

Wednesday, 9 May 2012

1

2 (10.00 am)

3

(Delay in proceedings)

4

(10.16 am)

5

DR MALCOLM COULTHARD (continued)

6

Questions from MS ANYADIKE-DANES (continued)

7

THE CHAIRMAN: Good morning.

8

MS ANYADIKE-DANES: Good morning, Mr Chairman.

9

Good morning, Dr Coulthard. There are a few things

10

to revisit with you and maybe a couple of substantive

11

points. One of the things I wonder if I could revisit

12

with you is the whole issue of re-zeroing.

13

Just so that we have it clear, what actually is

14

involved in re-zeroing? What physically do you do?

15

A. When you have a venous pressure device, essentially

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there is a central line, ie a tube, going into the

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child's vein, which is connected, and the fluid in that

18

is in direct continuity with the transducer, a small

19

pressure-reading device.

20

That will register a pressure trace, but in order to

21

zero it, what you're actually doing is determining at

22

what height on the child's body you're recording the

23

value of zero. The important point, just for people to

24

understand why we're doing that, is that what you're

25

wanting to do is to measure the pressure in relation to

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

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

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

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

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

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

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

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

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

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

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

1 neck. And you can see that the point that is being
2 taken as zero on the instrument is the point nearly
3 at the bottom where the horizontal bar is. That
4 horizontal bar in this case is a bar which has a spirit
5 level in it. So in this very, very simple model,
6 effectively you literally have a pressure column of
7 fluid which is being adjusted at the physical level of
8 the patient. Imagine if that was, say, attached to the
9 wall or at a stand and you jacked the patient's bed up
10 by 10 centimetres, you'd have to move the device up by
11 10 centimetres as well, otherwise it would read
12 a different level.

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

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

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

1 MR UBEROI: I simply wish to establish with the witness that
2 this is his invention, as it were, a solution to this
3 problem, but not something that was in general use in
4 1995.

5 A. No.

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

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

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

25 And just what he's talking about is that printout

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

5 A. Sorry --

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

9 A. Mm.

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

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

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

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

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

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

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

12 Personally what I have seen is just an ordinary
13 builder's spirit level that you would use to level
14 a shelf or something like that, itself maybe being 10 or
15 12 centimetres wide, being placed at one end very, very
16 carefully by somebody at one end of the child's chest
17 using the bottom marker, the bottom edge of the spirit
18 level. That's fine. Then somebody at the other end is
19 concentrating on putting the transducer at the right
20 level and maybe puts it at the -- this is something I've
21 seen happen -- top edge of the device, and then
22 automatically the readings will be positive by a value
23 equal to the height in centimetres of the actual
24 instrument.

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

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

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

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

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

19 A. No.

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

21 A. It is, yes.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

9 The instrument is ... If the instrument was
10 12 centimetres tall, that would make a difference of
11 12 centimetres of water pressure.

12 Q. Not mercury?

13 A. And the pressure transducer is actually calibrated --
14 you could do it in centimetres of water, but it is
15 actually calibrated in millimetres of mercury, and the
16 conversion factor of that because of the density of
17 mercury related to water is 0.144, which means that
18 12 centimetres of water converts to 9 millimetres of
19 mercury. So what I've then done is say: if I'd done it
20 with my spirit level at home, the one I happen to have
21 at home when I did this graph and took 9 millimetres off
22 all the values in theatre, you then achieve the trace
23 at the bottom, which would then indicate that when the
24 first reading was taken at something like quarter to 8
25 in the morning, after which time he'd arrived in theatre

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

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

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

15 A. In an erroneous way, yes.

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

25 A. Exactly.

1 Q. -- which is far more as you might expect if you were
2 actually getting accurate readings?

3 A. Exactly.

4 Q. And is therefore, towards the end of surgery, far more
5 consistent with the readings that they were getting in
6 paediatric intensive care; is that what you're saying?

7 A. That is exactly what I'm saying.

8 Q. Of course, nobody knows if they did actually introduce
9 that error. So far as I understand, all you are saying
10 is: if they did, that is what it would look like.

11 A. If they did and they would the same size spirit level
12 that I have at home, yes. Is it okay to just make one
13 other point, which I think is very important in relation
14 to why I made this speculation?

15 Q. Yes.

16 A. We talked yesterday -- and there has been a lot of
17 discussion from other experts -- about the possibility
18 that this monitor was actually -- the two possibilities
19 that it was either in continuity with the blood by
20 a fluid pathway by the blood in the right atrium or it
21 wasn't and it was in some way stuck in a neck vessel or
22 something like that.

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

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

8 Q. Yes.

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

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

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

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

17 Q. Thank you. Could I just ask you, if your hypothesis is
18 correct and if Adam's actual central venous pressure
19 would be as reflected in your bottom graph, what is the
20 significance of the level that you see there at
21 10 o'clock, which is just below 25? What's the
22 significance of that?

23 A. That would be a very high central venous pressure.

24 I mean --

25 THE CHAIRMAN: Is that effectively a fatal central venous

1 pressure?

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

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

9 MS ANYADIKE-DANES: Okay.

10 A. What you would anticipate to happen in a normally
11 controlled transplant situation would be for it to start
12 off at about 3, 4 or 5 and for the anaesthetist to
13 infuse normal saline through the procedure so that
14 it would gradually creep up to the target value of about
15 8 to 10 or 12 -- around 10 -- and that it would reach
16 that by the time that you got to the point where you can
17 open the clamps. That's what you would expect to
18 happen.

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

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

11 What then clearly happens on this trace is there is
12 a continuing creep up and then a sudden or an
13 exacerbation of that, so if you were to draw a smooth
14 line through the 8 o'clock until 10 o'clock, there's
15 an upward curve. That would be compatible with a child
16 being given too much fluid. So I think that around the
17 10 o'clock time, it would be compatible with a child who
18 had too much fluid.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

1 time, obviously.

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

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

9 Q. Exactly, yes.

10 A. If I was asked, for example, to see that at let's say
11 9 o'clock or 9.30 and could look at the trace up to
12 9.30, I would be surprised that it started off at high
13 as it did and I would be questioning why. I would be
14 suggesting this child did not need any more excess
15 fluid. The child has obviously, by 8.30, got a CVP as
16 high as you would already want it for opening the
17 clamps, and you certainly wouldn't want to continue
18 giving fluid.

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

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

6 A. Yes.

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

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

19 Q. In fairness to Dr O'Connor, she was not presented with
20 a value of -- as it appears on the screen it's just over
21 30, I think. She wasn't presented with that as being an
22 accurate value. What she saw was that figure, she asked
23 Dr Taylor about it, he gave her an explanation saying it
24 had started at 17 and it wasn't accurate for the reasons
25 that he gave, which you have been explaining to us and

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

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

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

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

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

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

6 A. Absolutely, yes.

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

11 A. Right --

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

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

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

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

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

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

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

12 Q. I understand?

13 A. -- and leave no permanent damage.

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

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

24 Q. Understood. Second option?

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

1 That would be my next question.

2 Q. Right.

3 A. "What was your last measurement of his plasma sodium?",
4 would be my next question. If you're giving that much
5 fluid, hyponatraemic fluid, you are going to make him
6 hyponatraemic. "What is his sodium?", would be my next
7 question.

8 Q. Okay.

9 THE CHAIRMAN: There has been a debate between you and the
10 other experts, doctor, about at what point Adam's
11 condition was irreversible.

12 A. Yes.

13 THE CHAIRMAN: When I asked you a few minutes ago about
14 whether that reading at about 10 o'clock of about 30 was
15 fatal, you said: no, if it was normal saline, it would
16 be reversible.

17 A. If it was due to -- that's right.

18 THE CHAIRMAN: In this scenario, you have a reading at
19 10 o'clock. It appears to be around 30.

20 A. Mm-hm.

21 THE CHAIRMAN: And you then find it's one-fifth normal
22 saline. Is that reversible?

23 A. That would depend. If you separate the medical issues,
24 which you can do quite clearly, compartmentalise them.
25 You have dealt with the surgery. The pulmonary side of

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

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

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

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

7 THE CHAIRMAN: Thank you.

8 MS ANYADIKE-DANES: Okay. Thank you.

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

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

22 A. Mm-hm.

23 Q. Perhaps just highlight that.

24 A. Yes, I can see it.

25 Q. That's completely unfilled.

1 A. Sure.

2 Q. Is that something that you would expect should be
3 filled, just as all the other measurements are?

4 A. Yes. I mean, you would expect it -- conventionally
5 it would be filled. What matters is what's in the
6 doctor's head rather than what's on the bit of paper.
7 And if somebody is watching the trace and it's
8 electronically available afterwards, then you could
9 argue whether that matters or not. But what matters
10 is that that is known. Yes, you would expect it to be
11 written down.

12 MR UBEROI: I'm happy for the question to be asked and the
13 answer given, but it is the anaesthetic record and I do
14 wonder really whether that was more a question for the
15 consultant anaesthetist.

16 MS ANYADIKE-DANES: It may be, but Dr Coulthard has been
17 dealing with a number of matters to do with CVP.

18 A. Perhaps from that perspective, I understand the
19 observation made. From the perspective of what was
20 happening at the time, if I arrived and I knew and
21 I could have a conversation with somebody who could tell
22 me what the CVP had been, I'd be happy at that. I would
23 not be happy that this would be the permanent end trace
24 because I would not then be able to go back to this
25 information later and see what had happened. Either it

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

3 Q. At some point?

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

13 Q. Ultimately recorded, okay. I also need to ask you, just
14 while we're on recording things, to do with the timings
15 of fluids. I wonder if we can go to Professor Savage's
16 statement at 002/2, page 19. I think it's the answer to
17 1(c), which is up at the top. On the third line:

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

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

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

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

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

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

5 So this is the evening, you can see the date of
6 26th. The note in the middle, which starts at 9.30,
7 that's a note that's made by Dr Cartmill, the SHO.
8 There you see:

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

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

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

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

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

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

1 Then his normal peritoneal dialysis until 6 am.
2 Then, of course, you have the information about the
3 cannula tissing. The point I'm going to ask you --
4 because this is your territory as a nephrologist,
5 getting the child ready, fit and in the best possible
6 condition -- slightly replete, I think you called it --
7 for his surgery. Do you expect these fluid
8 prescriptions and administrations to have attached to
9 them the times when they should start and the times when
10 they should finish? Should that be recorded anywhere?
11 A. Yes. I'm just kind of -- give me a minute just to get
12 to grips with the ... Sorry, this last one is a nursing
13 sheet, yeah? That's a --
14 Q. Yes, this is a nursing sheet. It's the only other place
15 you see a reference to 20 ml an hour, so far as I can
16 tell.
17 A. I think in clinical practice, what you would expect --
18 there's obviously a discussion, the junior doctor is
19 instructed that we're going to give 75 ml an hour of
20 intravenous fluids and then, reading into it, there's
21 then a discussion that actually we'll give that as 25 ml
22 an hour and some oral fluid until 6 o'clock, and
23 then ...
24 Q. Yes. The only issue is, do you record the time?
25 A. That sort of discussion would be done in this kind of

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

2 Q. Yes.

3 A. -- and that would be clearly made to the nurses.

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

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

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

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

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

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

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

16 The one that's up at the moment --

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

20 A. Okay.

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

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

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

1 A. If you give me a second, could I just look at the last
2 document?

3 Q. Of course.

4 A. That is the fluid chart where it will be recorded.
5 I can't see the whole thing. That's better. Right. So
6 we've got 2300 hours, the fluid type and ... I can't
7 see the ... To my reading of this, it looks to me --
8 I would interpret this -- yeah, that's better,
9 thank you -- as if it ... I don't ... It's not very
10 clear, is it? What one would hope to see is for each
11 hour, so for the hour, 23 to 24, in the horizontal row
12 labelled 24 on the left would be -- that would be what
13 the nurse would write at midnight and it would reflect
14 what happened between 11 o'clock and midnight. So the
15 hour that -- the row labelled "24" really means "23 to
16 24". What you would expect to be there on the left-hand
17 side on that -- in ... It depends how it's -- quite how
18 it's delivered. But you'd expect at least somewhere the
19 volume of fluid that was given that hour in a column,
20 which was clearly labelled "intravenous fluid".

21 Q. Yes. And what you can't see is any reference to the
22 75 ml an hour.

23 A. No, I can't see that at all.

24 Q. That's the point.

25 A. No.

1 Q. Thank you.

2 A. It's not clear what's happened there to me.

3 Q. Thank you. So my question to you was: since that had
4 been prescribed by the doctor, there should be some
5 reference to when it was administered and, presumably,
6 if it wasn't administered for some reason, that should
7 be recorded in the notes?

8 A. Yes.

9 Q. Thank you.

10 MR FORTUNE: Before we leave that document, could we ask
11 Dr Coulthard whether the document he's looking at is
12 a document he would expect to be completed by a junior
13 doctor or a nurse?

14 A. The document that's up at the moment, I would expect to
15 be completed by a nurse.

16 MS ANYADIKE-DANES: Thank you.

17 A. I know I haven't been asked another specific question,
18 but I don't want to leave it in a confused state. It
19 does seem to me that the information that we have here
20 suggests that the junior doctor was originally
21 considering -- or a discussion had led to considering
22 giving 75 ml an hour and then there's a change to giving
23 the fluid, from what we hear from Maurice Savage, to
24 giving it intravenously at 25 ml an hour and the rest
25 orally or nasogastrically. That's how I'm interpreting

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

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

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

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

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

16 Q. Yes.

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

1 writing.

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

5 A. It's not very clear.

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

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

21 Q. Yes.

22 A. -- on the information I have.

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

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

6 A. This would be very confusing.

7 Q. Thank you.

8 MR FORTUNE: To assist Dr Coulthard further, having referred
9 to Dr O'Neill, could Dr Coulthard first be shown
10 059-006-009? There's also some writing on the back of
11 the page, 010. That's Dr O'Neill's note at 11.30.

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

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

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

20 Okay, thank you.

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

1 A. Thank you. (Pause).

2 Okay, thank you.

3 MS ANYADIKE-DANES: Yes?

4 MR FORTUNE: Dr Coulthard, would that assist you in

5 understanding what the fluid regime overnight was to be?

6 A. No.

7 MS ANYADIKE-DANES: Thank you.

8 A. What this note is is a very clear note and plan about

9 the situation regarding going to theatre and various

10 important studies, the viral studies and so on, that are

11 necessary to get clear a plan of repeating his blood

12 tests and then a list of drugs to be administered in

13 theatre, which central line he would like given and

14 a discussion about which drugs to use, including

15 mannitol, his height, weight and surface area to aid

16 drug prescribing, and a post-operative drug. There's no

17 reference here to fluid management.

18 Q. Thank you. I wonder if I could now ask you another

19 question in relation to what you might have expected

20 Dr Savage to have known. Dr Savage was asked directly

21 whether he appreciated that it was possible during major

22 surgery, or during surgery of this sort in particular,

23 for the native kidneys to just simply shut down, which

24 is something which you had first raised in the experts'

25 discussions in Newcastle -- I think during the 9 March

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

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

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

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

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

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

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

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

24 MR UBEROI: He was.

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

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

15 Dr Coulthard?

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

18 Q. Yes.

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

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

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

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

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

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

12 A. Yes.

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

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

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

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

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

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

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

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

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

23 THE CHAIRMAN: After the event?

24 MS ANYADIKE-DANES: Yes.

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

1 different.

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

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

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

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

10 MS ANYADIKE-DANES: Yes.

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

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

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

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

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

1 both the chairman and my learned friend agreed with.

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

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

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

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

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

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

11 MS ANYADIKE-DANES: Yes.

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

14 MS ANYADIKE-DANES: Yes.

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

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

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

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

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

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

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

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

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

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

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

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

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

22 THE CHAIRMAN: It should have been, yes.

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

25 THE CHAIRMAN: Yes.

1 MR FORTUNE: And it would be reasonable --

2 THE CHAIRMAN: That's the end of Dr Savage's potential
3 involvement in this, that he's written a letter?

4 MR FORTUNE: I'm not saying it's the actual end, but the
5 ultimate decisions are taken either by the board or
6 indeed by the medical director because it would have
7 been open to the medical director to have suspended
8 Dr Taylor or, indeed, if he had any real concerns, to
9 have referred him to the General Medical Council for
10 investigation.

11 THE CHAIRMAN: I think I have already got Dr Haynes'
12 evidence on this, which is to the effect that he would
13 have had an informal meeting after the event with -- as
14 a medical director or clinical director, he would have
15 had an informal meeting with Dr Taylor and, if Dr Taylor
16 had appeared not to have accepted what had gone wrong or
17 not to have recognised what had gone wrong, he would
18 immediately have brought an end to the informal process
19 and would immediately have started a formal process.

20 MR FORTUNE: I wouldn't disagree with that, sir, but here is
21 Dr Coulthard, who is not wearing the hat, as Dr Haynes
22 was, of being a clinical director.

23 THE CHAIRMAN: Yes. Look, my immediate concern is I'm not
24 sure that I need really to hear much evidence on this,
25 if any evidence, from Dr Coulthard, because I -- this,

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

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

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

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

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

9 MS ANYADIKE-DANES: No, no.

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

12 MS ANYADIKE-DANES: Yes.

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

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

23 THE CHAIRMAN: Yes.

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

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

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

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

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

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

13 THE CHAIRMAN: Mr Fortune?

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

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

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

4 THE CHAIRMAN: Let me pause there. There are two separate
5 questions in that. One is whether it was common to do
6 it, okay? Let me ask you the first point. Was it
7 common for doctors to report other doctors to the GMC in
8 1995?

9 A. Not as common as it is now.

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

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

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

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

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

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

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

1 duty as a doctor to go to a higher authority.

2 THE CHAIRMAN: Okay. Mr Fortune?

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

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

10 THE CHAIRMAN: Okay.

11 MR FORTUNE: Thank you, sir.

12 THE CHAIRMAN: Is there anything more for this witness?

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

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

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

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

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

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

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

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

10 MR FORTUNE: Is that correct?

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

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

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

15 THE CHAIRMAN: Okay.

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

18 (The witness withdrew)

19 Timetabling Discussion

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

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

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

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

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

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

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

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

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

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

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

15 MR McALINDEN: Yes.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

11 MR UBEROI: Yes, sir.

12 MS ANYADIKE-DANES: Before you make a decision on that,

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

24 I recognise it that was a difficult couple of days.

25 Frankly, I think it's been very difficult for all the

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

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

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

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

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

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

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

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

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

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

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

14 MR UBEROI: I am grateful, sir.

15 THE CHAIRMAN: Mr McBrien.

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

19 MR UBEROI: No.

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

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

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

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

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

10 MR McALINDEN: Yes.

11 MR FORTUNE: Sir, can I come back to the week as a whole?

12 Because you started with Monday and, hopefully,
13 Sally Ramsay will not be required. If you look at the
14 remaining four days, the heaviest day is likely to be
15 Wednesday and Professor Koffman. I see my learned
16 friend raises her eyes in agreement. But in reality,
17 we have got six other witnesses who should be taken
18 quite shortly. In the circumstances, would it not be
19 possible for the two junior doctors to be brought
20 forward, one on to Tuesday, one on a Thursday with a bit
21 of arm twisting?

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

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

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

5 Let me go and double-check this information.

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

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

14 THE CHAIRMAN: Yes. That's not productive.

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

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

1 THE CHAIRMAN: Okay, thank you very much. 1 o'clock?
2 (12.12 pm)
3 (The Short Adjournment)
4 (1.00 pm)
5 (Delay in proceedings)
6 (1.15 pm)
7 PROFESSOR PETER GROSS (called)
8 Questions from MS ANYADIKE-DANES
9 MS ANYADIKE-DANES: Good afternoon, Professor Gross.
10 Professor, do you have there your CV?
11 A. Do I have it?
12 Q. Yes, with you. I will just provide with you a copy now.
13 (Handed).
14 A. When it was requested again, I was already travelling
15 and I had left Dresden, so I think what was sent to you
16 by my secretary was sort of a short form.
17 Q. That is it there, is it?
18 A. Yes.
19 Q. That's your short form without all your publications, as
20 I understand it.
21 A. It doesn't list my 180 publications.
22 THE CHAIRMAN: Good.
23 A. Too bad.
24 MS ANYADIKE-DANES: I just wonder if you could explain your
25 qualifications. I see that in 1977 you were certified

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

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

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

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

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

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

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

19 A. Mm-hm.

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

23 A. Yes, you could say that. Yes.

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

1 is that continental Europe, is it worldwide?

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

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

7 A. Included in there.

8 Q. Thank you.

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

12 Q. I understand.

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

18 Q. And number three?

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

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

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

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

25 So that has refined the questions that I would like

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

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

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

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

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

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

14 When I was in Berlin later, I did not work
15 particularly focused on hyponatraemia; I had other
16 things to do. But since I came to Dresden, which is in
17 1994, for the last 15 years I think I have performed six
18 more clinical published studies on hyponatraemia, how it
19 comes about, which patients get it, what the
20 consequences are and, most recently, in the last eight
21 years, how we can treat it. There is a new group of
22 substances, drugs, that have been developed for the
23 first time that enable doctors to treat hyponatraemia
24 specifically, something we never had to our disposal
25 ever before. And I have been involved in testing the

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

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

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

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

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

8 One of the issues I would like to put to you is the
9 question of the extent to which, when the anaesthetist
10 is in the operating theatre itself, the observable
11 features of the patient can assist in determining
12 whether the patient is being fluid overloaded. I will
13 give you an example, which may help. If we call up
14 011-002-007.

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

18 "The calculation --

19 THE CHAIRMAN: This is a statement by?

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

25 This is a letter that Dr Taylor wrote to

1 Dr Murnaghan. You'll see that the date of that is
2 30 November, so it's extremely close to the date when
3 Adam died. This is part of his, as you see there,
4 statement of Dr Taylor. That's going to turn itself
5 into a statement for the coroner, but Adam died on the
6 28th; this is being written on the 30th.

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

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

12 Then in parentheses he says:

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

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

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

22 Q. Yes, of course.

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

1 Q. I understand.

2 A. So we're not now talking about hyponatraemia, we're
3 talking about fluid overload, I understand.

4 Q. Let's first start with fluid overload, then we'll see if
5 it assists with hyponatraemia.

6 A. Your question then was: what could the anaesthetist
7 in the operating room have -- what kind of information
8 could he have used, if I understand you correctly, to
9 judge whether fluid overload was present or not;
10 correct?

11 Q. Yes. The observable features. So these descriptions he
12 gives.

13 A. And Dr Taylor, if I understand this sentence here
14 correctly, is saying that into this judgment, it has to
15 be fed into this judgment what the skin colour of the
16 patient is, whether the skin looks mottled, irregularly
17 coloured or not. What he can find out about the
18 peripheral perfusion, by which I understand whether the
19 hands are warm, the feet are warm and well perfused.
20 What the pulse volume -- the strength of the palpable
21 pulse -- might be, whether it is very flat or whether
22 it's strongly pulsating and perhaps what the pulse
23 response to fluids is.

24 MR UBEROI: May I rise? Sorry to interrupt, but perhaps we
25 could slightly rephrase that, if we may. Rather than

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

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

9 MR UBEROI: Thank you.

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

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

1 helped it.

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

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

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

15 A. Mm-hm.

16 THE CHAIRMAN: Thank you.

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

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

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

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

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

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

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

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

10 A. That's exactly the problem.

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

15 A. Is it possible to distinguish between dilutional
16 hyponatraemia?

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

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

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

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

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

8 Q. Yes.

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

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

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

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

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

7 This is the response of Dr Taylor:

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

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

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

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

25 "Explain in detail your assertion that the

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

3 And he says:

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

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

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

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

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

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

15 I suppose that this would have been so in Adam
16 during the time when he received these infusions from
17 7 am to 11 am on 27 November. It would have been
18 similar because I saw at least one measurement of
19 glucose that was obtained in him in the paediatric ICU
20 later on. It was 7-something, close to normal. He was
21 not a diabetic being under lack of insulin and being
22 unable to take up the glucose into his cells and leaving
23 it outside.

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

1 that then generates or makes this seemingly isotonic
2 0.18 per cent NaCl with 4.3 per cent dextrose or
3 glucose, what seems to make it isotonic in chemical
4 terms turns into hypotonic 0.18, one fifth normal saline
5 biologically in an organism that has glucose around.

6 This is why -- and I think Dr Taylor in his most
7 recent statement explained that he accepts that
8 0.18 per cent NaCl with this dextrose is a hypotonic
9 fluid. He said that in writing. And I think he said he
10 accepts that this led to dilutional hyponatraemia in
11 Adam and so on.

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

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

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

1 was always, so long as he wasn't a diabetic, his body
2 was always going to metabolise it in that way and you
3 were always going to be left with residual free water?
4 A. I think at the time this was made, could not be said to
5 have been a significant, a biologically relevant
6 argument.
7 Q. Thank you.
8 A. Would you perhaps give me a second to add something to
9 what I said before?
10 Q. Yes.
11 A. There is a tiny point I forgot to mention. I wanted to
12 mention it. I also wish to point out that there is
13 literature, specifically from the United Kingdom -- and
14 this is in the documents I sent to you -- that appeared
15 between 1985 and 2006. There are five or six articles
16 that speak about this particular nature of the
17 0.18 per cent saline with 4.3 per cent dextrose, which
18 is apparently a solution that was used in this country
19 preferentially by anaesthetists. There are several
20 publications that speak about the risks to patients in
21 terms of causing water retention in the post-operative
22 state and, with it, hyponatraemia, and warn against it.
23 There was even an alert, I think, that came out in --
24 I forget, maybe 2004 or so -- warning anaesthetists
25 against using this particular 0.18 per cent saline with

1 4.3 per cent dextrose and replacing it with half normal
2 saline was the proposal.

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

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

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

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

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

1 this is only a comparison.

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

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

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

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

19 Q. Why do you say that?

20 A. Well, because we know from the CT that was taking the
21 computerised tomogram that was obtained in Adam at
22 14.15 of the 27th, they saw diffuse -- I think Dr Anslow
23 said the brain is very swollen. And we know that the
24 cerebellum descended into the foramen magnum as a sign
25 of the swelling. We know that the autopsy describes

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

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

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

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

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

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

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

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

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

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

1 article by Arieff in the British Medical Journal --

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

8 A. Does it give me a?

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

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

13 Q. Yes, of course.

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

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

1 A. I would say that, yes.

2 Q. Okay, thank you.

3 A. If you would allow me to finish the earlier question.

4 I was sort of worried that there are no direct
5 measurements on how fast brain oedema can start to be
6 there. I therefore obtained additional literature.

7 I sent you a comment that quotes ten articles discussing
8 patients with acute hyponatraemia, many of them died,
9 not all of them. And it quotes, I think, seven articles
10 from experimental literature. And they do show that
11 within two to four hours from the induction of acute
12 hyponatraemia of the degree that was present here --
13 120 millimoles per litre plus or minus 2 millimoles per
14 litre -- that, indeed, when those animals then were --
15 all those animals developed epileptic seizures, coma or
16 died within two to four hours. When their brains were
17 sectioned, gross cerebral oedema was found. And when
18 they determined in these articles -- I'm now talking
19 about the experimental animals -- brain water content,
20 they could find that was increased 8 to 15 per cent.
21 These articles say -- I have them all with me. If
22 someone cares to see, we can go through that.

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

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

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

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

21 Q. Thank you. I'd like to take you to just a small part of
22 one of your reports, just so that you can give us your
23 observation or expand on your observation. It's
24 201-004-124. It's in the middle of, roughly, that long
25 first paragraph. You're talking about -- I think it

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

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

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

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

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

24 A. Yes.

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

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

3 A. Describing or remembering it differently.

4 Q. Yes.

5 A. Maybe not so much in Adam because he was smaller, but
6 usually the operative field is not very large, it's not,
7 frankly, open so that everybody could see from
8 a distance. Only the people very close to it have
9 a good look. There is bleeding around the kidneys,
10 covered by blood. There are other things around.

11 I think if you asked three surgeons about what the
12 kidney looked like in the first five minutes and in the
13 second ten, in the third ten, it could well be that you
14 get two different opinions even though they all saw the
15 same thing.

16 Q. I understand.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

19 A. Today, I would still say exactly the same. Transplanted
20 kidneys that are in very good conditions and function
21 100 per cent well sometimes, as the surgeons say -- I'm
22 sorry for saying that way -- they start peeing on the
23 table. It means they start producing a lot of urine
24 visibly while the patient is still on the table and,
25 therefore, a well-functioning transplant might have

1 contributed by excreting something like perhaps 200 to
2 300 ml of urine in the first 45 minutes.

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

8 Q. Yes.

9 A. And as I seem to remember it, the clamps were opened at
10 10.30; is that correct?

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

12 A. Approximately. So supposedly, it would have taken 15 to
13 20 minutes for the water production to have become more
14 copious. So by then, it supposedly would have been
15 10.50 to 11.00. So in trying to answer the question,
16 "Could this have possibly prevented the coning?", brings
17 us to the other question: when did the coning take
18 place? If you assume that the coning took place towards
19 the very end of the operation, which was near
20 12 o'clock, I think.

21 Q. Yes.

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

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

4 Q. The damage is done to the brainstem?

5 A. Yes.

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

11 A. Yes, I would say that.

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

15 A. Yes, I agree with you.

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

19 A. Mm-hm.

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

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

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

8 Dr Taylor gave twice as much. He gave between 2 and
9 4 micrograms per kilogram per minute. So I'm a bit
10 confused there. But I think he indicates somewhere that
11 the purpose was to help kidney perfusion.

12 Q. Yes.

13 A. And probably the best interpretation of this is to have
14 the dopamine contribute to the vasodilation, expected
15 vasodilation, in the kidney. If you give more dopamine
16 than the amount indicated, it turns from a potential
17 vasodilator into a vasoconstrictor. It's an agent that
18 has different effects depending on how much you give.
19 It seems to me that the two boluses he gave around
20 10 am, which was 2 micrograms or something like this
21 repeated within five minutes, was to increase blood
22 pressure. And apparently, at least this is what
23 I learned from his statement -- do we have a comment why
24 he gave the boluses? Someone said, I think it was him,
25 it was for blood pressure reasons.

1 Q. Let's start with the first mention of it. If we go to
2 058-035-133. If you look, this is the note from
3 Professor Savage. If you look at:
4 "In theatre to have."
5 Under (iv) you can see the dopamine.

6 A. Mm-hm.

7 Q. What did you say was the optimum?

8 A. 0.5 to 2 --

9 Q. So --

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

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

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

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

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

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

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

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

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

20 THE CHAIRMAN: Sorry, let's go back a bit. Professor Gross
21 originally said that the amount given was twice the
22 amount in the literature. He said that range was 0.5 to
23 2 and then he was taken to this document which says 2 to
24 3. And then the professor said, in fact, there's
25 a dispute in the literature as to what the amount is.

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

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

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

10 A. I don't mean to.

11 THE CHAIRMAN: Thank you.

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

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

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

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

1 appears to have been, to try and induce more
2 vasodilation specifically in the kidney. It is believed
3 to have this vasodilating effect at this dose, I said
4 0.5 to 2 micrograms per kilogram preferentially in the
5 kidney. That was the initial intention. And I think
6 nephrologists in general -- myself included -- would not
7 be critical of this.

8 Q. Thank you.

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

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

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

22 Is that the statement to which you were referring?

23 A. Yes.

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

1 A. Exactly.

2 Q. -- to improve the blood flow to the donor kidney;

3 is that right?

4 A. Yes.

5 Q. If that's what you wanted to do with it, then when would

6 you be infusing that in relation to the release of the

7 clamps?

8 A. Yes, that's a good point. It didn't strike me before,

9 but you're quite correct. If that was the intention,

10 you would probably start to do this half an hour before

11 release of the clamps. But this infusion was given from

12 the beginning of the operation or from the beginning of

13 infusions.

14 Q. And what would be the effect of doing that as opposed to

15 doing it briefly, if I can put it that way, before the

16 release of the clamps just to encourage dilation?

17 A. The dose of the dopamine, that is being given here is so

18 low that there would probably be no discernable effect

19 on peripheral perfusion, blood pressure, systolic, mean

20 blood pressure. I guess one would have to say no

21 significant discernable effect. Also no harm.

22 MR UBEROI: Can I just rise by reference to that, sir?

23 I hope I'm not going to confuse matters further.

24 011-014-101, please, which is also the deposition.

25 A quarter of the way down:

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

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

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

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

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

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

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

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

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

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

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

1 significance might be. It may be, sir, that there's
2 absolutely no significance at all, whether you started
3 with a low dose and added two boluses at roughly
4 10 o'clock of the amount that is said. There may be
5 absolutely no significance in the way that he said there
6 would actually be no discernable effect in the first
7 lot. If there's no discernable effect, then there's
8 nothing that needs to be asked of Dr Taylor and it
9 really doesn't matter why he had two boluses at
10 10 o'clock if it has no discernable effect and that was
11 the question I was going to put to Professor Gross.

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

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

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

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

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

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

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

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

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

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

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

19 MR HUNTER: Correct.

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

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

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

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

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

22 Q. Dr Taylor.

23 A. Doctor?

24 Q. Taylor.

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

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

13 Q. Thank you.

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

16 Q. Yes, exactly.

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

22 A. The what?

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

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

4 A. This is a difficult question.

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

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

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

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

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

9 Q. Yes.

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

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

12 A. He knew it?

13 Q. Yes.

14 A. I mention it only because it's an article that reports
15 16 children. So one reason perhaps for why it is not so
16 well-known amongst all the groups I mentioned before
17 could have to do with the fact that this is considered
18 paediatrics.

19 Q. Yes.

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

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

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

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

10 Q. Yes.

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

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

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

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

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

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

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

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

9 A. Yes.

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

18 A. Yes, I agree with you.

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

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

1 Q. No.

2 A. It is believed that the majority of hyponatraemic
3 patients that we see have mild hyponatraemia, 128, 130.
4 About 8 to 15 per cent of a hospital population that's
5 being measured at random will turn out to be
6 hyponatraemic in that range. The symptoms are usually
7 mild. They're forgetful, they're a little bit
8 depressed, they can't concentrate. These kind of
9 things. And it is believed that their quality of life
10 is lower than need be, particularly relevant in elderly
11 people. It is believed that they fall more often and
12 fracture bones. So the hyponatraemia community thinks
13 that, in the next ten years, doctors need to be educated
14 that it's now possible to treat hyponatraemia --
15 I mentioned this in going by an hour ago, I think -- we
16 sort of have to try and improve quality of life and
17 prevent fractures and those kinds of things from
18 treating mild hyponatraemia. But that is something
19 else.

20 Q. Yes.

21 A. It doesn't really have much to do with our case here
22 today.

23 Q. But in terms of educating about the more severe types
24 that lead to the sorts of consequences for Adam, what
25 do you think is the work still to be done in relation to

1 that?

2 A. Sorry, I missed that.

3 Q. No, no.

4 A. I could have said that also before. There has been --
5 now that specific therapy is available, there has been
6 a rather strong educational effort that took place
7 in the literature over the last three years and I think
8 almost all medical and nephrological, endocrinology
9 journals have run an overview and educational articles
10 very recently, in the last two or three years, in how
11 best to establish the differential diagnosis, what the
12 consequences of treatment are, which kinds of
13 hyponatraemia should be treated and in which way. So
14 the field is sort of receiving renewed attention
15 recently and now that doctors have a tool available to
16 them to improve these patients' lots, everybody is more
17 interested than they ever were before and hyponatraemia
18 is now something that at each congress you go to,
19 they're having a major session, there are a bunch of
20 speakers and there are symposia and there is a lot of
21 educational activity in terms of the scientific
22 community and its branches going on to sort of direct
23 attention on this disorder and improve it. Does that
24 answer your question?

25 MS ANYADIKE-DANES: Yes, it does. Thank you very much

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

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

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

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

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

21 MS ANYADIKE-DANES: Yes.

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

25 A. "Precipitous" is speed and --

1 THE CHAIRMAN: Large is --

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

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

9 A. Yes.

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

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

15 So that was your thinking in January 2011. And
16 then, I think, you had cause to revisit that and think
17 about that in a report that you wrote in relation to
18 your discussions of the experts' meeting on
19 22 February 2012.

20 Where I'd like to take you to is reference
21 201-015-235. I should say that the date of this report
22 is 18 March of this year. So this is now your thinking
23 in the light of all that you have seen and read.

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

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

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

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

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

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

1 mercury, which is not very high.

2 So subtracting 17, which I believe was real from the
3 75, which I learned from the tracing was really measured
4 and present there, gives us 58 millimetres of mercury
5 remaining. I am aware that some literature -- this
6 difference is also called the perfusion pressure, the
7 driving force that forces blood to go through brain
8 vessels called the perfusion pressure, being defined as
9 the difference using the mean arterial pressure and
10 subtracting the pressure at the venous end from that
11 gives us 58. The literature says for an adult brain,
12 a perfusion pressure of less than 60 millimetres of
13 mercury is called a critical perfusion pressure, meaning
14 that this is where hypoxia -- not enough oxygen delivery
15 to the brain tissue -- may begin when it falls below
16 this critical perfusion pressure.

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

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

4 Well, then I thought that, at 58, Adam has about
5 a 20 per cent reserve -- from 58 to 46 or 45 is about
6 a 20 per cent difference. That 20 per cent reserve in
7 being able to lower the blood pressure before getting to
8 this critical point -- of course, lowering the blood
9 pressure is the same as slowing the blood flow down by
10 20 per cent or having 20 per cent fewer red blood cells
11 carrying the oxygen there, go through there.

12 Then I noticed that his haematocrit had been 31 at
13 either the evening before or 7 am, I forgot, making him
14 slightly anaemic, but the next measurement of
15 haematocrit -- haematocrit is the definition of density
16 of red blood cells in 100 ml of blood. So 31 volume
17 per cent of 100 ml initially were red blood cells, and
18 when it was next measured in him at 9.32 am, I think the
19 value was 18 or 19 -- was it 18? So that's more than 40
20 per cent less. It's not necessarily blood loss.

21 I think as Dr Keane has also been indicating, much of it
22 may have been haemodilution. He received a lot of
23 Hartmann's and HPPF, isotonic solutions -- 1.1 litres, I
24 think -- between 7 and 9.30.

25 But the brain doesn't care how this anaemia comes

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

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

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

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

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

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

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

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

20 Q. Yes.

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

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

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

18 In the last two weeks we came across two more recent
19 articles, one by a person called Oztas in 2000 in
20 experimental animals and a second more recent one in
21 2010 or 2011 by Yalcin-Cakmakli. I have it with me.
22 They both claim that with more modern techniques,
23 they're now finding that, in osmotic brain oedema, the
24 blood-brain barrier breaks down for a few hours and then
25 becomes tight again, implying that if they are

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

7 Then this argument about the head-down position.
8 Number 1, and number 2, there possibly being a little
9 difference between the back of the head and the front of
10 the head could have led to measurable effect, probably
11 still not as important as what we mentioned in terms of
12 the CVP and the anaemia, just five minutes ago, but it
13 could have generated a little difference between
14 posterior and anterior brain. That is based on
15 10 years' evidence. There are only two articles and
16 this is apparently the first time that people are saying
17 in osmotically-induced brain oedema, the capillaries
18 become leaky, the blood-brain barrier breaks down, it
19 opens up, it allows water and salt under elevated
20 hydrostatic pressure to escape, which does not happen
21 with a tight blood-brain barrier. And then that third
22 argument could play a little more than academic role.
23 I would like to put it that way.

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

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

4 A. Yes, this is -- I would call this an educated guess.
5 But my thinking was that around the time at
6 approximately 9.32 am when this measurement of the serum
7 sodium being 123 and haematocrit having fallen to
8 19 per cent became known was, given the hypothesis
9 I advanced before, the point where the forces driving
10 the formation of this brain oedema reached its maximum.
11 It was a time when there was probably still an osmotic
12 gradient present and the anaemia was at its most severe
13 point. You may remember that immediately after this,
14 they gave the first bag of red blood cells leading to an
15 increase of the haematocrit again. And that, to my
16 view, makes it perhaps a little more likely to assume
17 that around this time, plus or minus 10 minutes or so,
18 would be a more likely assumption for the descent of the
19 cerebellum and brainstem to have occurred rather than
20 maybe an hour later when there may still have been an
21 osmotic gradient.

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

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

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

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

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

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

20 MR UBEROI: May I just potentially add a few sub-categories
21 of -- I don't know if the question is seeking to elicit
22 1995 or 2012 or both.

23 MS ANYADIKE-DANES: 1995 and 1996.

24 MR UBEROI: Thank you.

25 MR FORTUNE: I rise now because on what basis can

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

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

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

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

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

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

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

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

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

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

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

25 Professor Gross embarked on this area by saying

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

8 A. (Witness nods).

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

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

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

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

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

14 THE CHAIRMAN: Thank you.

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

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

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

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

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

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

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

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

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

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

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

18 THE CHAIRMAN: Mr Fortune?

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

1 taken some time to ask.

2 MS ANYADIKE-DANES: Well --

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

5 MS ANYADIKE-DANES: I think we have.

6 THE CHAIRMAN: Have you exhausted everything?

7 MS ANYADIKE-DANES: I think I have.

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

9 Professor, thank you very much --

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

12 THE CHAIRMAN: Go ahead, Mr Hunter.

13 Questions from MR HUNTER

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

16 A. To?

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

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

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

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

17 Q. Thank you, professor. If again, for the purposes of
18 clarification -- there was some confusion over the
19 timing of the boluses of dopamine and I think you were
20 a bit confused as to why the dopamine was being given at
21 10 o'clock in the context of the statement. In other
22 words, where Dr Taylor had said that the rationale for
23 this was to increase the perfusion pressure to the donor
24 kidney, which at that stage was not looking good. And
25 I think you were -- that didn't sort of fit with you,

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

4 A. I thought --

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

14 A. I'm not quite sure I understood.

15 THE CHAIRMAN: He gives which timing at 10 am?

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

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

19 MR HUNTER: That's correct, sir.

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

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

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

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

13 A. We're talking about the boluses?

14 THE CHAIRMAN: Yes.

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

25 THE CHAIRMAN: It doesn't add up?

1 A. Yes, that doesn't add up. That's all I wanted to say.

2 MR HUNTER: That, in fact, is the point I was making,
3 professor.

4 MR UBEROI: I'm not really [OVERSPEAKING]. The witness has
5 commented -- offered his evidence twice now -- that
6 there is a raised eyebrow to the timing of it. I don't
7 think it's for this witness to speculate any further
8 than that. It is not proper for him to be asked to do
9 so and it is plain that he cannot.

10 THE CHAIRMAN: I thought Mr Hunter was going to go on to
11 some other point, but what the professor's just repeated
12 is the evidence he gave earlier this afternoon,
13 Mr Hunter. To put it bluntly, he thinks it looks
14 curious. The different strands of evidence do not
15 easily sit together.

16 MR HUNTER: What I was putting to him, sir, was, of course,
17 the contemporary in his evidence of the time --
18 Dr Taylor's statement of 30 November, which was, as we
19 know, two days after.

20 THE CHAIRMAN: Yes, but that's what found its way into his
21 inquest statement, which is the extract that he has
22 already been taken to. In fact, it's the same statement
23 that you're reading from.

24 MR HUNTER: It is.

25 THE CHAIRMAN: It's not a different statement.

1 MR HUNTER: The point is, sir, that it was made at the time.

2 THE CHAIRMAN: But I think, with all due respect, I'm not
3 sure there's much more Professor Gross can say about
4 that. That might be a matter I will have to look at in
5 deciding how I can try to make the picture fit together,
6 if I can.

7 MR HUNTER: There's one other quick matter.

8 Professor, if you as a nephrologist had gone in to
9 a theatre where you saw a CVP reading of 30 -- and
10 you've said here today that your evidence is that you
11 think the CVP reading here was accurate. So if you go
12 into a theatre and you see a CVP reading of 30, and then
13 you see a serum sodium result of 123, can I ask you what
14 would you do?

15 MR FORTUNE: Sir, I'm going to rise at this stage because,
16 certainly, there has been evidence from Dr O'Connor on
17 that matter insofar as her arrival in theatre is
18 concerned. How is Professor Gross to be able to answer
19 this matter? Because there has to be an assumption he's
20 aware or made aware of the serum sodium level being 123.

21 THE CHAIRMAN: And it was not Dr O'Connor's evidence that
22 she was aware of that? Is that point?

23 MR FORTUNE: She was certainly aware of the CVP.

24 MS ANYADIKE-DANES: It was Dr O'Connor's evidence that she
25 was not aware of that 123 until after the inquiry

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

7 MR HUNTER: She certainly saw the CVP, sir.

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

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

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

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

23 (The witness withdrew)

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

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

18 (4.05 pm)

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

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