute to Adam's hyponatraemia."

And I don't think that anybody has thought that that did, but it's what you go on to say. You say:

"It was the renal failure of his native kidney that has prevented the excretion of major amounts of free water. But had the transplant functioned [and this is a point I would like you to expand upon] well, it is likely that it would begun to excrete free water. That then could have contributed to reducing the degree of hyponatraemia present in Adam."

I wonder if you could expand on that and explain that.

A. Today, I would still say exactly the same. Transplanted kidneys that are in very good conditions and function 100 per cent well sometimes, as the surgeons say -- I'm sorry for saying that way -- they start peeing on the table. It means they start producing a lot of urine visibly while the patient is still on the table and, therefore, a well-functioning transplant might have

contributed by excreting something like perhaps 200 to 300 ml of urine in the first 45 minutes.

Of course, that relates only to Adam's hyponatraemia and the degree of it and that then being reduced by Adam being put by that new kidney into a negative water balance. The clinical question is: could this have helped him at this point this time?

Q. Yes.

A. And as I seem to remember it, the clamps were opened at 10.30; is that correct?

Q. It's not entirely clear. It says approximately 10.30.

A. Approximately. So supposedly, it would have taken 15 to 20 minutes for the water production to have become more copious. So by then, it supposedly would have been 10.50 to 11.00. So in trying to answer the question, "Could this have possibly prevented the coning?", brings us to the other question: when did the coning take place? If you assume that the coning took place towards the very end of the operation, which was near 12 o'clock, I think.

Q. Yes.

A. Then this could be perhaps conceived that it could have helped in that respect. My personal opinion is, you're aware from my comment, that probably the coning happened earlier and then this water, potential water excretion

would not have been relevant towards reversing it. It's believed to be an irreversible condition. Once you have coned, then this is it.

Q. The damage is done to the brainstem?

A. Yes.

Q. So for this to be of any benefit to Adam, it depends on knowing more precisely when the clamps are released and having a better estimate as to when the coning process had reached the stage when his brainstem was irretrievably damaged?

A. Yes, I would say that.

Q. And if you knew both those things, you'd be able to tell whether the functioning of the transplant would have assisted him?

A. Yes, I agree with you.

Q. Thank you. Then I wonder if you might help us with another matter. Adam was administered dopamine in two boluses at 10 am.

A. Mm-hm.

Q. Can you help as to what the purpose of that or, rather, maybe a better way to put it is what the benefits of doing that might be?

A. I'm a tiny bit confused on that issue too, but I would like to say this: I think I have learned from Dr Taylor that the original idea of adding a low dose continuous

dopamine infusion during the operation to his regimen was to apply something that is called renal dose dopamine in the literature, believed to dilate the kidney to allow the kidney, in this case the expected transplant, to be perfused better. Now, for this, the published dose to be applied is 0.5 to 2.0 micrograms per kilogram per minute.

Dr Taylor gave twice as much. He gave between 2 and 4 micrograms per kilogram per minute. So I'm a bit confused there. But I think he indicates somewhere that the purpose was to help kidney perfusion.

Q. Yes.

A. And probably the best interpretation of this is to have the dopamine contribute to the vasodilation, expected vasodilation, in the kidney. If you give more dopamine than the amount indicated, it turns from a potential vasodilator into a vasoconstrictor. It's an agent that has different effects depending on how much you give. It seems to me that the two boluses he gave around 10 am, which was 2 micrograms or something like this repeated within five minutes, was to increase blood pressure. And apparently, at least this is what I learned from his statement -- do we have a comment why he gave the boluses? Someone said, I think it was him, it was for blood pressure reasons.

Q. Let's start with the first mention of it. If we go to 058-035-133. If you look, this is the note from Professor Savage. If you look at:

"In theatre to have."

Under (iv) you can see the dopamine.

A. Mm-hm.

Q. What did you say was the optimum?

A. 0.5 to 2 --

Q. So --

A. -- micrograms per kilogram per minute, so the same unit as here. The 2 I would agree with. The 2 to 3, in my understanding, is a little bit high. I would like to add, however, in order to prevent that I'm creating a controversy that is not necessary, there is dispute in the literature on whether the dopamine is really doing this vasodilation in the kidney and at which dose it is doing it best. The 0.5 to 2 that I've quoted, I took from guidelines.

Q. Just the part that I think you were asking to see, if we pull up 011-014-097.

MR FORTUNE: Before we pass from that, is Professor Gross criticising Professor Savage for this prescription? Because if so, this is a new matter.

MS ANYADIKE-DANES: The question is, Professor Gross, is that prescription one that you would be critical of?

Not necessarily the dopamine, but maybe the level at which it is being prescribed or the rate.

A. The opinion in the literature is that this dopamine is harmless. You can give it and maybe you help the kidney a bit, or you cannot give it. If you do not give it, not give dopamine at all, you don't do any particular harm. So the dopamine in the literature, in this situation, is considered something that is not essential.

Q. Yes. I think maybe the reason why Mr Fortune rose to his feet is you had described a rate or an amount that you thought was optimal and then you said that if you give more than that it has certain effects. I think that's the issue. What is the amount that you would have to prescribe to achieve those effects?

MR FORTUNE: Actually, the question is: is this a criticism --

MS ANYADIKE-DANES: It depends what the answer to that is, if I can put it that way.

THE CHAIRMAN: Sorry, let's go back a bit. Professor Gross originally said that the amount given was twice the amount in the literature. He said that range was 0.5 to 2 and then he was taken to this document which says 2 to 3. And then the professor said, in fact, there's a dispute in the literature as to what the amount is.

So as I understand it now, not only is there a dispute in the literature, but the professor also says that giving this is regarded as being harmless. If that's right, then it's hard to construe this as a criticism of Professor Savage.

MR FORTUNE: That's all I wanted to establish, sir.

THE CHAIRMAN: Do I understand you correctly? This is an area of debate, but you're not being critical of Professor Savage.

A. I don't mean to.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Can we just clear this up? The negative effects that you described might occur in the administration of dopamine: are you saying or are you not saying that this amount could result in that?

A. Actually, what you term "a negative effect" is what I called vasoconstriction. Let me point out to you that anaesthetists sometimes use this not necessarily as a negative effect, but as something to elevate blood pressure. They give more dopamine to increase blood pressure temporarily, so it's not necessarily negative.

Q. I understand. Are there therefore two reasons why you might administer it?

A. Yes. There may be two different reasons. And the initial reason here, I believe, should have been, or

appears to have been, to try and induce more vasodilation specifically in the kidney. It is believed to have this vasodilating effect at this dose, I said 0.5 to 2 micrograms per kilogram preferentially in the kidney. That was the initial intention. And I think nephrologists in general -- myself included -- would not be critical of this.

Q. Thank you.

A. The literature says you can try that, maybe it works. Nobody is 100 per cent sure about it. There used to be lots of discussion in the literature whether we should still give dopamine or leave it. This was 1995 and I'm talking about the last 10 years.

Q. Yes. Can I pull up where you wanted to go to, which was where it's actually mentioned in Dr Taylor's statement? It is almost in the middle of that page. You'll see it says that after an initial period of apparently good perfusion, it wasn't appearing to perfuse well:

"A low-dose dopamine infusion had been commenced near the start of the case to improve the blood flow of the donor organ."

Is that the statement to which you were referring?

A. Yes.

Q. So the purpose of that was to dilate the blood vessels --

A. Exactly.
Q. -- to improve the blood flow to the donor kidney; is that right?
A. Yes.
Q. If that's what you wanted to do with it, then when would you be infusing that in relation to the release of the clamps?
A. Yes, that's a good point. It didn't strike me before, but you're quite correct. If that was the intention, you would probably start to do this half an hour before release of the clamps. But this infusion was given from the beginning of the operation or from the beginning of infusions.
Q. And what would be the effect of doing that as opposed to doing it briefly, if I can put it that way, before the release of the clamps just to encourage dilation?
A. The dose of the dopamine, that is being given here is so low that there would probably be no discernable effect on peripheral perfusion, blood pressure, systolic, mean blood pressure. I guess one would have to say no significant discernable effect. Also no harm.
MR UBEROI: Can I just rise by reference to that, sir? I hope I'm not going to confuse matters further. 011-014-101, please, which is also the deposition. A quarter of the way down:

"There are two small increases in the systolic blood pressure around 10 am corresponding to two small boluses of dopamine."

So that's a second reference to, as it were, in the deposition of Dr Taylor.

MS ANYADIKE-DANES: Yes, I was just coming to that.

MR UBEROI: "The rational for this was to increase the perfusion pressure without fluid".

MS ANYADIKE-DANES: Thank you very much. That's my next point.

The first point is that you have Dr Taylor having a low dose of this early in the proceedings, if I can put it that way, for the reasons that you've just been looking at. Whether this was additive or whether it's just a better description of what he said he was doing, but it then seems to be that he then gave two small boluses of dopamine, which would appear to be in addition to that low dose that he had started quite close to the beginning. Two small boluses of dopamine and he gives the amount there. So that's really quite small by comparison to the original prescription or description of it that Professor Savage had written in the notes.

MR UBEROI: If I might add, it may appear from that, from us picking out these two extracts, as my learned friend and

I have just done, but one of the difficulties with this is that this is an issue that has not really arisen before. It seems to have emerged in the last couple of days and wasn't an issue which was focused upon or particularly put to Dr Taylor before, if I may say. And it seems to have rather been emerging to the foreground over the last couple of days. So in characterising what Dr Taylor did or didn't do, I'd draw that very much to your attention. I'm not really sure that one can cherry-pick from this deposition what the true position on the dopamine was. That's certainly no fault of Dr Taylor. As I say, this really seems to have been emerging only in the last couple of days.

MS ANYADIKE-DANES: That might be another matter for the witness statement request.

THE CHAIRMAN: Sorry, no. We're not just going to build up and longer and longer further witness statement for Dr Taylor to respond to. He's already provided one written additional witness statement after his oral evidence finished. We've discussed this morning a further one and we're not going to run the inquiry on the basis that we just adds bits and pieces and then, at the very end, put all the outstanding fragments to him.

MS ANYADIKE-DANES: That is why I said it might be, sir. What I'm going to ask the professor is what the

significance might be. It may be, sir, that there's absolutely no significance at all, whether you started with a low dose and added two boluses at roughly 10 o'clock of the amount that is said. There may be absolutely no significance in the way that he said there would actually be no discernable effect in the first lot. If there's no discernable effect, then there's nothing that needs to be asked of Dr Taylor and it really doesn't matter why he had two boluses at 10 o'clock if it has no discernable effect and that was the question I was going to put to Professor Gross.

MR UBEROI: Finally, sir, from recollection, it also strikes me as something that -- again this is off the top of my head -- that Dr Haynes wasn't particularly taken through in great detail as to the effect of dopamine. So you're again into this issue where you're potentially crossing disciplines and someone with the knowledge of a nephrologist is being asked to comment on the actions of an anaesthetist, and again, I think that raises the risk of unfairness and there's again the rather unattractive concomitant of the way that this seems to have emerged over the last couple of days for the first time.

MS ANYADIKE-DANES: That I understand, Mr Uberoi. I'm not actually asking him to comment on the conduct of

Dr Taylor; I'm asking him about the effects of certain things. He is not in a position to say why Dr Taylor may or may not have done anything. What we have is two descriptions of amounts of dopamine, apparently, being infused at an approximate point in time. All I'm seeking from Professor Gross is what he thinks the effects of that would be. That is all.

THE CHAIRMAN: I'm also concerned about the risk of overanalysis by a whole range of people coming from different disciplines of what was or was not done on specific issues. This inquiry is obviously complex in some areas and I'm anxious to ensure that we don't make it unnecessarily and excessively complex by overanalysis.

MS ANYADIKE-DANES: I understand that, sir, but he has already given a view as to one element of the dopamine administration. It would seem odd not to ask him to say whether, if you add on this amount of dopamine in two small boluses at a particular point in time, it makes any difference to his original view of no discernable effect.

THE CHAIRMAN: Let me hear what he says, Mr Uberoi, and we'll see if it is going anywhere.

This is, professor, something that was given at the time that you would have -- you have said, a few minutes

ago, you would have expected the dopamine to be given at roughly 30 minutes or so before the clamps came off.

A. That is sort of the unclear point here, that ... If we say the clamps were opened at 10.30, then ... The problem is --

MR HUNTER: [Inaudible: no microphone] confusing the professor. Looking at Dr Taylor's statement here where it says that the small increases in BP around 10 am corresponding to two small boluses of dopamine, presumably being given at 10 am. And that is confusing to the professor. If we take the evidence that the clamps were apparently off at 10.30, would that be correct? Sorry, that the boluses of dopamine were given at 10 am according to the statement, but we seem to think that the clamps were released at 10.30.

A. But it says here, where the yellow marking ends, talking about the donor kidney, "which at that stage was not looking good and not producing urine" --

MR HUNTER: Correct.

A. -- implying that the clamps are already removed, otherwise you can't say that. So this implies that the clamps were removed earlier or the time is not correct.

MS ANYADIKE-DANES: Yes. I understand. That wasn't actually the point I wanted to ask. You had previously given your view as to what the effects might be of

giving dopamine, that low dose of dopamine, somewhere towards the start. I think your personal view is there may be no real point or benefit in doing it, but in any event, you didn't think it was likely to cause any harm at all and I think your concluding view was that it would have no discernible effect. So that was that.

Then, helpfully, my learned friend pointed out the fact that dopamine is mentioned again further on and this time it seems to be mentioned in a slightly different way. So you have this low dose towards the start for a particular purpose, which you don't think is likely to have any discernible effect, and then, on top of that, it would appear, there are these two small boluses at roughly 10 am of this particular amount -- very, very small amount -- certainly much smaller than Professor Savage had put in the medical notes and records. And my question to you is: having got the underlying one that is of no discernable effect, what might be the effect of adding these two small boluses to that? That's my question.

A. As I interpret this, this was written by who?

Q. Dr Taylor.

A. Doctor?

Q. Taylor.

A. So if this is written by Dr Taylor, my interpretation

is that they were all looking at the transplant and they were seeing perhaps that it didn't quite pink up as much as they had expected. And they were saying: we tried the low-dose dopamine to improve or to generate the best possible conditions for transplant improvement and contribute to this by low-dose dopamine. Apparently, that strategy hasn't helped very much or didn't work. The transplant still looks kind of irregular and it's not nice and bright red all over. Let's see if we can increase the systemic blood pressure, the driving force of pushing blood through the kidney, and one way of doing that is to give boluses of dopamine.

Q. Thank you.

A. I think that may have been the thought behind this. The time may not be correct.

Q. Yes, exactly.

Then, finally, I would like to take you almost back to where we were at the beginning when you were explaining about your experience with hyponatraemia. I would ask you about what's been called the Arieff article. Of course, we know that he's not --

A. The what?

Q. The Arieff article. Of course, we know he's not the only person who worked in that study, but it has become known in the course of this inquiry as "the Arieff

article". I think you know the one I'm talking about, published in 1992. So far as you are aware, how widely known was that article?

A. This is a difficult question.

THE CHAIRMAN: I presume if you were at the Mecca of hyponatraemia in Denver, you knew about it, did you?

A. At that time of course I was no longer in Denver, I was back in Germany. I am afraid I would have to say, even though this turned out to be a landmark article, very important article, it was not widely known. I think it was known to many nephrologists because they're reading this kind of -- electrolytes is considered to be the field of nephrology and endocrinology. It is my experience that the knowledge in the field of electrolyte disturbances, hyponatraemia amongst them, with anaesthetists is better than with many internal medicine people. Whether anaesthesiologists would have read this article, I kind of doubt. I think it probably was -- I'm sure it was not well-known amongst internists [sic] in Germany.

I think it was not very well-known amongst nephrologists in Germany. I think it was not very well-known amongst anaesthetists, even though it was a very important and a very deserved article. It was nothing that people would talk about like they talk

about the first cardiac transplant in the world or something like this. It was known only, I think, or primarily I think in specialist circles and it sort of made its way to become known later on.

What I do not know and have difficulty telling is how this might be with paediatricians. We're talking about -- Dr Taylor is a paediatric anaesthesiologist; is that correct?

Q. Yes.

A. That, I wouldn't be sure. I mentioned this because --

Q. He has already stated that he knew of the article.

A. He knew it?

Q. Yes.

A. I mention it only because it's an article that reports 16 children. So one reason perhaps for why it is not so well-known amongst all the groups I mentioned before could have to do with the fact that this is considered paediatrics.

Q. Yes.

A. And because hyponatraemia in adults has somewhat different circumstances, brain size, and this kind of thing, reserve -- it may have been a little better known amongst paediatricians and paediatric anaesthesiologist, yes.

Q. I wonder if I can ask you this: leaving aside the Arieff

article, which warned, from a layperson's point of view, about the dangers of dilutional hyponatraemia in otherwise healthy children -- so children who had minor conditions or were going in for minor surgery and so forth, so not Adam's situation at all. Leave aside that. The condition of dilutional hyponatraemia, how well-known was that so far as you're aware?

A. I guess what you're asking me is how well understood was dilutional hyponatraemia.

Q. Yes.

A. There, I'm afraid I have to disappoint you too. Even nowadays, 15 years down the line, most people in internal medicine -- and I'm afraid in paediatrics as well -- do not understand dilutional hyponatraemia very well. Put it another way: if you asked 100 doctors what is the cause of dilutional hyponatraemia, I'm afraid 85 will say, "Oh, it's a salt deficiency disorder. You need to give them salt and everything turns out to be better", which is the sort of naive -- I'm sorry -- thinking induced by the terminology "hyponatraemia" -- "too little natrum", sodium -- like hypokalaemia is indeed too little potassium in your body -- and only 10 or 15 will know it's too much water.

So many doctors, unfortunately, have concepts in their mind of what hyponatraemia is, what causes it and

how it can be improved, that are not correct. That may sound very surprising to you, but the cause of it is that for 60 years we didn't have a specific treatment and salt tablets or salt treatment was tried frequently, sort of as an indirect and not very helpful poor man's therapy of hyponatraemia, which oftentimes did not help.

So to answer your question with a short sentence, the majority of doctors, in my experience, have erroneous concepts of what dilutional hyponatraemia is and are not well educated about it. Something that's changing now because we have specific therapy, but this is not an issue for our discussion here today.

Q. I understand. So whereas paediatric anaesthetists might be alive to the condition, generally, amongst doctors, they are not, as I understand you to say, sufficiently well understanding of this particular condition?

A. I don't know many paediatric anaesthetists, but I had to give postgraduate training presentations to anaesthetists on several occasions. They have to be re-certified every so many years and they have to take courses before this. And my experience in those lectures was that anaesthetists understand hyponatraemia probably better than the average doctor I was talking about before. And in my experience, at least, anaesthetists are sort of sensitised to hyponatraemia

and they know hyponatraemia in the context of an operation is something that can turn dangerous quickly. So they are sort of in a somewhat different position there.

Q. Yes. But if one is speaking generally about doctors -- and I think, when you were talking about hyponatraemia earlier, you said you would get calls from people from all different disciplines --

A. Yes.

Q. -- where hyponatraemia had developed in their patient. So if that's possible for that to develop in a number of different circumstances, what I'm understanding you to say is that those doctors who may have a patient like that still -- even today, I think you were saying -- many of them are not sufficiently sensitised to the condition, how it develops and its effects. Would that be a fair way of summarising what you were saying?

A. Yes, I agree with you.

Q. If that's the case then, what do you think are the big issues that need to be conveyed even now amongst the more general medical population, if I can put it that way, about hyponatraemia, or dilutional hyponatraemia specifically?

A. This takes us to a new field. It doesn't directly have to do with Adam.

Q. No.
A. It is believed that the majority of hyponatraemic patients that we see have mild hyponatraemia, 128, 130. About 8 to 15 per cent of a hospital population that's being measured at random will turn out to be hyponatraemic in that range. The symptoms are usually mild. They're forgetful, they're a little bit depressed, they can't concentrate. These kind of things. And it is believed that their quality of life is lower than need be, particularly relevant in elderly people. It is believed that they fall more often and fracture bones. So the hyponatraemia community thinks that, in the next ten years, doctors need to be educated that it's now possible to treat hyponatraemia -- I mentioned this in going by an hour ago, I think -- we sort of have to try and improve quality of life and prevent fractures and those kinds of things from treating mild hyponatraemia. But that is something else.
Q. Yes.
A. It doesn't really have much to do with our case here today.
Q. But in terms of educating about the more severe types that lead to the sorts of consequences for Adam, what do you think is the work still to be done in relation to

that?
A. Sorry, I missed that.
Q. No, no.
A. I could have said that also before. There has been -- now that specific therapy is available, there has been a rather strong educational effort that took place in the literature over the last three years and I think almost all medical and nephrological, endocrinology journals have run an overview and educational articles very recently, in the last two or three years, in how best to establish the differential diagnosis, what the consequences of treatment are, which kinds of hyponatraemia should be treated and in which way. So the field is sort of receiving renewed attention recently and now that doctors have a tool available to them to improve these patients' lots, everybody is more interested than they ever were before and hyponatraemia is now something that at each congress you go to, they're having a major session, there are a bunch of speakers and there are symposia and there is a lot of educational activity in terms of the scientific community and its branches going on to sort of direct attention on this disorder and improve it. Does that answer your question?
MS ANYADIKE-DANES: Yes, it does. Thank you very much

indeed.

Mr Chairman, subject to anybody else, I don't have any further questions.

THE CHAIRMAN: Okay. Are there any issues arising?

MR HUNTER: Perhaps if we could be allowed a few minutes.

THE CHAIRMAN: Yes.

(2.51 pm)

(A short break)

(3.18 pm)

MS ANYADIKE-DANES: Mr Chairman, I just have a few points to take up with Professor Gross. Two of them arise out of his reports.

Professor Gross, I wonder if I could take you to your report at 201-004-105. This is a report that you prepared for the inquiry dated 2 January 2011. I would like to take you to your paragraph numbered 3 where the question that you're asked and which you then go on to address is:

"Does this degree of hyponatraemia --

That is the hyponatraemia that was noted in Adam and that is addressed in the preceding paragraph: 123 millimoles at 9.32 am through the blood gas machine and then, by laboratory testing, 119 millimoles.

So the question was:

"Does this degree of hyponatraemia explain the

observed degree of cerebral oedema and the associated herniation of the brainstem into the foramen magnum, causing Adam's death?"

What you proceed to do is to put the arguments for and against. So first comes the argument for and then, over the page at 106, are the arguments against it. And then at 107, you summarise the position. You summarise it by saying:

"On the basis of the evidence and calculations that the precipitous and large fall of serum sodium to levels of severe hyponatraemia were sufficient to cause significant cerebral oedema and herniation of the brainstem into the foramen magnum, leading to demise."

If we pause there before going on to the next sentence, that large fall, that's the rate of fall that you were explaining to the chairman that was so significant from the point of view of Adam, so important as a cause of his demise. And then you go on to say --

THE CHAIRMAN: The "precipitous" is the reference to the rate of fall, isn't it?

MS ANYADIKE-DANES: Yes.

THE CHAIRMAN: Then the "large" is the amount that it fell, so you have the rate at which it fell plus the amount by which it fell? That's the combination?

A. "Precipitous" is speed and --

THE CHAIRMAN: Large is --

A. From 132 to 123, so we call it the delta --

MS ANYADIKE-DANES: I think because, in fact, Dr Coulthard, during your expert meetings in Newcastle, talked about falls that could be of that magnitude, but over a very much longer period and that could have a different outcome than if it happens with this speed. So that's your point.

A. Yes.

Q. Then you go on to your next sentence, which is:

"However, additional events must have occurred to lead to an increase of brain weight beyond that which is explained by osmotic swelling and the nature of those additional events is speculative."

So that was your thinking in January 2011. And then, I think, you had cause to revisit that and think about that in a report that you wrote in relation to your discussions of the experts' meeting on 22 February 2012.

Where I'd like to take you to is reference 201-015-235. I should say that the date of this report is 18 March of this year. So this is now your thinking in the light of all that you have seen and read.

Where I would like to start is your study of the literature, and I think that's what you were explaining

to the chairman and saying that you have looked at the literature and surveyed it to see what it really says about the positions being taken. Then you talk about:

"A small additional contributing event could have been Adam's rapidly progressive anaemia."

Can you just explain how that works in terms of your current thinking?

A. I was directed towards this question of what the anaemia might be meaning originally because anaemia was also a consideration in the initial verdict in 1996 and then I saw it come up again in the arguments that Dr Lesley Dyer used and I started to read up about this. I came to the following conclusion. I believe that it is correct to say that Adam's central venous pressure was 17 millimetres or higher. I belong to those that think that this was a real measurement and it was measuring a pressure that was present at the venous end of his cerebral vessels.

I did go back to the pressure tracing from the operating room that's all measured by Dr Dyer and it's sort of difficult to interpret and I had to use calipers and it took half an hour. I also confirm what Dr Dyer has stated that, for the first hour or so, Adam's mean arterial pressure in the entire circulatory system was -- the mean, not the systolic -- 75 millimetres of

mercury, which is not very high.

So subtracting 17, which I believe was real from the 75, which I learned from the tracing was really measured and present there, gives us 58 millimetres of mercury remaining. I am aware that some literature -- this difference is also called the perfusion pressure, the driving force that forces blood to go through brain vessels called the perfusion pressure, being defined as the difference using the mean arterial pressure and subtracting the pressure at the venous end from that gives us 58. The literature says for an adult brain, a perfusion pressure of less than 60 millimetres of mercury is called a critical perfusion pressure, meaning that this is where hypoxia -- not enough oxygen delivery to the brain tissue -- may begin when it falls below this critical perfusion pressure.

Dr Dyer referred us to an article by Chambers -- it's in the documents -- from 2006. A group of workers that had found out that in children of Adam's age, the critical perfusion pressure may be much less than 60. They talk about 48 in there. And Professor Kirkham during one discussion said that since this Chambers paper was in traumatised infants, the critical perfusion pressure in infants of Adam's age without trauma, such as from car accidents or so on, might be even a little

lower than 48. So let's assume that the critical perfusion pressure in a child of Adam's age without trauma might be between 45 and 48.

Well, then I thought that, at 58, Adam has about a 20 per cent reserve -- from 58 to 46 or 45 is about a 20 per cent difference. That 20 per cent reserve in being able to lower the blood pressure before getting to this critical point -- of course, lowering the blood pressure is the same as slowing the blood flow down by 20 per cent or having 20 per cent fewer red blood cells carrying the oxygen there, go through there.

Then I noticed that his haematocrit had been 31 at either the evening before or 7 am, I forgot, making him slightly anaemic, but the next measurement of haematocrit -- haematocrit is the definition of density of red blood cells in 100 ml of blood. So 31 volume per cent of 100 ml initially were red blood cells, and when it was next measured in him at 9.32 am, I think the value was 18 or 19 -- was it 18? So that's more than 40 per cent less. It's not necessarily blood loss. I think as Dr Keane has also been indicating, much of it may have been haemodilution. He received a lot of Hartmann's and HPPF, isotonic solutions -- 1.1 litres, I think -- between 7 and 9.30.

But the brain doesn't care how this anaemia comes

about. The haematocrit fell by more than 40 per cent. But he had a reserve in terms of the critical blood pressure of only 20 per cent. So in my eyes, this means that he transgressed the red line, causing the brain to receive too little oxygen.

There is one uncertainty in this that I couldn't find out about. When oxygen supply is in a critical zone, usually the blood vessels in the brain are able to respond to this by dilating and increasing the flow. One can assume that in Adam's situation, he was already maximally vasodilated and this mechanism didn't add, but there's no way of being sure about this.

If there was no further vasodilation possible, this, I believe, should or must have made, as it says here, his brain hypoxic. And a consequence of hypoxia, the first consequence is brain swelling, and I believe that in the maybe 20 or 30 minutes before his coning, hypoxia of his brain due to this low perfusion pressure -- basically because of the high CVP -- plus the dramatic fall in his haematocrit, in his red blood cell density and oxygen delivery to the brain, those two together caused him to have hypoxia or ischaemia, you could call it -- it's similar -- to his brain, and should have caused additional brain swelling. Something that I believe, if I understand the statements by the brain

pathologist, Dr Squier, correctly, is possibly to have happened within a relatively short while before his death without having caused major ischaemic changes in the brain that they could have picked up during autopsy later on.

As you may remember, there are no dramatic changes. Several statements say "no ischaemic changes in the brain", but when she was asked about this, Dr Squier said, "Yes, it takes a certain time and, theoretically, it's possible he had hypoxic changes, but we cannot discover it in the histological findings". So that was my thinking there. And that's why I think this may have added further to Adam's -- even though I said before and I still think, though, that the osmotic changes on the basis of the hyponatraemia alone, given the velocity with which it came into being, would be sufficient to have caused Adam's brain to develop coning on the basis of the literature I mentioned two hours ago when I talked about these ten articles and patients and seven articles in experimental literature fitting into that speed of the hyponatraemia, the same degree of hyponatraemia and the water changes in the brain having been measured and so forth. On the basis of this, I think the osmotic changes alone would have sufficed to cause that coning. But given the CVP -- which I think

was real -- and the haematocrit drop-off, I believe an additional element with reasonable likelihood was hypoxia of his brain having caused more brain swelling and the effect of it would have been to move the time of the coning up towards the beginning of the operation.

Q. Yes. I'm going to ask you to expand on that, but if you go to your next paragraph where you talk about another factor that could have contributed to the brain swelling. That's really the position of Adam's head during the surgery. The surgery's lasting about four hours or so and it's known that he was in the head down position, head slightly turned to one side. Are you able to express any view on how significant a contribution that position might have made to his cerebral oedema?

A. I think that this third thought or concept or idea, the effect of it is probably quite minor. The way it was written down here, I think this is a comment from early March this year or so.

Q. Yes.

A. But when I wrote this down, it was kind of an academic argument, which I think Dr Coulthard has also pointed out correctly because, given that the so-called blood-brain barrier is intact, which I believed at the time it would be in osmotic cerebral oedema, the

permeabilities of that inner lining of blood vessels, the permeability to water and sodium is so low that the resulting differences are minuscule, academic.

However, we discovered in the meantime two articles that claim -- what I said before was based on two older articles saying the blood-brain barrier in osmotically induced brain oedema is intact. These older articles -- one from the 60s and one from the 80s -- one by Wasterlain and one by Adler, they're in here if you want to see them. They had injected dye in experimental animals -- trypan blue, that was standard technique in those days -- and found that even in osmotic brain oedema, this dye, which distributes all over the body to all other organs, would not leave brain capillaries and go into brain and be demonstrable there. It's the classical definition of a tight intact blood-brain barrier.

In the last two weeks we came across two more recent articles, one by a person called Oztas in 2000 in experimental animals and a second more recent one in 2010 or 2011 by Yalcin-Cakmakli. I have it with me. They both claim that with more modern techniques, they're now finding that, in osmotic brain oedema, the blood-brain barrier breaks down for a few hours and then becomes tight again, implying that if they are

correct -- which I guess we have to see by further literature in the future, you want to see this confirmed, you know. If they were correct, then we would have to say that in osmotically-induced brain oedema, the brain capillaries are as leaky as are the capillaries in the leg or the liver.

Then this argument about the head-down position. Number 1, and number 2, there possibly being a little difference between the back of the head and the front of the head could have led to measurable effect, probably still not as important as what we mentioned in terms of the CVP and the anaemia, just five minutes ago, but it could have generated a little difference between posterior and anterior brain. That is based on 10 years' evidence. There are only two articles and this is apparently the first time that people are saying in osmotically-induced brain oedema, the capillaries become leaky, the blood-brain barrier breaks down, it opens up, it allows water and salt under elevated hydrostatic pressure to escape, which does not happen with a tight blood-brain barrier. And then that third argument could play a little more than academic role. I would like to put it that way.

Q. I understand. If you go to the page where you have your conclusions, which is over the page at 236, some of this

you have said, but it really is that second paragraph under the conclusion which I am being asked if you could expand and explain that.

A. Yes, this is -- I would call this an educated guess. But my thinking was that around the time at approximately 9.32 am when this measurement of the serum sodium being 123 and haematocrit having fallen to 19 per cent became known was, given the hypothesis I advanced before, the point where the forces driving the formation of this brain oedema reached its maximum. It was a time when there was probably still an osmotic gradient present and the anaemia was at its most severe point. You may remember that immediately after this, they gave the first bag of red blood cells leading to an increase of the haematocrit again. And that, to my view, makes it perhaps a little more likely to assume that around this time, plus or minus 10 minutes or so, would be a more likely assumption for the descent of the cerebellum and brainstem to have occurred rather than maybe an hour later when there may still have been an osmotic gradient.

Maybe by that time, an hour later, the sodium concentration may have dropped by one or two more millimetres [sic], but the haematocrit, having gone back up to 25 or 28 ...

So that is my thinking there, but as I said, this is an educated guess. It's hard to know. As I said before, I'm not quite sure about whether vasodilation was possible or not. I assume it was not possible. I am not quite sure whether the blood-brain barrier was leaky or tight. I assume it may have been leaky, but I don't know for sure. I don't know whether the serum sodium was exactly 123 at 9.32 as they had measured on this online blood gas machine. Maybe central lab at that time would have found 130 or 129. I'm sorry -- would have shown 120 or 119. I guess this issue has been raised before, that these online machines are good machines, but because they're not being serviced very well, people don't have time for that on the ward, they sometimes don't work that reliably and it's possible that Adam's serum sodium at 9.32 am could have been a little lower than the measure at 123 on that machine.

So because of these uncertainties I would like to be a tiny little bit vague in predicting. Also, as I mentioned before, there are no direct measurements saying so much water can be transported across the brain vessels into the brain in this much time. I had to quote a number of articles where similar patients had been described and extrapolate from there, all of which is not extremely scientific, but is just by comparison

saying: this is the most likely. But it seems to me that this could have been the likeliest point that I can think of.

Q. Okay. Can I then ask you one final set of questions because they're really to do with the same issue.

When you were talking about how well-known in 1995 or 1996 dilutional hyponatraemia was, you had, I think, marked a distinction between possibly what anaesthetists knew -- certainly paediatric anaesthetists -- and what maybe the rest in general medicine might know. The question is this: if one looks at three sorts of clinicians, this is in relation to your experience as to how well they are likely to be familiar with at least the condition of dilutional hyponatraemia, even if they don't know exactly the mechanism, how it works, how you treat it, but at least have heard of it. The first category is neurologists. The second would be anaesthetists. And the third would be paediatricians. Are you able to express a view?

MR UBEROI: May I just potentially add a few sub-categories of -- I don't know if the question is seeking to elicit 1995 or 2012 or both.

MS ANYADIKE-DANES: 1995 and 1996.

MR UBEROI: Thank you.

MR FORTUNE: I rise now because on what basis can

a consultant nephrologist give an opinion which in itself must be speculative?

MS ANYADIKE-DANES: Yes, Mr Fortune, you're quite right. It does require him to express a judgment and he expresses it on the basis that he sits on the editorial board of five journals, two of which are widely circulated, as I understand it, in Europe, including the United Kingdom, and one of which is widely circulated in the US and worldwide.

THE CHAIRMAN: Let's be very careful about this. As I understand the evidence that the professor has already given, he says that dilutional hyponatraemia may have been better known to paediatric anaesthetists than anaesthetists generally. And beyond that, in terms of other doctors, including paediatricians, you said, when I asked how well understood was dilutional hyponatraemia, you said that, even now, 15 years later, most doctors and paediatricians don't really know about it or understand it.

If that is the extent of his evidence, I'm really loath to allow him to speculate to break down into sub-categories neurologists, paediatricians and anaesthetists. I think that that is, by definition, highly speculative and not very helpful to me.

MR FORTUNE: Sir, I was about to say, by way of a question,

how are you going to be helped by any answer to that question?

THE CHAIRMAN: I don't think I am. I don't think I am because of the difficulty which the professor has already had in speculating or estimating the degree of knowledge held by different doctors.

MS ANYADIKE-DANES: I understand. Then can I ask you this -- well, the chairman has directed, so I will move on to a slightly different point.

If there is a measurement of serum sodium of 125, say, even if you're saying that in 1995/1996, we're talking about, many clinicians would not have been very aware of dilutional hyponatraemia as a condition, or at least how to treat it, what its mechanisms were, how it arises, is that a reading of serum sodium that is likely, whether or not you fully understand dilutional hyponatraemia or not, likely to have prompted concerns so far as you're aware?

MR FORTUNE: Sir, I rise at this stage because that's another way of putting the same question, really. It's: take a figure and how would you address it? The question started with a reference to clinicians.

MS ANYADIKE-DANES: Well, maybe if I can put it in this way so you see the way I'm going.

Professor Gross embarked on this area by saying

he is known as the professor who deals with hyponatraemia and many clinicians would contact him if they felt that their patients were in that condition, if you like, to seek his help as to maybe to confirm the diagnosis or how to treat it. And he expressed that as coming from clinicians across many different disciplines; is that correct?

A. (Witness nods).

Q. In that case, these clinicians are being prompted presumably by some level of serum sodium that is causing them to contact Professor Gross for his assistance. So the point that I'm trying to get at is: what is the level at which people are contacting you about -- what is their level of concern?

THE CHAIRMAN: Let's see where it takes us, but I think he can say, as a general point, that if he is being contacted by different people in your own hospital, what is it which is prompting them to make that contact. I think that's legitimate. I think it was certainly leading by saying "if the sodium level was 125".

When you said, at the start of your evidence, professor, in explaining your knowledge and your expertise, that you are the, as we would say here, the go-to person if somebody believes that they have a problem. What is it which prompts people to go to

you? Is it a reading below a certain level or is it because of other signs or what is it?

A. Obviously, there can be many reasons. But to answer your question, what concerns doctors almost no matter where they are -- I'm now talking about patients with chronic hyponatraemia -- is when the serum sodium concentration is around 120 or less or 122 or less, between 125 and 135, they oftentimes try to handle this themselves. If they do see that, despite their efforts, the patient gets worse every day, goes from 125 to 124 to 123, somewhere below 125, they start calling me. And if it's below 120 they always call me. This is chronic hyponatraemia. Adam had acute hyponatraemia.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Thank you. And when they call you, just so that maybe this gives a better understanding of it, what is it they're explaining to you that's given -- apart from the serum sodium level, what is it that they're explaining to you that's giving rise to their concern?

A. They are usually concerned that further worsening of the hyponatraemia their patient is having now would lead to consequences they wouldn't like to see, like grand mal seizures, epileptic fits. People can die from that. Andy Warhol died from hyponatraemia and grand mal

seizure, which happened to him in a hospital in 1987, and doctors know that. Say they are unsuccessful in their attempts in a given patient to limit the worsening of the hyponatraemia from day-to-day, then they get very upset and they call and they say: what should be I doing differently and how can I help this patient quickly to bring the sodium back up to 125 or 128 and prevent worse from happening?

Q. That is just what I wanted to ask you. When they are contacting you, are they simply saying: the serum sodium is wherever it is and it was such and such and it has done X and I have done Y and I don't seem to be able to rectify the situation? Are they talking about it in the context of hyponatraemia or are they simply saying, "We've got low serum sodium levels and we don't really know what to do about it"?

A. Probably the latter. What do you mean by "in the context" though?

THE CHAIRMAN: What are they saying to you? Are they saying, "I've got a patient with a consistently low reading of 120 or 121"; is that what they're saying?

MS ANYADIKE-DANES: Or, "I have a patient who seems to have hyponatraemia and I don't seem to be able to address it properly"?

A. They usually say, "I have an 82 year-old patient and he

had hyponatraemia of 123 yesterday and I did this and this and I just got a reading back and now it's 120. Don't you think I have to do something about it right now and what do you think I should be doing?" This is the commonest situation. Sometimes they report symptoms to me. They say, "I've someone at 124 and he's doing a lot of falls and he's so confused, do you think it could be because of the hyponatraemia and what should I be doing to find out?"

Q. That's the point I was getting at. Even though they don't actually know -- that's why they're coming to you, that's why I think the chairman called you the go-to person -- how to rectify it themselves, they're recognising that they've got hyponatraemia to address?

A. They do this more now than 10 years ago because hyponatraemia is slowly becoming better known. It can be like you imply, yes.

THE CHAIRMAN: Mr Fortune?

MR FORTUNE: I was just wondering where this line of questioning was going because what is quite clear is that there are professional colleagues of Professor Gross who recognise him as an expert in this field, who ring him up and set out their concerns about a patient and say in terms, "Professor, what should I be doing?" That's the question which my learned friend has

taken some time to ask.

MS ANYADIKE-DANES: Well --

THE CHAIRMAN: Okay. Have you exhausted that line of questioning?

MS ANYADIKE-DANES: I think we have.

THE CHAIRMAN: Have you exhausted everything?

MS ANYADIKE-DANES: I think I have.

THE CHAIRMAN: Right, okay. Thank you very much.

Professor, thank you very much --

MR HUNTER: Sir, sorry, I have two issues I would like to put to the professor.

THE CHAIRMAN: Go ahead, Mr Hunter.

Questions from MR HUNTER

MR HUNTER: First, professor, if I take you back to the boluses of dopamine.

A. To?

Q. The boluses of dopamine. If you remember, it was put to you that Dr Taylor had said that the two boluses of dopamine were given at around 10 am. I would like to ask you, first of all, what effect would the two boluses of dopamine have had on the CVP, if any?

A. My thinking would be that the dopamine was effective on the arterial side of the circulation, the pressure in the arteries. We've said before that the mean arterial pressure, initially, was 75 millimetres of

mercury and higher later on. The CVP measures the venous side, lower 17 millimetres increasing to 20 and a little more than ... So what you're asking me is: is it possible that the dopamine -- it caused two increases of the mean arterial pressure, could they have increased the mean arterial pressure in such a way that by pushing more blood through the brain capillaries, it contributed to changing the CVP, maybe increasing it a little bit?

I would have to say I don't know precisely, but I couldn't rule this out. I would estimate that this should be a very small effect, not -- if the systemic arterial pressure increased 3 or 4 millimetres of mercury in response to these two boluses, it might translate in increasing the CVP by 1 millimetre of mercury. That would be my guess. Not much more than this.

Q. Thank you, professor. If again, for the purposes of clarification -- there was some confusion over the timing of the boluses of dopamine and I think you were a bit confused as to why the dopamine was being given at 10 o'clock in the context of the statement. In other words, where Dr Taylor had said that the rationale for this was to increase the perfusion pressure to the donor kidney, which at that stage was not looking good. And I think you were -- that didn't sort of fit with you,

would that be correct, given that the time of this apparently was 10 o'clock and I think you then said maybe the timing isn't correct?

A. I thought --

Q. Can I just put to you before you answer that and ask for your comments on this -- Dr Taylor, by way of a letter dated 30 November -- which was two days after Adam's death -- wrote to the trust in preparation for his deposition. This was the first time that he had raised this matter, a matter of two days afterwards when the matter will obviously have been very fresh in his mind. So he gives the timing then at 10 am. Could I ask to your comment on that in relation to the timing?

A. I'm not quite sure I understood.

THE CHAIRMAN: He gives which timing at 10 am?

MR HUNTER: The timing for the two boluses of dopamine.

THE CHAIRMAN: That's what ends up in the statement which the professor was referred to; isn't that right?

MR HUNTER: That's correct, sir.

THE CHAIRMAN: What are you asking the professor to comment on?

MR HUNTER: I think what the professor was saying was that this would make sense that you would give the boluses of dopamine to help the perfusion pressure in the kidney; is that correct, professor? And this is at 10 o'clock.

So in other words, what I'm putting to the professor is that, if this is correct, then the clamps have been off by this time?

THE CHAIRMAN: Sorry, I thought what the professor's confusion was was about the statement, which was that, in his eyes, there's some query about the value of giving dopamine at all, but if you give it, you give it before the clamps come off in order to assist perfusion when that starts. Whereas Dr Taylor's statement, on one reading, was that he had given the dopamine after the clamps had come off and after the kidney did not seem to be perfusing well --

A. We're talking about the boluses?

THE CHAIRMAN: Yes.

A. What confused me was that the protocol seemed to say clamps came off at 10.30 or 11. This statement by Dr Taylor about the boluses says the boluses were given around 10. Half an hour before, according to the other evidence, the clamps came off. The reason he gave for the boluses was the kidney was not looking well perfused. This is a statement you can make only after having taken the clamps off. To me, it would make sense to make that -- or to terminate that statement at 10.40 or 10.45. I can't see -- so something doesn't fit here.

THE CHAIRMAN: It doesn't add up?

A. Yes, that doesn't add up. That's all I wanted to say.
MR HUNTER: That, in fact, is the point I was making, professor.
MR UBEROI: I'm not really [OVERSPEAKING]. The witness has commented -- offered his evidence twice now -- that there is a raised eyebrow to the timing of it. I don't think it's for this witness to speculate any further than that. It is not proper for him to be asked to do so and it is plain that he cannot.
THE CHAIRMAN: I thought Mr Hunter was going to go on to some other point, but what the professor's just repeated is the evidence he gave earlier this afternoon, Mr Hunter. To put it bluntly, he thinks it looks curious. The different strands of evidence do not easily sit together.
MR HUNTER: What I was putting to him, sir, was, of course, the contemporary in his evidence of the time -- Dr Taylor's statement of 30 November, which was, as we know, two days after.
THE CHAIRMAN: Yes, but that's what found its way into his inquest statement, which is the extract that he has already been taken to. In fact, it's the same statement that you're reading from.
MR HUNTER: It is.
THE CHAIRMAN: It's not a different statement.

MR HUNTER: The point is, sir, that it was made at the time.
THE CHAIRMAN: But I think, with all due respect, I'm not sure there's much more Professor Gross can say about that. That might be a matter I will have to look at in deciding how I can try to make the picture fit together, if I can.
MR HUNTER: There's one other quick matter.

Professor, if you as a nephrologist had gone in to a theatre where you saw a CVP reading of 30 -- and you've said here today that your evidence is that you think the CVP reading here was accurate. So if you go into a theatre and you see a CVP reading of 30, and then you see a serum sodium result of 123, can I ask you what would you do?
MR FORTUNE: Sir, I'm going to rise at this stage because, certainly, there has been evidence from Dr O'Connor on that matter insofar as her arrival in theatre is concerned. How is Professor Gross to be able to answer this matter? Because there has to be an assumption he's aware or made aware of the serum sodium level being 123.
THE CHAIRMAN: And it was not Dr O'Connor's evidence that she was aware of that? Is that point?
MR FORTUNE: She was certainly aware of the CVP.
MS ANYADIKE-DANES: It was Dr O'Connor's evidence that she was not aware of that 123 until after the inquiry

started because her evidence was that it was not clipped to the original notes in the way that it is now, so she never actually saw it. There is then an issue, which you'll ultimately have to determine as to how it got attached to the notes, but her clear evidence was that she didn't see it.

MR HUNTER: She certainly saw the CVP, sir.

THE CHAIRMAN: But the question you were going to put is: what would do you if you saw a CVP of 30 and you knew the sodium was 123?

MR FORTUNE: And that's not clarification, sir.

THE CHAIRMAN: But it isn't that. Even that wouldn't be the full question because you would then have to ask the witness about what he would do if he then asked Dr Taylor for an explanation and got the explanation which Dr Taylor gave, would that be enough or not, and we've covered that evidence today with Dr Coulthard. I'm not sure it's in any way helpful to me to go over the same areas again with this witness. So does that bring an end to the questioning?

Professor, thank you very much indeed for coming a long way over to help us with the inquiry. Thank you.

(The witness withdrew)

Can I say that we have contacted the solicitors for Mrs Murphy in relation to the possible evidence on

Monday of Ms Ramsay. You'll understand that Ms Ramsay's report was more -- she made general comments about the nursing. She was specifically critical of Miss Murphy and there has been an exchange. So I am hoping to hear later on today or tomorrow what their position is on Ms Ramsay. If they don't require Ms Ramsay to give evidence, then we will not be sitting on Monday. We will not be sitting tomorrow. Unfortunately, we do have to sit on Friday because we do have to deal with Dr Montague on Friday and fit in one other witness. I will tell you on Friday at the latest, if not tomorrow, whether we're sitting on Monday. We are trying to sort out the rest of next week. At the moment, it rather looks as if we're going to have to sit from Tuesday to Friday, which is not ideal, but if we can make any improvement on that, we will and let you know this Friday. Thank you very much.

(4.05 pm)

(The hearing adjourned until 10.00 am on Friday, 11 May)

I N D E X