Tuesday, 13 November 2012

(10.00 am)

(Delay in proceedings)

(12.35 pm)

THE CHAIRMAN: Good afternoon. I'm sorry for the late sitting. Let me explain on the record how that has come about. Yesterday afternoon, after we reached the later stages of Dr Scott-Jupp's evidence -- which will be finished in a few weeks' time -- I expressed concern to inquiry counsel, Ms Anyadike-Danes, about the fact that I had now heard extensive evidence from Professor Neville, Dr Aronson, Professor Cartwright and Dr Scott-Jupp. We have to finish Professor Neville and Dr Scott-Jupp and then we are to hear from Dr MacFaul today.

I asked her to explore with the parties whether there was any consensus about the extent to which it was necessary to go through with Dr MacFaul all of the issues which we've already gone through over a number of days with the expert witnesses because I feel that I have a fairly clear grasp and, in fact, there's fairly significant consensus about many of the things which went wrong during Claire's treatment between 21 and 23 October 1996.

What I wanted to happen this morning was to find if

there was a trimmed way in which we could go through Dr MacFaul's evidence while recognising that there are specific issues which will have to be raised with him, not least the issue about his criticism, which is based on a textbook which was not the current edition in 1996.

I was therefore extremely surprised and very disappointed to learn that when that meeting started today, it progressed into Ms Anyadike-Danes being advised that there were objections being raised from at least two parties to the very fact of Dr MacFaul giving evidence and to his qualifications and experience in the sense of whether they qualified him to be an expert witness.

As you all know, issues have been raised in correspondence which we have circulated about Dr Waney Squier and whether her evidence should be accepted by the inquiry, and that correspondence is ongoing. There has not been a single piece of paper received by me to suggest that there was any reason why Dr MacFaul should not give evidence. Not one. And this morning I understand that issues have been raised about the basis on which he is qualified to give evidence.

I will not tolerate this inquiry being disrupted by last-minute objections about the qualifications of experts. It's utterly unacceptable. And what I'm now

going to do is, between now and lunchtime -- which may be a little bit late -- to ask Dr MacFaul to give evidence in detail about his CV, which you all have, and after we hear that evidence, I'll break for lunch and anyone who wants to make observations or wants to make a submission about Dr MacFaul continuing to give evidence as an expert can do so after his CV evidence has been heard.

So Dr MacFaul, please could I ask you to come forward?

DR RODERICK MACFAUL (called)

Questions from MS ANYADIKE-DANES

MS ANYADIKE-DANES: Good afternoon, Dr MacFaul. Do you have a copy of your CV there with you?

A. Yes, I do.

Q. Thank you. Can I first ask you to confirm when you became a consultant paediatrician?

A. In 1978.

Q. Before then, can you confirm where you had carried out your training?

A. Well, I trained in general paediatrics in military hospitals. I had done my first paediatric post in Leeds, on the academic unit.

Q. Sorry, can I ask you please to keep your voice up a little bit?

A. Sorry. During my senior registrar years, I trained in paediatric neurology in Great Ormond Street and a portion of the time in Guy's.
Q. And that was between 1975 and 1976; would that be right?
A. Yes. But I continued ... As far as I know, I was still up to -- to 1978 ...
Q. Yes. If we go to 311-039-002, which is the second page. Perhaps we'll pull that up. Then you will see:

"1976 to 1978, senior registrar in paediatric neurology, Hospital For Sick Children, Great Ormond Street."

So after your registrar year, you carried on for a further period until 1978.
A. Yes. Half of my time in the military hospital in Aldershot and half of my time in Great Ormond Street.
Q. Thank you. If we go over the page to 003, we don't see it there, we see at the top where it has "1978, consultant paediatrician". When did you stop full-time clinical work?
A. In March 2006.
Q. Thank you. Just so that we have it for a point of reference: from your appointment at Pinderfields General Hospital as a consultant paediatrician, have you done all your clinical work at that hospital?
A. Yes.

Q. And can you help us with the size of that hospital? Perhaps when you first were appointed and perhaps up until you retired in 2006.

A. When I was first appointed, we had something like 55 children's beds. I also had supervision of children in the local hospital for children with severe learning disability -- at the time, called "mental handicap".

Q. What size hospital was that?

A. The mental handicap had long-term residential care for about 30 children.

Q. Thank you.

A. And Pinderfields Hospital also had a burns unit on which there were children's beds and we had a paediatric intensive care bed allocated in the intensive care unit because the hospital housed the regional neurology and neurosurgical unit.

Q. I was going to ask you about that.

THE CHAIRMAN: Sorry, just slow down for a moment.

MS ANYADIKE-DANES: Is it possible for the microphone to be brought slightly closer? Sorry.

Just to recap, in addition to the number of beds in the children's department proper, if I can put it that way, there were also children's beds in specialist units that you and your paediatric colleagues would be responsible for?

A. Yes.
Q. And you also had, outside of that, a responsibility for the beds in the mental hospital for children?
A. Mental handicap hospital.
Q. Thank you. I wonder if you can help explain how you came to take up your position as a consultant in Pinderfields General Hospital.
A. I was interested in paediatric neurology. I did not want to do it full-time. There were very few paediatric neurology posts anticipated in the country when I was due to finish my training and I was steered towards Pinderfields Hospital by my chief at Great Ormond Street, Dr John Wilson, who told me that this post would be very suitable because they were looking for a consultant paediatrician with an interest in neurology to support the regional service for neurosurgery and neurology, which was housed there.
Q. Can you explain a little bit about the service in neurology that was actually being delivered by Pinderfields Hospital at the time you were appointed?
A. We provided a regional neurosurgical unit and, in Yorkshire, there was also neurosurgery provided in Leeds. Because we had a neurosurgeon interested in children, something like two-thirds of children's neurosurgery was carried out in Pinderfields Hospital

and the post-operative care of those children was jointly shared by me and the neurosurgeons.

Because acute coma of unknown sort was transferred to neurosurgery, we also took in children with acute coma from the southern part of the Yorkshire region, so we included there Bradford, Huddersfield, Halifax, Pontefract, Dewsbury and some patient from York, and some from South Leeds. So we had a system whereby we would be delivered children with acute coma, some of whom had head injury and some of whom had acute encephalopathy, and I was involved in the care of the children with acute encephalopathy.

Q. Were you involved in the care of children with neurological presentations more broadly than the acute encephalopathy?

A. When paediatricians in the region encountered complication of meningitis, which was normal general paediatric care, and if they had extradural collections or had developed hydrocephalus, they would come into the hospital in my care and I would work with the neurosurgeons in their management. Similarly, if children had a subdural haematoma from, say, child abuse and needed care, they would come in under my care and we would share with the neurosurgeons.

In addition, because I was known to have

a paediatric neurology interest and at that time there was no paediatric neurologist full-time in Leeds, I had referral practice from consultant paediatricians in the region for outpatient consultations on acute complex problems such as degenerative disease, in which I had a special interest, and sometimes complex epilepsy. And that continued until we were able, in the late 80s, to appoint a full-time paediatric neurologist in Leeds, and then, quite properly, that work drifted towards Leeds, and at that time I suppose about 1 in 10 of all my new referrals when I was doing that work would be from other consultants.

Q. When that specialist neurological work went to Leeds, so your hospital continued to provide general paediatric service to the area --

A. Yes.

Q. -- did you deal with any neurological presentations in those patients who came in the normal way as general paediatric patients to the hospital?

A. Yes.

Q. So you didn't immediately transfer them to Leeds?

A. No. If a child had an acute coma, we would manage the child in our own unit, including intensive care, up to about 1994. And in 1994, there was a general trend towards centralising children in paediatric intensive

care units. But until that time, we managed them in our own unit because we had, until the neurosurgeons left, the ability to do intracranial pressure monitoring. And after they left, we could still do continuing EEG, for example, on a patient on the ward all the time, cerebral function monitoring. And we were able to get bedside EEGs as well as sending the child to the department, and we had good access to scanning, which was supported, not as many district general hospitals have, by a general radiologist; we had two or three neuroradiologists reading our scans.

THE CHAIRMAN: Just to get it clear, is this post 1994?

A. Yes, just for a couple of years, then we continued to have visiting neuroradiology until I retired.

MS ANYADIKE-DANES: So if I understand you correctly, until a certain period, even after the specialist service had been transferred to Leeds, you continued to manage children who came to the hospital who then had some neurological complication, if I can put it that way, or aspect of their condition, up until the point of intensive care?

A. Yes. If they required ventilation, the general trend was to move children to the paediatric intensive care unit in Leeds and so, up to that point, we would care for them.

Q. And were you caring for children like that in 1996?
A. Yes. And until 2006.
Q. You have told us what the size of the beds were for children when you started in 1978; what was it in 2006?
A. We had 43 children's beds with, I think, probably two or three burns unit beds.
Q. Thank you.
THE CHAIRMAN: Sorry, just pause for a moment.
Doctor, post 1994, as I understand it, then, you cared for children who had various neurological conditions until they needed ventilation, and at that point they were moved to the PICU in Leeds?
A. Yes.
THE CHAIRMAN: Does that mean that during those years, you would, from time to time, have discussed with the specialist service in Leeds the condition of various children, whether it was timely now to send them to Leeds for PICU or whether they stayed with you for a while longer?
A. That would be a matter of individual choice because there were some children where we would need to ventilate because they were clearly having problems with breathing, but there would be others where elective ventilation was obviously the choice.
THE CHAIRMAN: Yes, but in order for those choices to be

made, did you have to liaise with Leeds?
A. Yes.
THE CHAIRMAN: I mean, you couldn't just put a child in an ambulance and the child arrives in Leeds a short time later?
A. No.
THE CHAIRMAN: This leads to discussions with the PICU unit in Leeds; is that right?
A. Yes, and there was a time, ultimately reached, when all children who needed ventilating would be retrieved by the regional paediatric intensive care unit. In other words, an ambulance would come with a team and take them away.
THE CHAIRMAN: Right.
A. But that wasn't always the case until about 2002. We occasionally had to do the intubation and ventilation in our own hospital and then I would accompany -- or my colleagues, if they were on call -- we'd take the child to Leeds in the ambulance.
MS ANYADIKE-DANES: So you'd effectively stabilise them if there was going to be that sort of transfer?
A. Yes.
Q. You would stabilise them and, if you were on call, you would accompany them to Leeds?
A. Yes.

Q. And, as the chairman had said, that whole decision to do that and when that would happen and in what circumstance was a matter of discussion between, if it were you on call, you and your intensive care colleagues and perhaps the specialist neurological colleagues in Leeds?
A. Usually the intensive care. Usually we didn't involve the paediatric neurologists at that point.
Q. So it'd be the intensive care. Just so that we have it correctly, a child who had come in like Claire is a child that could have come into your hospital any time from when you took on your consultant appointment up until you retired?
A. Yes, and that was the sort of child that received what we call level 1 intensive care, which is otherwise labelled high-dependency care. And because many children on children's wards are in that category and realising that they needed some focus on their needs, I set up, when I was in the Department of Health, a working party, which produced the Department of Health report on level 1 intensive care in district general hospitals and I chaired the working party and we reported in 2001.
Q. I was going to come then to your research interests and work.
THE CHAIRMAN: Before you do, could we get an idea of how

many hours or shifts Dr MacFaul worked?

MS ANYADIKE-DANES: Thank you very much indeed.

Before we do that, just one question. How many other consultants were working with you as consultant paediatricians in Pinderfields when you went there as a consultant and in 1996?

A. Well, unfortunately, when I went there as a consultant there were only two, which meant that there was me and another colleague. That was quite hard work, but it was the nature of things. As we went on, we appointed more consultants and I think, in about 1994, we had four. And when I was seconded to the Department of Health, they kindly gave us money to backfill, so my time spent in London was backfilled by the addition to our team of another consultant full-time.

Q. So you didn't lose out on consultancy cover, you had that for when you went to London?

A. Yes. But then after that, we worked on a one-in-four rota and that continued until 2006 when I retired.

THE CHAIRMAN: When you say "after that", do you mean from 1994?

A. Yes.

THE CHAIRMAN: Okay. Would you explain, please, the rota that you were working on from 1994? "One in four" -- maybe you need to spell it out for me.

A. Right. That would mean that over a month, one in four nights would be spent on call, taking all patients. But the way that it worked out was anchored to the weekends. The weekend would start for me and for my other colleagues at 9 am on a Friday. For my other colleagues it would finish at 9 am on a Monday. But for me, because I was always on call on a Monday as well, my weekend would be one in four, starting 9 am Friday morning and finishing 9 am Tuesday morning, after which I would carry out a ward round.

MS ANYADIKE-DANES: So one week in every four, that's what you and your colleagues did? Well, you did the extra one, you ran into the Monday up to Tuesday. Can I ask you about that? Leaving aside the one week in every four, every week what was your commitment in terms of work in the hospital?

A. There would be usually one or two nights on call. It depended then on my Department of Health commitments.

Q. But a typical week?

A. I'd always be on on a Monday, without exception. And it was part of our practice at that time to remain in the hospital until 10.30 at night. Each of our consultants did that so that we could do a ward round at about 8 or 9 pm and then a walk round the ward before leaving. Because we brought that arrangement in from around 1993,

the consultants in the team agreed to do it on the basis they could have one day off a week. And I had one full day off a week and I worked in London on that day.

So in a routine week I would do the Monday, and then mostly I would do another day, and it was usually Thursday.

Q. Pause there. So you would do the Monday. You said that because you worked on to 10.30 at night, you would do an evening ward round and a walk around before you actually came off duty.

A. Yes.

Q. Did you do morning ward rounds as well in your hospital?

A. Yes, we'd do them every morning when you're on call, and after on call, we would do a ward round on the Tuesday morning, but from time to time I was having to go to London at around 9 o'clock on a Tuesday morning, and that is why we had the additional consultant appointed and I would hand over.

Q. If you weren't required to go to London on the Tuesday, would your normal week be you would start off at 9 o'clock on the Monday, you would do a Monday morning ward round, you would then do an evening walk round and a walk through the children's department --

A. Yes.

Q. -- and then you would leave at about 10.30 and you would

come back and do the Tuesday ward round?

A. Yes.

Q. Is that correct?

A. Yes.

Q. Then you said you went to London. So if you weren't going to London on Tuesday, does that mean you might go to London on the Wednesday?

A. Yes.

Q. And then I think you said that there would be another day when you would be in the hospital and that was typically a Thursday.

A. And a Friday.

Q. And a Friday. So the Thursday, would it be the same, that you would come in at 9 o'clock, do your morning ward round, stay there until 10.30, do an evening walk round, literally a walk through, if I can put it that way, just before you left.

THE CHAIRMAN: So you did a ward round at about 8 or 9 and a walk round before you left?

A. Yes.

MS ANYADIKE-DANES: Ward and walk, yes. Would you then come back on Friday and do a Friday ward round?

A. Well, we started on call on a Friday. If I was not on call on the Thursday night, then the consultant who'd been on on the Thursday would do the ward round.

Q. I understand. So that coming back on the Friday was only if that was going to be your one in four?
A. I would be back in the hospital all day and I would do a clinic in the afternoon.
Q. I understand. So if you were then going to do your one in four, you would go on and literally carry on until the Monday and go into your normal Monday routine, finishing up if you weren't going to be in London on the Tuesday morning?
A. Yes.
Q. Thank you.
THE CHAIRMAN: So for those of us outside the Health Service, is that a full-time contract?
A. Well, that was one of the problems about working in London. I think that it would be fair to say that it was busy, but that was the nature of the work.
THE CHAIRMAN: Okay.
MS ANYADIKE-DANES: Apart from that work, did you also have research work that you carried out?
A. Yes.
Q. Can you tell us about that with particular relationship to paediatric neurology?
A. The focus on my research portfolio following my senior registrar years was not very great other than in the management of bacterial meningitis. In bacterial

meningitis, the research work I was involved in was that when I was in the college I realised we needed to set up a study on meningococcal disease, which includes meningitis, and I applied for a grant from the Meningitis Research Foundation together with David Baum, who was the director of the research unit in the college, which was a successful application, to do a study of all deaths in children from meningococcal meningitis, and having got the grant I did not remain in touch with the actual research project because we handed it out, we contracted it to St Mary's in London, who had a specific interest in meningococcal disease. Having set it up, I then observed what went on.

Q. Did you say roughly when that happened?

A. That was around ... It must have been around 1993/94.

Q. If one looks at your CV at 311-039-003, one sees that in addition to your other administrative duties, you were a member, from 1981 to 1991, of the Yorkshire Regional Health Authority Neurological Services Working Group.

A. Yes.

Q. Did any of that drive your research at all? Did it lead to research work?

A. No. It was mainly to do with the need to get paediatric neurology set up within the region and to ensure that children receiving neurosurgical care were getting it

properly.

Q. And so what exactly did that membership entail?

A. It meant going to a meeting about two or three times a year. It set up a special working group which was myself and the other part-time general paediatrician with neurological interest to try to make a case for the establishment of a paediatric neurology consultant post in Leeds, which was successful, and I think, towards the end of the 1980s, that appointment started, if not a bit earlier.

Q. Did you have any interest in the British Paediatric Neurology Association?

A. Yes, I was a member from around 1977, I think, 1978, and I was co-opted to their executive committee when I ran their Annual General Meeting in -- we had one meeting a year, scientific meeting, and I convened that in Leeds. I can't remember the exact date. It was probably around 1989.

Q. Then I was really asking you about your research. So that's how it started, I think, so far as you recall. But had you had earlier publications in issues to do with paediatric neurology before you actually had funded research? I'm thinking perhaps in particular at 005 of your report.

A. Yes.

Q. If I take people to it, if you see the fourth one down, "Neurological abnormalities in patients". That was published in the Archives of Diseases as early as 1978. If we move further down, we see you with Green, "The duration of admission for febrile convulsions". That's 1985.

A. Yes.

Q. And then if we -- I'm not quite sure what that last one is, syringomyelia?

A. Syringomyelia, yes. It's a neurological/neurosurgical problem in children.

Q. Which was published in the British Journal of Neurosurgery right down at the bottom there?

A. Yes.

Q. Then if one goes to 006, continuing on, you see, I think it's four up from the bottom in 1999, "Determining common presenting problems to paediatric Accident & Emergency". Did that have a neurological element to it?

A. Well, the purpose of that study was a study which Sir David Hull and I were joint grant holders for. He was based in Nottingham and it was a grant from Children Nationwide, partly supported by the Meningitis Research Foundation. What we were trying to do was to look at the care of children who presented not with a diagnosis, but with a problem. And included in those,

the commonest problem presenting is breathing difficulty. The next is feverish illness, the next is diarrhoea and vomiting, and the next is seizure. So we did a literature review on the management of these children and produced guidelines and these publications come from the Nottingham collaboration. As I say, I was the joint grant holder for that.

That interest then continued because, at the time, we were trying to look at structured methods of assessing acutely-ill children, and it was called "Recognising acute illness in children", and I chaired the group and was the grant holder for that in the college research unit to see if we could improve a recognition of acutely-ill children.

That led to a number of publications which have been lately produced, and towards the end of my career, we had been involved, until last year, in a collaboration from Pinderfields and from the Oxford University funding from NHS to look at this further, how do you recognise acutely-ill children. That included, of course, coma and meningitis.

Q. Sorry, just so that we're clear, is it how you recognise them? Does it also move into how you treat them?

A. Well, the intention there was to try to work out a method of identifying the children and the work that

came from Nottingham was the initial management, which would lead to a diagnosis. Once a diagnosis was established, we felt that it was better for clinicians then to refer to the management of that specific condition.

Q. Okay. So it was a guide on how to recognise the underlying condition of the child who presents?

A. Yes.

Q. So if we translate it just briefly into a child like Claire, it would be to provide some assistance on how to recognise what was happening as she presented in the way that she did on 21 October?

A. Yes. Now, the College produced a guideline on management of reduced conscious level in children, and I've referred to that in my report, and have made some criticisms of it. That guideline was produced because the Reye's Syndrome Association or Foundation funded it, and they funded it following a working party which was held over two days, and I was the facilitator for one of the three or four sessions on that working party, which led to the work which has produced the guideline. But I was not involved in the guideline. The author of it, Richard Bowker, was in our team producing guidelines and is the co-author of the book which I have published on -- together with colleagues -- how to produce an

evidence-based guideline.

THE CHAIRMAN: Doctor, just pause for a moment. The paper which is highlighted on the screen in front of you, that's published in Paediatrics Today in 1999; is that correct?

A. Yes.

THE CHAIRMAN: If it ends up being published in 1999, how much work has gone into it before that? Is it something that is done over a few months in 1999?

A. Oh no. That was the result of the studies we set up in Nottingham Accident & Emergency department, myself and Sir David Hull, and I think we started that in 1994/95. So it does take time obviously to produce the data gathering, which enables the study to reach a publication.

THE CHAIRMAN: Then on the next page, if we go on to page 007 for a moment, is it the fifth entry, you have a further publication in 2004:

"Armon, Stephenson, MacFaul: The impact of presenting problem-based guidelines."

Is that a follow-up, does that lead on from your dissatisfaction with the guidelines that you were just referring to?

A. Yes, because many people issue guidelines, but they don't study (a) whether they are put into place or (b),

if they are put into place, whether they have any effect. We expected, for instance, in diarrhoea and vomiting that producing a guideline, we would reduce hospital admissions with gastro-enteritis. It didn't work out that way; they went up. But at least the guidelines was adhered to.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Just above that, you have "An evidence and consensus based guideline for the management of a child after a seizure" --

A. Yes.

Q. -- and that's 2003.

A. Yes.

Q. Just prior to that, if we go very briefly back to 006, you had talked about a series of papers that came out of that work that you were describing. Would I be right in saying that the last two papers on that page where you were with Eccleston and also with Armon, that those papers to "... test the inter-rater reliability of interview data on parental Accident & Emergency attendance" and also the "What are the common medical presenting problems to Accident and Emergency?". They were all papers that came out of that same piece of research or project, if I can put it that way.

A. Yes.

Q. Then if we go to the page where the chairman had taken you to, which is 007, I think perhaps in addition to that, just above that, there's a further one with Armon on "An evidence and consensus based guideline for acute diarrhoea management"; is that the one that you were referring to?

A. Yes.

Q. So you'd done some of work on that. Then I think Armon, Hemmingway, the chairman took you to, but there's another one a little further on down, the last three on that page, with Lakhanpaul, all three with him. The first one of those three dealing with "Children presenting with acute breathing difficulty", and then the "Risk score to stratify children with suspected serious bacterial infection". It came out in 2011 and there is another one also dealing with serious bacterial illness. Is that all part of the same project that you're talking about?

A. Not entirely. The last two papers came out from the college research working group recognising acute illness in children, which I chaired. That was funded with a different funding stream, partly from the Department of Health, partly from Children Nationwide, and partly from the Meningitis Research Foundation.

Q. But within it, it had a neurological component to deal

with that?

A. Yes, because we're looking for acute bacterial illness of the brain as well as other things.

Q. Exactly. If I could then take you to 008. If one looks towards the bottom of that page, the "Paediatric service and standards reviews". That first one:

"Paediatric member of regional inquiry into paediatric services (Grantham) and baby deaths."

Which is Allitt. Is that the precursor to the Clothier --

A. It is. That was a regional inquiry and then there was a national inquiry set up. The regional inquiry was chaired by a retired, I think, district general manager. It had a children's nurse and it had me as the paediatrician. There were three of us. We were assisted by a barrister.

Q. So you were a tribunal member for that?

A. Yes.

Q. If we work our way down to the "Review of paediatric services in the London region". Can you explain what that review was? When did it take place?

A. That took place in around 1994, 1993, something like that. It was a review of all paediatric services in the London hospitals. It including neurosurgery, cardiac surgery and all tertiary specialties, that is the

specialties largely housed in regional centres outside London. They were dispersed amongst a number of teaching hospitals in London. We reviewed the services available for each of those and made recommendations. Sir David Hull is the chair and I was the secretary, if you like, in the sense of putting together the work and --

Q. Sorry, what was the purpose of that?

A. The purpose of it was to try and rationalise services and avoid duplication and to try to bring, for example, neurosurgical services for children on to sites which had paediatrics, because in London there were some neurosurgical services which were looking after quite ill children where there was no paediatric department on site. The implementation of it, because it caused obviously quite a lot of difficulty amongst the hospitals, has been very slow.

Q. Yes. And then we see the penultimate one on that list:

"Review of children's neurosurgical services for South of Thames."

Are you able to explain what that review --

A. That preceded the London review and I was asked -- I think when I was honorary secretary of the British Paediatric Association -- to conduct that review. Again, because of the problem of lack of collocation of

neurosurgery with paediatrics.

Q. Thank you. At the bottom of that page, you'll see that you start with your current research activities. And you deal with those more extensively over the page at 009 if we go to that.

For example, if you were to help us with the "Development of acute illness severity scale for use in acute general paediatric practice". First, it says it's funded by "WellChild funding"; is that right?

A. Largely so. I had some sources of funding from my Department of Health budget and we were also helped by Meningitis Research funding. That's the one that I've referred to already as "Recognising acute illness in children".

Q. So this is to try and have some common scale by which you can try and measure where the child is?

A. Yes. It is trying to disentangle the clinicians' subjective assessment of how ill a child is and trying to identify if there are elements of that subjective overall impression which we can structure and thereby provide a method of teaching people how to recognise acute illness. It hasn't worked, I have to say, and the most reliable is the overall assessment.

Q. But in any event, the study was to look at that and to see whether that was possible?

A. Yes.
Q. And in the course of that, presumably, you spent quite some time considering neurological problems in children?
A. Oh yes. They were included, yes. And we've tried to do the statistical analysis of presentation in coma.
Q. And can I ask who you worked with in carrying out that research?
A. Yes, with Professor Michael Levin from St Mary's.
Q. And his discipline?
A. He's a consultant paediatrician, but his special interest is in meningococcal disease and meningitis and intensive care.
Q. Yes.
THE CHAIRMAN: Is that more acute than, for instance, low readings on the Glasgow Coma Scale or are neurological observations part of that?
A. We used the other scale for this purpose, which was the AVPU scale -- it's simpler -- which is the "Alert, voice, only response to voice, only response to pain, or unresponsive". The AVPU scale. We used that one.
THE CHAIRMAN: So that's an alternative to the Glasgow Coma Scale?
A. It's simpler.
THE CHAIRMAN: Right.
MS ANYADIKE-DANES: In the course of doing that work, does

that mean that you considered the advantages and disadvantages of scales such as the Glasgow Coma Scale?

A. Yes.

Q. Then if one looks just at "Critical care of children" -- firstly, I should say, that because you have referred to your work as "the department", is that what we see immediately before that?

"Medical adviser in paediatrics and child health -- Department of Health, England, 1996 to 2003."

A. Yes.

Q. And all these matters that you have indicated below -- "screening", "critical care for children", then over the page, "medicines for children", "acute care for children", "child health", "evidence-based health" -- that all came within that appointment?

A. Yes.

Q. Did you work with other clinicians in providing that advice to the department?

A. Yes.

Q. Which other clinicians did you primarily work with?

A. Well, there was the -- the Department of Health convened a paediatric intensive care working party and I sat on it, so there was a range of paediatric intensivists on that, some from an anaesthetic background, some from paediatric. I don't remember the names, but they're

published in the publications. When I convened the working group on high-dependency care in children, level 1 intensive care, which I chaired, I worked with paediatric intensivists.

Q. That's that third bullet under:

"The critical care of children, Department of Health. Set up and chaired Department of Health working party, setting standards for high dependency care."

A. That's correct.

Q. If we go over the page, just to give an illustration of the sort of thing, under "Medicines for children", we see at the fourth bullet:

"Brokered the successful proposal to provide a BNF for children."

So that was part of your work to end up with a British National Formulary that was targeted towards paediatrics?

A. Yes, it wasn't an easy negotiation for reasons I can go into about the issue of unlicensed medicine, if you want me to. But it was necessary to get legal approval as well as meeting what was called the Medicines Control Agency's senior staff at that time, now the MHRA. It was produced because NICE didn't want to do it and the college production, which was called "Medicines for Children", needed to be updated and there was no funding

to do it, and it seemed the logical way was to go to the BNF. I was able to get the support of the Chief Medical Officer and the legal department of the Department of Health and eventually the MHRA/Medicines Control Agency, to support that. And the only thing that was outstanding when I left was the funding. That's been resolved.

Q. So that's going ahead?

A. It has gone ahead.

THE CHAIRMAN: Doctor, in all of this additional work you've done, how important has it been for you to continue to be a consultant paediatrician and practice?

A. It's essential. I remember many times people have said to me, "What are you doing up here? When did you last see a patient?", and my point was that I didn't think you could do it properly without. I remember one particular day when I was asked in London by somebody, who said, "When did you last -- and I said, "I did a lumbar puncture this morning, before I came to London, I've just been telephoned the result".

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Then just on that page, under the "Acute Care for Children", that final bullet -- this is, I think, part of what you might have been taking to the children before. No, the final bullet on the page:

"In 2000, originated the project and commissioned the Department of Health DVD training materials, 'Spotting the sick child', for recognition of acutely-ill children."

Is that part of the work you were talking about.

A. Yes, I thought it was a good idea to use video because it is much easier to use video to convey to juniors how ill a child is. I took that a bit further by trying to do it in telemedicine, but the purpose of it was to ask for -- I specified and arranged the funding to get this done, and it was done for me on behalf of the Department of Health by the London Hospital, a consultant in Accident & Emergency did that and it was very popular. Then I arranged funding for the next version of it, which is out now, and I think it has been generally well received.

Q. Thank you. If we go over the page --

THE CHAIRMAN: Sorry, before you do, could you go two above that:

"Carried out a study of out-of-hours needs of children for the 'Hospital at Night' project showing that paediatrics needed a different solution from adult services."

If there's a way to encapsulate that briefly, what did that involve?

A. The purpose of it was -- because it had been suggested that a hospital could be covered, because of a shortage of junior doctors, by a team which would cover all specialties. I didn't feel that was right for paediatrics. So in order to study how often paediatric staff would be recalled for crises in the night, we got diaries filled in by three or four district general hospitals. The funding came from the Department of Health, but we were able to recruit these hospitals to do it. We identified that collapses or crises or children deteriorating rapidly occurred throughout the 24 hours and required specific paediatric input. And on the basis of that, paediatrics was excluded from the Hospital at Night team concept and paediatric teams were left to remain responsible.

THE CHAIRMAN: We've heard quite a bit of evidence over the last few weeks that while children can bounce back very quickly from being ill, they can also plummet very quickly --

A. And unexpectedly so.

THE CHAIRMAN: Is that part of what that study was looking at?

A. Yes. The point is that children -- there is a notion that you can identify people in hospital who are likely to, in the terms that paediatricians use, "go off".

That's not always possible and so this was a study to quantify the number of recalls by registrars out of hours and when they occurred out of hours. They do occur in the early hours as well as in the evening when the hospitals tend to be better staffed.

THE CHAIRMAN: Sorry, this is house officers recalling registrars or registrars recalling consultants?

A. We didn't look at the consultant recall. I may have the data.

THE CHAIRMAN: But it's house officers recalling registrars?

A. It's the call of registrars and -- it's the call of the middle grades. In some hospitals that's a senior SHO, in others it's a staff grade or a registrar.

THE CHAIRMAN: Thank you.

A. I may have the data on consultant recall. I'd have to go back.

MS ANYADIKE-DANES: Thank you. Then if we go over the page, 011, we see just on that second bullet, just above "child health":

"Identified the need and provided the argument in 2002 on the requirements for nurses with enhanced skills in general, neonatal and community paediatric practice."

Perhaps you can help us with that a bit and explain the extent to which, in the course of your research, you've actually had a look at the services that are

required for nurses in terms of acute paediatric care.

A. There was a concern for many years that some nurses working on a children's ward did not have a children's nurse qualification. That has been overcome, thank goodness. As far as the advanced skills are concerned, it had become relatively common practice to have advanced nurse practitioners working in neonatal intensive care units and there was a notion that perhaps we could build up a team of nurses who could do paediatric intensive care -- in other words, not neonatal intensive care but paediatric intensive care -- and thereby provide better support for paediatric intensive care units throughout the 24 hours. That would require gathering a cohort and training advance nurse practitioners. It has been much slower to implement than advanced nurse practitioners in neonatal care.

Q. In the course of doing that, did you carry out research into --

A. No.

Q. So you were just working on the study to see the feasibility of providing that enhanced service?

A. Well, to try and negotiate it because there was a certain opposition from the Department of Health nursing branch because they could see that the pool of nurses

generally was being tapped off by all sorts of people wanting to have advanced nurse practitioners. I mean adult medicine, as well as children.

Q. Have you done any other work on the nursing side?

A. No, I don't think so.

Q. Thank you. I think earlier when you first described your coming to Pinderfields, you said, when you came as a consultant, you also worked in the hospital for mentally-disabled children, or with mentally-disabled children.

A. Yes.

Q. I was just looking under your heading "Child health", and you say about seven bullets down:

"Provided the advice and information to set the 'Quality Projects' programme for the care of disabled children."

What did that involve?

A. Well, at that time, the Quality Projects agenda was initiated by the social care branch of the Department of Health and the work was to try to make sure that the social care services in a locality were aware of the numbers of children with severe disability and how severe it was in order for them to provide their services. That was the purpose of that work.

Q. Thank you. Then just over the page, finally, 012. This

is "Evidence based care":

"Initiated the existing children's programme for topics for NICE: asthma inhalers, growth hormone, urinary tract infection, head injury, and feverish illness in children."

Just very briefly, can you help as to what that involved?

A. Yes, at the time that I was working in the department there were no children's projects in the pipeline for NICE guidelines, so it was necessary to identify a shortlist. At that time, we had to negotiate from the Department of Health point of view against other competing demands from other branches in the department such as areas of adult practice. So in consultation with my colleagues in the College, I gained support for the notion of going for asthma inhalers, growth hormone as well because there was concern it was overused or underused, and urinary tract infection because there was a wide consensus about how you managed those.

Because of the concern about how head injury was managed particularly in relation to whether you should scan them in Accident & Emergency, I felt, having read the literature on this, there was a lot of work in the States showing that the easiest way to deal with that was to scan them rather than do a skull X-ray and

observe. Observations included GCS, Glasgow Coma Scale. It was better and safer to scan. So the result of that was to, when the specialists worked on it, come up with recommendations for scanning head injury rather than skull X-ray and observation.

The feverish illness in children was a difficult topic because NICE, up until then, had wanted to look at diagnoses or particular therapies; they were not minded to look at a presenting problems. But from our studies that we had done, we could see that feverish illness was the second commonest presentation and I felt it was necessary to produce a guideline on that because the Americans had done a very good one. And NICE accepted it eventually, but it required about 18 months of negotiation, and I was hoping that I would be able to do it in Nottingham, but I ran out of funds. It had already cost us 350,000 to produce the breathing difficulty, the diarrhoea, and the seizure, and we had no more money for the feverish illness. So I thought the best way was to ask NICE to do it, and they did it, nicely.

Q. Thank you. Just to round up to 2008, your work with the Department of Health continued and we see what you were doing over the period 2004 to 2007. That included children's prescribing. Was that a development on from

your BNF project?

A. Yes, it was to do with ... I was asked to set up a working group and provide guidance on improving the quality of prescribing in children with a view to providing the specification for electronic prescribing.

Q. And then 2007 to 2008 has your national clinical lead child health programme, Connecting for Health. Just before I leave your CV, I want to come to what you did in the hospital in terms of your hospital positions. If one looks at 003, you were clinical director at the Women and Children's Services from 1997 to 1998, and then clinical director of the Children's Services from 1998 to 2005, which is just before you retired.

A. Yes.

THE CHAIRMAN: Let's go back one more, one above that. The one above that, doctor, is "Divisional coordinator in the medical division, 1993 to 1996"; what did that involve?

A. That involved providing the lead in the management structure in our own hospital for all non-surgical specialties, which were clinical specialties. So we had a surgical divisional coordinator and we had a medical divisional coordinator, and in that role I worked in general management as the lead clinician for the non-surgical clinical services, which was adult

medicine, neurology, and elderly care.

MS ANYADIKE-DANES: Can you recall --

THE CHAIRMAN: And paediatric?

A. It included paediatrics, yes.

MS ANYADIKE-DANES: Can you recall the size of the hospital at that time in terms of beds?

A. The size of the hospital? I think it was probably about 500. I can't be precise, but it's that sort of size.

Q. And what area did it service in terms of population? Are you aware of that?

A. Well, the hospital had a mixture of some regional services. It had spinal injuries, it had burns, it had neurology, neuroradiology was still there. And that was serving a population of perhaps three-quarters of a million. So far as the burns were concerned, a much larger population. For the general hospital services, it was covering a population of about 250,000 to 300,000. I say "about" because we provided services for South Leeds as well as the Wakefield conurbation and it is very difficult to identify exactly what population you're serving, but it was in that order.

Q. Thank you. Then those two clinical director positions, did one service develop into another?

A. Yes, what happened was that, of course, neonatal care is part of obstetrics, so they thought it was reasonable to

have a women and children's director. That's common in district hospitals, sometimes led by a gynaecologist, sometimes led by a paediatrician, and I accepted that role to keep the children's services element. But I obviously relied heavily on my gynaecological colleagues to help with the women and children's, because it required things which were common to surgery, such as theatre access.

Then when the children's services became bigger, when we amalgamated with another district hospital in Pontefract, so that we are now covering a bigger population of 300,000 for children within Wakefield alone, and the other hospital had about 25 children's beds, I think, so we were then getting up to something like 70 children's beds with about ten consultants, I felt that it was reasonable and negotiated that we should have a separate clinical services directorate, and I was director of that.

Q. And as I say, you continued on in that until just before you retired entirely?

A. Yes. I handed over a year before because I felt my successor should have a year when I was still around.

MS ANYADIKE-DANES: Thank you. Mr Chairman, I have nothing further.

THE CHAIRMAN: Are there any questions for Dr MacFaul about

his CV before we break for lunch? Ms O'Rourke?

MS O'ROURKE: Sir, is it okay to ask it direct?

THE CHAIRMAN: No, it's not. Ask it through me, please.

MS O'ROURKE: It was to ask about his paediatric neurology appointments as put on his CV. It says:

"1975 to 1976, registrar at Great Ormond Street."

But under the same entry he's, in fact, got three posts: one at Guy's Hospital, one at Northwick Park, and one at Great Ormond Street. It appears only the Great Ormond Street one is paediatric neurology. I was wondering whether that was a rotation as a registrar with four months in each or how it was and how much of it was actually paediatric neurology.

Sir, the second question --

THE CHAIRMAN: The first question, doctor. Can you deal with that?

A. How much of that particular rotation was neurology?

MS O'ROURKE: Was it rotation? Because you're in three different hospitals which are geographically separate, although all in London. So was it four months in each or were you covering all three hospitals in the one rotation?

A. No, I was moving from one hospital to another. So I spent some of my time in Great Ormond Street doing paediatric neurology. I went to Guy's to do

developmental paediatrics and I spent some time at Northwick Park as a paediatric registrar. I think that was probably -- well, must have been three or four months. Three months, I suspect, because somewhere in there I did some cardiology, but it doesn't appear.

MS O'ROURKE: Therefore, the amount of time as a registrar in paediatric neurology may only be three or four months?

A. At that stage, yes.

MS O'ROURKE: And then the next one simply related to the next entry, which is 1976 to 1978. You've got yourself listed as Cambridge Military Hospital in Aldershot and Great Ormond Street. Again, geographically different. I think you said in answer to questions that you were half time at one and half time at the other. What does that mean: does it mean that you did 12 months as a senior reg at Great Ormond Street and then moved to Cambridge or that during the same period you were spending so many days a week at one and so many at the other?

A. It was the latter option. It was over a two-year span and I spent some of my week in Aldershot and some of my week in London and commuted.

MS O'ROURKE: Thank you. I have no other questions.

THE CHAIRMAN: Just to give me a better picture, do I take

it from this then that if in Leeds in, what, the late 1980s there wasn't a paediatric neurologist, that the specialty of paediatric neurology emerged relatively recently?

A. Well, yes.

THE CHAIRMAN: Leeds is obviously a fairly big city.

A. Yes. When I went there, there was a paediatrician, Dr Forsythe, who was a general paediatrician who was interested in epilepsy and he did some of the paediatric neurology referrals, but there wasn't a full-time paediatric neurologist. My particular interest when I moved there was in neurodegenerative diseases and complex neurological conditions as well as other things, neuromuscular disease. So between the two of us, we had a sort of portfolio, but we both felt that the Yorkshire region should have a paediatric neurologist.

THE CHAIRMAN: Sorry, the point I'm getting at is that in the late 1980s the Yorkshire region did not have a paediatric neurologist.

A. That is correct.

THE CHAIRMAN: And was that uncommon in regions through England or was Yorkshire a way behind?

A. I think Yorkshire was a bit behind, to be honest.

THE CHAIRMAN: Since then, since the 1980s, has the discipline of paediatric neurology developed?

A. Yes, it's increased.
THE CHAIRMAN: And become more specialised?
A. I think it has been, yes. I think that it would be fair to say there are now three or possibly -- I'm a bit out of date -- four paediatric neurologists in Leeds, one of whom has an interest in paediatric muscular disease and the other two in epilepsy. So within paediatric neurology, there is a trend now as there are more posts for more specialist care. It wasn't unusual in the 1980s for there to be only one paediatric neurologist in a region.
THE CHAIRMAN: Well, would a young paediatrician now in his or her 20s or 30s know as much about neurology as you would have learnt in your 20s and 30s?
A. I think that's difficult because the more people you have seeing the same pool of patients, the less individually experienced you become, and therefore -- in other words, the numbers of cases remain the same, but you are diluting by increasing the number of consultants. There are solutions to that, which don't necessarily involve direct patient care. It's communication.
THE CHAIRMAN: Okay. Thank you very much, doctor.
We'll take a break now and we'll sit again at 2.20. Thank you.

(1.41 pm)

(The Short Adjournment)

(2.20 pm)

(Delay in proceedings)

(2.27 pm)

THE CHAIRMAN: Ladies and gentlemen, unless there's any objection, I'm going to ask Dr MacFaul to start to give his evidence. No? Okay.

Doctor, thank you.

MS ANYADIKE-DANES: Good afternoon.

A. Good afternoon.

Q. What I would first like to do is to have on record the reports that you've provided. You've provided a full governance report, that's right, isn't it --

A. Yes.

Q. -- in which the clinical elements have formed a part, if I can put it that way?

A. Yes.

Q. Or the consideration and the views taken on the clinical issues have formed a part.

A. Yes.

Q. The full governance report is, I believe, 238-002-001. Subsequently, the clinical elements of that and the written material on which that was based were extracted and put into a shorter version, which is a report that

has been circulated more recently for the purposes of what we call the clinical hearing; that's correct, isn't it?

A. I'm not sure exactly what has been reported. I produced a large document, which had an appendix D in it, but that was a large amount of reference material.

Q. Yes.

A. So the main report and appendices A, B and C, as far as I know.

Q. Well, let me take you to what has actually been provided. If we go to 238-002-001. This is your full report, but for which we have taken out the clinical elements and circulated that ahead of time because we were circulating only that which dealt with the clinical matters being discussed in this clinical hearing, if I can put it that way.

So the easiest way to see that --

THE CHAIRMAN: Sorry, you've taken out the governance elements.

MS ANYADIKE-DANES: Sorry. If I got that the wrong way round, I apologise.

(Pause).

Sorry, Mr Chairman, there seems to be a slight error here. There's a technical matter. I wonder if you'd allow me to deal with that to make sure we pull up the

appropriate things with the appropriate redactions because I think, if we go through this, we'll end up looking at the full report the whole time.

THE CHAIRMAN: Well, let's not do that.

(2.32 pm)

(A short break)

(2.37 pm)

THE CHAIRMAN: Are we sorted out?

MS ANYADIKE-DANES: I think we're about to find out, Mr Chairman. I hope we are.

THE CHAIRMAN: We are.

MS ANYADIKE-DANES: There we are.

So if I just orientate you through it, if we go to the next page, 004. That wasn't exactly what I was expecting, but nevertheless, let's deal with that. You can see from what remains, which is not redacted, these are the particular parts that have been retained in this report and circulated for the purposes of dealing with the clinical issues. So you can see the first is your chapter 1, "Summary of illness and subsequent events". And then your headline comments and then chapter 2, "Acute encephalopathy", and the detailed commentary on the clinical care given to Claire. And right down at the bottom, you can see annex A, "Guidance on acute encephalopathy", which was available in 1996.

Please go over the page, 006. Then there's annex B, which is the midazolam prescription, and annex C, which is the detailed clinical chronology and copies of selected clinical records. So that is the clinical aspects, if I can put it that way, of your report.

Subject to something I'm going to ask you in a minute, do you adopt that?

A. I adopt that report, but I have submitted two further amplifications.

Q. Yes, which we're going to come to.

Then your next report is "Supplemental report on the fluid regime used in Claire Roberts". That's dated 3 September of this year. The reference number for that is 238-003-001.

It is a single page and it deals with a very specific point, which related to a particular edition of Forfar & Arneil.

A. Yes.

Q. You were then provided with a report from Professor Young, which dealt further with the issues to do with the relevant texts that were available, which indicated the state of knowledge of treating, through fluid management, neurological presentations in children in 1996.

A. Yes.

Q. If I can pull that up, that's 238-004-001. It is called your response. Just to orientate you, if I pull up witness statement -- this is Professor Young's witness statement, he's a professor of medicine and director of the centre for public health at Queen's. He has put in two witness statements. This is the one that your report responds to in particular. It's witness statement 178/2, and page 2 will show the substance of it.

It goes on and it has attached, or submitted with it, a number of extracts from textbooks and papers and so forth. And you have seen that?

A. Yes.

Q. And that's the report that you are responding to in that last report that I put up for you, which is dated -- I don't have the date immediately, but anyway. It's November of this year.

A. Yes.

Q. Professor Young has also provided a further report, which deals with the Glasgow Coma Scale, and one can see that at 178/3, and page 2 will show its substance. So the comments on the interpretation of the changes in the Glasgow Coma Scale of Claire over that period.

You saw that yesterday; am I right?

A. Yes.

Q. And you therefore have not included any response to that in your report.
A. That is correct.
Q. But are you in a position to be able to address it?
A. Yes.
Q. Thank you. Then if we go back to your most recent report because that is where you deal with the question of the edition of Forfar & Arneil and what the state of knowledge is. You deal with that in substantive terms in your most recent report. So I'm going to ask you to explain matters from there. But do you adopt those three reports, subject to anything that you say now to clarify your position?
A. Yes.
Q. Thank you. Maybe if we can pull up 238-004-001. If it's possible to pull up alongside that -- because we see the start of the matters to which you're responding -- witness statement 178/2 at page 2.

In some respects that encapsulates matters. This extract rather confusingly, given how we've just arranged to have the shortened version in the system, actually comes from your larger report, but the point is reflected in other places in your shortened report, and I think you're familiar with the issue.
A. Yes.

Q. The issue, as one sees it on that page, is that you have cited from the third edition of Forfar & Arneil, which is dated 1984, the treatment there for cerebral oedema requiring to be presumptive, and therefore a restriction of fluids. And if we go over the page, leaving your report up there, to page 3, 178/2 at page 3, the comment there is that:

"At the time of Claire's treatment, that particular edition was 12 years old. In many respects, it was completely out of date and, in any event, had been replaced by the fourth edition of the textbook, which was published in 1992 and then reprinted a number of times, once in 1994 and then in 1996."

Just for completeness, there was a further edition in 2003; is that correct?

A. There was another edition, the fifth edition, in, I think, 2001, but the one that I have referred to is the sixth edition, 2003.

Q. Yes. And just so that we bookend your consideration of that particular text, on the one hand you have the 1984 edition and on the other hand you have the 2003 edition. It's not the 2003 edition that we're going to ask for your views on; it's what you made of the 1984 edition in relation to the edition that was current at the time of Claire's admission. But can I just ask you why you

chose the 2003 edition also to look at?

A. The simple answer to that is that I accept that I should have made greater reference to the fourth edition, and I have said so in my response, and that was a fault on my part. But when constructing the report for you, I had immediately to hand the 1984 edition and I had immediately to hand the sixth edition.

Both of them, in essence, give guidance, which, in principle, in the management of acute encephalopathy in regard to fluids in particular, but in other aspects, were substantially the same. The wording has changed, but they were substantially the same. I therefore, at that point, did not seek the interval edition because I did not see any main difference. The wording had changed and, in particular, the wording in relation to 0.18 per cent saline being contraindicated, and in the mention of hypotonic fluid. And it would have been better for me to have checked the 1992 edition, which I have subsequently acquired. But it was pointed out to me by the team, who had that copy, that the wording had changed and hence I produced that supporting document in September.

But I would say this: the main principles of management in the 1984 edition and in the fifth edition had not changed in essence. I would make another point,

and that is that one of the advantages of referring to the 1984 edition is to establish that the awareness of hyponatraemia and the risk it posed to worsening of cerebral oedema was not of recent concern in late 90s. It had been present for a long time and continued to be present.

The wording in the fourth edition has changed. I don't know why it has changed and I think it is less easy for interpret for a clinician who is having to refer to it quickly. Because instead of that stark and clear warning about that fluid, it chooses to use other terminologies, which I can come to in time.

Q. Thank you. I'm going to ask you a little bit more about the differences between them and whether there is any underlying difference, as you see. One of the things I wanted to see is, in terms of the 2003 edition, which you did have, which is the edition which would have been current when Claire's parents came to the Royal to have an explanation of what had happened to their daughter in 1996?

A. The 2003, sixth edition, to which I have referred.

Q. Is that one of the reasons you were looking at that edition?

A. The main simplistic reason is that I had it to hand.

Q. I wonder if it's possible to put up the two versions,

one from the fourth edition and one from the third edition. I'm trying to see if we actually have those paginated.

Let's first take the point that's made in the first paragraph. In the first paragraph, what Professor Young is saying is at the time of Claire's treatment the 1984 edition was 12 years old, which it was.

A. It was 12 years old, but was in current use until 1992.

Q. Yes, but it had been superseded.

A. Yes.

Q. And the claim that's made is that, in many respects, it was completely out of date and had been replaced. And the bit that you have relied on and reproduced in annex A of your report has been rewritten.

A. It has been rewritten.

Q. Can you explain, firstly, what guidance was being given by the 1984 edition as to how to address neurological presentations or, more specifically, cerebral oedema in relation to the management of fluids?

A. Well, the 1984 edition was highlighting the risks of hypotonic solution and specifically mentions 0.18 solution. The edition also advises anticipatory care as being ideal. In other words, in an acute encephalopathy to envisage that this problem might occur and take steps even before a blood sodium measurement is made, if you

like, to avoid it. And to some extent, although in separate wording, the 2003 sixth edition, reiterates that.

Q. Let's first of all deal with: what is the danger that is anticipated that should be being addressed by the presumptive restriction of fluid?

A. The development of inappropriate ADH secretion is one. The other is the knowledge that, in brain oedema, the use of hypotonic fluid can pose a threat because one of the causes of hyponatraemia which is encountered in acute brain disease is water overload, and that is reiterated in the following edition, the one current in 1996, but not in such specific wording.

And the danger is that the hypotonic solution provides excessive quantities of what is called "free water". That is water not locked, if you like, to the sodium and in a situation where there is inappropriate ADH secretion, which also causes hyponatraemia, you wish to avoid giving a fluid which will contribute to the problem and make it worse.

Q. Can we just be clear about that? The fact that cerebral oedema involves the swelling through fluid of the brain --

A. Yes.

Q. -- and can continue on, if unchecked, to lead to coning

and death --
A. Yes.
Q. -- is that something that was appreciated not just in 1984, but also in 1996?
A. Yes.
Q. Readily?
A. Yes.
Q. Certainly by neurologists?
A. And in general paediatrics because the problem exists in bacterial meningitis and bacterial meningitis is commonly treated by general paediatricians.
Q. So not only would a neurologist certainly know that, but a general paediatrician would also know that particular mechanism for the development of cerebral oedema leading to coning and death?
A. Yes. And it is echoed, the warning, in other texts, which I have referred to in my report and which are used by paediatricians in training.
Q. So if that mechanism was known, I think what you're saying is that a way to deal with that before that became so advanced, if you could foresee that that may be the pathway that the child is on, to act presumptively to restrict the fluids?
A. Well, as I have said in my report, that would be ideal/high quality standards. But I would not expect

a general paediatric unit to have appreciated that risk immediately Claire was admitted. And I can explain on that.

Q. Yes.

A. But if Claire had been admitted with her condition straight to a neurology unit or straight to an intensive care unit, it is more likely that the anticipatory approach would have been adopted. More likely, not certain, but more likely.

Q. Is that true whether you're speaking of 1984 or 1996?

A. Yes.

Q. We'll come in a minute to why you say that with confidence about 1996. But part of your basis for saying it in 1984 is that, apart from any other thing, there was a clear reference in a textbook to that effect.

A. Yes, and it was much more clearly stated in the 1984 edition than it is in the 1996 edition and, in my supplementary report, I have given the text to show, but I have also demonstrated in my comments how the user is having to switch from one part of the textbook to another. There are two elements to that. You either have a textbook, which is used for training, and, if you like, building your knowledge. That is more likely to be, for paediatricians in this country, Forfar & Arneil.

Or you have a textbook, which is used for both, that's training and quick reference, and the Nelson textbook is favoured by many for quick reference because it is much more tightly worded and would be used at the point of care more often, although many use Forfar & Arneil. And I suppose the point that I was trying to establish in referring to the 1984 edition -- and I again reiterate that I regret that I did not consult the edition, for reasons I can explain in a moment, of 1996.

The reason that I think it's important is that that would have been used by Dr Steen, for example, or Dr Webb -- very likely to have been used in their training. Because it was still current until 1992.

Q. So that would be your starting point, to try and understand what people were trained with --

A. Yes.

Q. -- and might have used early in their clinical career?

A. Yes.

Q. And that's 1984?

A. Yes. Although I was not able to put my hands on the 1996 edition, that was the one which we were using obviously in our own department. We would have had them on the wards --

THE CHAIRMAN: Sorry, it's not the 1996 edition, it's the 1992, surely, is it?

A. Well, there was the 1984 edition, which is what we're talking about, Mr Chairman.

THE CHAIRMAN: Sorry, just a moment ago you said:

"... that although I wasn't able to put my hands on the 1996 edition."

A. Yes.

THE CHAIRMAN: Sorry, "the edition which was current in 1996 --"

A. That's what I mean.

THE CHAIRMAN: "-- which was, in fact, the 1992 edition."

A. It was reprinted, but perhaps it would be best to say third and fourth. It was the fourth edition.

THE CHAIRMAN: Shall we get this clear then? This might be the easiest way through it: the third edition is 1984, the fourth edition is 1992, and the sixth edition is 2003.

A. And the current edition in 1996 was the fourth edition.

THE CHAIRMAN: Let's use that terminology as much as we can.

A. The fourth edition did not substantially change the advice given. I accept that that wording has gone and, in my own unit, for instance, we did not change our approach because of anything it stated. The reason that I didn't check it was because I didn't have it to hand and I didn't expect the wording to have been so strikingly changed, but it was.

MS ANYADIKE-DANES: If I can ask you this: the underlying reason, which is to prevent the continued development of cerebral oedema by dealing with fluid management before it got to the point of no return, if I can put it that way, so dealing presumptively with it, had anything happened in the research and between 1984 when that edition first came out and -- well, that's the third edition, and then the fourth edition. Had anything happened to change that underlying relationship between the management of fluid and the slowing down of the development, or maybe halting it altogether, of cerebral oedema?

A. Well, not in practice. But what had changed was a certain amount of information had come from a research study published in 1990, which showed that the management of inappropriate ADH secretion, which generally was done by fluid restriction, could be improved by the addition of additional sodium to the intravenous fluid, not necessarily hypertonic saline, but just increasing the amount of sodium. So it was further support to continuing to adopt a cautious approach in the use of very hypotonic solution and it favoured the use of 0.45 or normal saline. That is an American publication. It is referred to by Dr Kirkham in her review in 2001, and it is referred to in the

paediatric neurology textbook, the Swaiman and the Menkes, which come from the States.

Q. And when did that research start to break through into publications that would be more commonly available?

A. Well, I think it has filtered slowly. I think that the most potent piece of guidance available at the time was to avoid hypotonic fluid, but unfortunately the wording in the fourth edition of Forfar is not so precise and it leaves the user to grapple with maintenance of homoeostasis, avoidance of inappropriate intravenous fluid, and it warns against the fact that inappropriate intravenous fluid therapy can contribute to the problem. It is not user-friendly in that respect. But the principles which lie behind that are maintenance of homoeostasis is correction of any electrolyte disturbance.

Q. Sorry, can I just ask you to pause there and explain what that is? What is homoeostasis is its maintenance?

A. Maintenance of homoeostasis is trying to make sure that the normally physiological status of the child is maintained, and that includes temperature, fluid balance, and keeping the electrolytes within the normal range. It's the keeping of the electrolytes within the normal range which forms a subset of maintaining homoeostasis, and that includes giving higher sodium

content, especially when a low sodium is identified. But the counsel of perfection is to anticipate that in acute brain disease.

Q. So if you're going to try and keep the electrolytes within the normal range, it's not so prescriptive as to how you do that, but that might involve either restricting fluid and/or increasing the sodium content of fluid?

A. The logical pathway is to avoid giving a fluid which donates a lot of free water, and that would include 5 per cent dextrose and fifth-normal saline. 0.45 per cent saline also donates an element of free water. Normal saline doesn't, but it's isotonic. The guidance of maintaining homoeostasis is to anticipate the fact that sodium might drop, to be very careful about fluid administration, but once a low sodium is identified -- that is outside the normal range, which is common in paediatric practice, very common, but in acute brain disease is a red flag warning. That is the point that was relevant to Claire because, although she did not have hyponatraemia by definition, her blood sodium was outside the normal range. There was derangement.

THE CHAIRMAN: Sorry, this is at the point of admission, you mean?

A. Yes. So she was outside the normal range. One of the

responsibilities would be to restore, if you like, homoeostasis, but I am not critical and I have not been critical in my report of the use of fifth-normal saline by the paediatric junior doctors overnight on the night of admission. I have said "high-quality ideal practice", but I'm not thereby saying that what was done was wrong; I think it was not inappropriate for the junior doctors on the evening of admission and overnight to use fifth-normal saline, but the matter changed the following day.

MS ANYADIKE-DANES: And by the following day, what do you say should have happened in relation to what was knowledge in 1996?

A. Well, the difference is that when Claire came in, her conscious level was disturbed, but it was not absolutely clear that this was going to be persistent or get worse. So it would be reasonable to adopt an observation period of time to see what the trajectory of the illness was.

By the following day, she had experienced reduction of conscious level for a sufficient period to be in an acute encephalopathy framework. At that point, it was necessary to be particularly careful about the fluid balance and particularly careful about monitoring it. So I believe a blood sodium should have been done in the morning of the 22nd.

Q. And that is something that you believe that, given the state of knowledge at the time, a general paediatrician could have worked out that a blood sodium was actually required for Claire in the morning of the 22nd?

A. Yes, for two reasons --

THE CHAIRMAN: I think this bit is easy because I don't think there's any resistance to the proposition that there should have been that test done on the Tuesday morning; isn't that right? Dr Sands, Dr Stevenson and people who were involved on Tuesday morning accept that there should have been a blood test done on Tuesday morning.

MR GREEN: Yes, very simply.

THE CHAIRMAN: And I think Dr Steen's position would be the same, Mr Fortune?

MR FORTUNE: Yes, it would, sir.

THE CHAIRMAN: Okay. So there's no dispute about that point.

MS ANYADIKE-DANES: If that had been done -- and this now takes you into the heart of the matter in terms of what might have been the response to it -- and it showed that it continued to be outside the normal range, as you have put it, so perhaps was lower than the 132 it had been from the bloods taken, say, about 9.30 on the evening of the 21st, then we get into the issue of the state of

knowledge as to what should be the response at that stage.

A. Yes. Well, at that stage it is, on balance of probability, but conjecture, that the blood sodium would have been more deranged and the response would have been twofold: one to consider why, and the other to take an action. And the causation of such an observation would be either water overload or syndrome of inappropriate ADH secretion or a combination of the two. And the action would be to stop giving fluid which donates excessive water, and that is to change from the fifth-normal saline to at least 0.45. And the other would be to reduce the fluid intake, that is fluid restriction. Both of those were advised in 1984 in the third edition and advised in the fourth edition and were current in 1996 in general paediatrics and in paediatric neurology texts.

Q. So as you're saying, it would have been common knowledge for paediatricians and certainly for neurologists to respond to a continuing low out-of-range serum sodium level by restricting the amount of free water and increasing perhaps the sodium content of any fluids being given?

A. Yes. The one point I would make, because it's relevant, is that we often see low sodium, in paediatric practice,

in children who come in with a range of acute illnesses, including respiratory, and I have given in my report some details from my own hospital to show the frequency. On the whole, it doesn't need managing. The one that does need managing is where you have an acute brain disease because of its potential very serious complication of cerebral oedema. So the situation is specific to the management of acute encephalopathy. And by saying that, I'm addressing some of the comments which Professor Young has made.

Q. Yes. So if we're clear, it wouldn't just be because she had another low out-of-range serum sodium result, it would be she had that and was presenting with some sort of neurological condition?

A. Yes, because if, for example, she was alert but dehydrated because of a vomiting illness, and you found a low sodium, all you would do would be to increase the sodium content of the intravenous fluid; you wouldn't fluid restrict.

Q. Yes. And the fluid restriction comes from the concern that the neurological presentation is actually indicating that the brain is already swelling?

A. Yes.

Q. And if the brain is already swelling, you want to stop that because of the potential fatal consequences of it,

and that's what dictates the restriction?

A. Yes.

Q. It's that bit that I want you to help us with. The logic of it seems clear. There are two elements to it: one, you restrict the amount of fluids going in; two, the amount of fluids going in have their sodium density or concentration increased.

A. Yes.

Q. That two-pronged approach to that is where I want you to help us with how you can be so certain that that is something that would have been appreciated by a neurologist in 1996.

A. Well, I think a neurologist should have had that as part of his training in the management of acute neurological disease.

Q. And why do you say that?

A. Because it is part of the management of acute neurological disease in the brain and it is something which occurs. Therefore, it is simply just part of an approach to take in dealing with those problems. I feel that a neurologist providing care in a regional centre, dealing with acute encephalopathies, would have known that. Certainly should have known it.

Q. Well, let's put it this way: up until whenever the transfer of perhaps the more serious children went to

intensive care in Leeds and that whole aspect of that service went to Leeds, is that how you were treating patients who had that presentation in your hospital?

A. Yes.

Q. Is that because that's how you were trained to do it?

A. In part. It was also from the experience that I had in dealing with a range of acute encephalopathies, including Reye's syndrome, and knowing that this was a problem; it's also well documented in head injury and neurosurgical conditions. So it was just part of our routine practice. It was also referred to in my training.

Q. I wonder if I could just ask you about Reye's syndrome. As I understand what you have just said, Reye's syndrome is one of those conditions which falls into this category of being treated by a reduction, if you have good reason to suspect it's there, in the fluids and an increase in the sodium content of the fluids; is that right?

A. Yes.

Q. Without maybe getting overly technical for the laypeople, what is Reye's syndrome?

A. It's an acquired disease of the mitochondria in the cells. What are mitochondria? They are the factories within the cells, the chemical processing plants within

the cells, which are supposed to be efficient, and something affects them and makes them stop doing that efficiently. When you get that happening, brain swelling occurs because the mitochondria in the brain are disturbed and the mitochondria in muscle are also disturbed and the mitochondria in the liver and the heart and the kidney are disturbed. The kidney and muscle problems are not so critical. The really critical feature is the involvement of the liver where fatty infiltration occurs and is evident on post-mortem. It is diagnosed by finding out-of-range coagulation on a blood test. Why? Because the liver makes the coagulation factors.

And it is identified by finding abnormal liver function tests because the liver isn't working properly, so you get deranged liver function tests. You can find abnormal enzymes in the blood related to the muscle. You get an elevated blood ammonia in some and it's characteristic to get a low glucose, but not in all. In my experience, not all. Most of the Reye's syndrome reported to the national reporting body in Yorkshire came from our unit, for reasons to do with the fact of diagnosis, I suspect.

But it is thought to be linked to the use of aspirin, treating acute intercurrent illness, so since

aspirin is withdrawn, the incidences have fallen dramatically, but it still occurs.

Q. What is the feature of it that requires it to be treated in the way that you've identified to the cerebral oedema?

A. In Reye's syndrome, cerebral oedema is a major problem, from the mitochondrial dysfunction.

Q. So that produces the cerebral oedema --

A. It does.

Q. -- which you then address in the way that you were generally speaking about addressing cerebral oedema?

A. Yes. In any condition where the brain swells because of the disease of the brain -- any disease of the brain can produce brain oedema because the neurones swell, and infection, particularly encephalitis, meningitis. What you don't want to do is to make that worse, and making it worse is giving too much free water and that's the doctor side of it, if you like, iatrogenic. The syndrome of inappropriate --

Q. Sorry, can I pause you there and stay with Reye's syndrome because there's a particular element of that what I want to ask you about. When I had asked you about what you thought would be the knowledge that was current in 1996 about how you treat cerebral oedema or address it in relation to the fluid management, and you

mentioned Reye's syndrome, you said that what you have just described is something that you would expect a neurologist, a paediatric neurologist, to have that knowledge as part of their training. You would expect an element of it, if not all of it, to be known by general paediatricians.

In your first report, you express the view that, in the Children's Hospital in particular, you would have expected them to have developed some guidelines around that because they had Dr Glasgow, who I think you referred to, as one of the leading experts of Reye's syndrome and its treatment. Can you help us by explaining that a little?

A. John Glasgow has produced a number of publications. He was present at the Reye's syndrome workshop that I chaired in, whatever it was, 2001, and he is well-known to be an expert in it. He has a special interest in Accident & Emergency medicine at the Children's Hospital.

Q. And can you help with when he would have been publishing or engaging in the research in that area so that one could begin to say that even if people, more generally than neurologists and paediatricians, didn't appreciate that, which is not your position, they certainly ought to have because they had in their own hospital somebody

who was somewhat of an expert in a particular condition which is treated in that way?

A. I can't from the top of my head just produce the references, but they were certainly around that time. It is possible to find out from the Internet?

Q. By around that time, do you mean 1996?

A. Yes, or before.

Q. Thank you. Sorry, I had interrupted what you were saying. I wanted you to address that while you were mentioning that.

A. It is one example of a brain disease which causes brain swelling. There are others, and it's rare, but it has to be covered. Encephalitis, obviously, and meningitis both do the same thing, they cause brain swelling, therefore the management is to stop that getting worse.

THE CHAIRMAN: Let me take you back a few minutes, doctor, if I can. There is a consensus now that a test which should have been done on Claire on the Tuesday morning wasn't done. You have then said, although it's a conjecture, that the probable test result would have been lower sodium either because of SIADH or water overload.

A. Yes.

THE CHAIRMAN: And the response at that time would have been to change to at least 0.45 and to restrict fluid intake.

A. Yes.
THE CHAIRMAN: You then said that you restrict the fluid because the neurological presentation indicates that the brain is already swelling. In the absence of a fresh blood test on the Tuesday morning, would the continuing lack of consciousness now extending over a prolonged period have given that indication in any event?
A. Yes, but I would say that, at that time, I think, quite appropriately, the paediatric registrar sought a paediatric neurology opinion.
THE CHAIRMAN: You see, what I'm looking at is what is the -- I mean, it is conceded all round that a test which should have been done wasn't done.
A. Yes.
THE CHAIRMAN: And that is, at the very least, a contributory factor to the decline of Claire as Tuesday went on, without this problem having been identified as clearly as it might have been.
Doctor, you know that Dr Webb's position is that he misunderstood the clinical records to mean that the reading of 132 had been obtained on the Tuesday morning. You've expressed some surprise in your report about that interpretation. But let's assume for the moment that it's right and let's assume that, as Tuesday moves on, it is understood somehow that the reading of 132 comes

from that morning. It's low-ish, but it's not necessarily, on its own, particularly concerning; isn't that right?

A. Well, the view that I've expressed is that for a general paediatrician in a child without encephalopathy, it is not particularly significant. But I've also taken the view that for a paediatric neurologist where there is acute encephalopathy, even a measurement of 132 should have been a red flag that this common and very serious complication of hyponatraemia was evolving because it is well recognised over that time -- and I have given the sources from the textbooks -- that this was a problem that was well recognised. So I believe his action should have been, when he saw Claire, to have taken the steps to deal with it already, even on a figure of 132.

THE CHAIRMAN: Thank you.

A. I have another rider on that. When Dr Webb saw Claire, the range of blood investigations which had been carried out was limited. And the guidance in 1984, third edition, and in the fourth edition in Forfar & Arneil, the guidance in the Nelson textbooks and the paediatric neurology textbooks, all -- certainly the Forfar & Arneil -- include a range of investigations. They were not done.

So the next step for Dr Webb to have done at the

2 o'clock consultation, in my view -- and supported by the guidance of the time -- is further blood tests then. So that even if the sodium was thought to have been done in the morning, another blood test should have been done for liver function tests, for blood ammonia and, possibly, toxins. And had that been done as a consequence of his consultation, the blood sodium, which on balance of probability would have been much lower, would have become available and knowledge would have been there towards the end of the afternoon on the 22nd, so the omission of that 2 o'clock blood test compounded matters.

THE CHAIRMAN: Thank you.

MR GREEN: In his report, Dr MacFaul also expresses a view on Dr Webb's explanation for misunderstanding, as he would have it, the 132 result in the ward round note. It's at ...

THE CHAIRMAN: Page 21?

MR GREEN: Page 26, in fact, paragraph 121. So the reference is 238-002-026. If we take it up about halfway down it reads:

"Although in his statements [this is referring to Dr Webb] he ascribes this oversight to the fact he thought this test had been done just before he saw Claire. This is difficult to understand because the

entry of the result is in the notes in handwriting and timed."

I just wondered if the inquiry would be assisted if Dr MacFaul were briefly to elaborate on that.

THE CHAIRMAN: Since I raised the point, let's look at it. Could we bring up 090-022-053, please? What you see, doctor, on the left-hand page is the continuation of the note of the ward round, which occurred at some point around 11-ish, a bit after 11, on the Tuesday morning.

A. Yes.

THE CHAIRMAN: The blood result is written in on the fourth line --

A. Yes.

THE CHAIRMAN: -- and is followed then by "on examination". It continues down to the plan.

A. Yes.

THE CHAIRMAN: Your report on the right side of the screen, at paragraph 121, suggests that you have some difficulty in understanding how Dr Webb, at 2 o'clock, would have understood that reading of 132 to have been from that morning.

A. Yes, for two reasons. One is that the only blood test that had been done had been done the night before and was written in the notes in the previous page or whatever that related to the midnight entry. Now,

I appreciate that the entry of the result followed Dr O'Hare's consultation.

THE CHAIRMAN: Mm-hm.

A. But there's nothing in the notes which says there that a blood test has been done, other than that previous one the night before.

THE CHAIRMAN: Right.

A. I can see how he would make a mistake on that one. It's not too difficult to see. But that's the ward round. There's nothing in the notes before that which says another test has been done.

THE CHAIRMAN: So your point is that, if I understand it, it looks to you like a mistake which he made --

A. Yes.

THE CHAIRMAN: Sorry, it is a mistake that he made?

A. Yes.

THE CHAIRMAN: Because it wasn't a reading from Tuesday morning?

A. No. There was one done the previous night and that's the same results.

THE CHAIRMAN: Yes. Your second point, which I think you've just extended on, is that even if he did -- even if that error is just a simple one, and these things do happen --

A. Yes, they do.

THE CHAIRMAN: -- so let's not be over critical of Dr Webb for that --

A. No.

THE CHAIRMAN: -- the fact is that, at 2 o'clock, having been called in to see a child who was causing concern, which was why he was brought in in the first place, he should have required more tests to be carried out, including a blood test?

A. Oh yes, yes.

THE CHAIRMAN: So even if he thought that that was a morning blood result, at 2 o'clock he should have required a further blood result and further tests along the lines which you have just described?

A. Yes, as a minimum liver function tests and consideration of blood toxicology. And they would have included the urine and electrolytes because they're done at the same time as a liver function test.

THE CHAIRMAN: So the problem isn't so much, on this interpretation, that he misread the timing of the only blood test which had been done to date; the real problem is the lack of investigation which follows?

A. Yes, and when I was approaching this brief, I was approaching the brief to look at governance issues, taking regard particularly of clinical issues. And one of the ways I approached that was to consider what was

done in clinical terms and to try and match that with the practice of the time and awareness of the time of this condition, given 1996, and the guidance available in various texts.

Clearly, I have erroneously referred to the third edition and I accept that, but I did not expect there to be a change and there wasn't a change in the advice given in the third and fourth about the range of investigations which should be done, including blood tests, and it does include in both the liver function test and monitoring of electrolytes.

By the monitoring, if he judged that was morning, he might have thought it had been done, but he hadn't extended the range of blood investigation, and that I am critical of.

THE CHAIRMAN: Can I ask you it in this way: Professor Neville, for one, was more critical of Dr O'Hare than you have been, Dr O'Hare being the registrar overnight. He was saying she did a competent examination, but he thought that the range of tests which she ordered to be carried out was too limited. As I read your report, you're less critical of her for that and you've accepted, in broad terms, that it was acceptable for her to do what she did and then allow things to be picked up in the morning, particularly in

light of how Claire had recovered or not recovered or progressed overnight.

A. Yes.

THE CHAIRMAN: So if there is criticism of Dr O'Hare from others for the narrowness of the testing which she did, which she required on Monday night, does that emphasise your criticism or do you think it adds weight to your criticism of Dr Webb for the lack of testing which he required on Tuesday at 2 o'clock?

A. Well, I think that the general paediatric position at that time, that's the midnight, was not sort of locked into the framework of acute encephalopathy. Dr O'Hare had chosen to let events take their course for a while to see what was going to happen, and many children are a bit off it, particularly around midnight after an illness. So I can see how her thinking was going as a paediatric registrar.

I'm much more critical of the paediatric neurology approach because Dr Webb should have been aware of the need to extend the range of investigation at that time because she was well-established, by that 2 pm consultation, to be well within the framework of acute encephalopathy management.

I appreciate that I'm being very hard on Dr Webb. I think the point is that I was asked to look at how

things should have been done in an ideal world. But I still feel that, at that point, there was a major omission.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: So much has been made of the differences in the wording between the two editions, the third and the fourth.

A. Yes.

Q. I am trying to see if you can help us with the underlying purpose of the assessment and treatment and therefore to see if it is your view that the underlying understanding of the interrelationship between the application of low-sodium fluids in quantity and the development of the cerebral oedema, that relationship -- whether anybody thought that that had changed over the time.

A. Well, my view, as I come back to it, is that it was evident in 1984, having now looked in detail at the fourth edition. It is quite clearly encompassed in all the guidance there, although there's a fault in presentation of it. And it's echoed again in 2003. So that core management in terms of fluid management and awareness of brain oedema and inappropriate ADH has not changed over that era, nor has the role of fluid restriction in syndrome of inappropriate ADH.

Q. If we go back to some of the points that Professor Young wanted to make. He makes that very basic point, of course, that the wording is different.
A. Yes.
Q. But he also goes on to talk about guidance on fluid management before and after 1996.
A. Yes.
Q. And he deals with that, he starts to deal with it at 178/2, page 5, of his witness statement. He, in some detail, deals with it, so he starts with Arieff and others in the paper that was published in the British Medical Journal in 1992, dealing with the 16 cases of hyponatraemia in children, who were undergoing surgery. And essentially, the tenor of his point at this stage is that if you were looking to see what people understood about the development of hyponatraemia, then this paper would be putting you in the direction that that is a complication that can arise associated with surgery or the immediate post-surgery period.
Do you have any comment about how he has addressed that starting point for hyponatraemia or even if that is the appropriate way to look at the issue that you are concerned with in Claire's treatment?
A. Hyponatraemia can occur after surgical conditions. It's an unusual complication. Hyponatraemia in acute

encephalopathy is a common -- it's integral to the management, it's a common condition, relatively, almost 30 per cent of bacterial meningitis, N per cent of viral encephalitis, and so on. So within acute encephalopathy, the child is much more at risk of hyponatraemia related to intravenous fluid and inappropriate ADH secretion.

The Arieff paper, in my view, is highlighting an occasional complication, which is not very frequent, but it's highlighting it, and the complication that can arise from fluid administration in a child who was previously conscious and where the fluid administration has caused the brain oedema. That's a completely different kettle of fish, if you like, to the child who already has a disease of the brain which is likely to develop cerebral oedema and then using a therapy which can actually make it worse.

Q. Yes, that's exactly what I was going to ask you to explain. That's why I said in the context of the issue that you want to deal with in relation to Claire's management and treatment. So here, are you saying that some of these papers that Professor Young is dealing with are talking about the knowledge that people might have had at the time that the mere application of hypotonic low-sodium fluids could itself produce

cerebral oedema and therefore that particular connection between the two things is something that one ought to be alert to and that was part of what his paper and others thereafter were dealing with? Whereas it seemed to me that you were trying to emphasise something different, which is: this is a brain that has its vulnerabilities because there is a problem already there and it's how you ensure that you do not exacerbate that problem and, in fact, arrest it before it carries on to reach fatal results. Is that the distinction that you're trying to make?

A. Yes.

Q. Would you accept that that particular link between the development of hyponatraemia through the administration of low-sodium fluids per se, and therefore that link to cerebral oedema -- how widely known would you consider that to have been in 1996, whether it's from the general paediatric side or the paediatric neurological side?

A. I think amongst general paediatric practice, I think it is probably less well-known than in paediatric neurology practice, save for the management of bacterial meningitis, where anticipatory care by the use of higher sodium content is advised. But that is something which was not generally known as well as the management of encephalopathy by a paediatric neurologist.

Q. Okay. So as it happens, you've obviously seen the clinical notes. It would appear that that is something that at least Dr Stewart understood. It's a very brief note, but it may give a pointer to the level of understanding at least for one of the junior doctors at that time in 1996, and we can pull up 090-022-056. You can see right at the top there at 23.30, just below his recording of the serum sodium result of 121, then you see, if you like, his thinking, "hyponatraemia". He queries fluid overload and low-sodium fluid and he also queries SIADH.

Were you surprised that he reached that formulation or how did that accord with what you would have thought a junior doctor in paediatrics would have appreciated in 1996?

A. Well, I think he obviously did read his textbooks and he gleaned something from them. Because it's not only in Forfar & Arneil -- junior doctors at SHO level tend to use more readable textbooks for quick learning. They would study the Forfar & Arneil textbook when preparing for membership. I don't know whether Dr Stewart was preparing for membership. But the textbooks that were used by SHOs would include Forfar & Arneil, but they would also include little handbooks and the one that I've referred to is the vade mecum, and they would also

use "Hospital Paediatrics" by Milner & Hull -- and I have given extracts from that in my more expanded response to Professor Young -- where those problems are highlighted in management of coma, and obviously Dr Stewart has studied well.

Q. But just so that we're clear about the distinctions that you are making between the brain, which has become vulnerable for some reason -- of course, it's that underlying reason one wants to get at.

A. Yes.

Q. But before that proves to be fatal, you need to address that, recognise that there is an underlying reason like that and address it.

A. Yes.

Q. And then there is the line of publications that I had started with, Arieff and his colleagues, to raise with you, which is leaving aside that, no problem necessarily with the brain at all, you just apply too much low-sodium fluid and produce hyponatraemia in that way.

A. Yes.

Q. At this stage, she has two factors one can take into account. One is that she continues, despite the anticonvulsant medication, to be presenting with her neurological problems. The other is you have now a second serum sodium result, roughly 24 hours perhaps,

from bloods taken, and this one is very definitely below the register. And worryingly low, would you say?

A. Yes.

Q. So at this stage, it's not clear maybe to a paediatrician whether there is a problem that started with a vulnerable brain and has not been assisted or not a vulnerable brain, but just too much low-sodium fluid having been ascribed. Irrespective of which it turns out to be, is the response to that the same from your perspective and would that response, as you now give it to the chairman, be something that would have been appreciated in 1996?

A. Yes. And had those results been available late morning or mid-afternoon, no doubt the registrar would have come to the same conclusion: that it required management.

Q. So for whatever reason, however you have got there, you have got to a worryingly low serum sodium level, and that has to be addressed, and as I understand you to say, it would be addressed in the same way whether it had been produced because there was an underlying neurological problem that was responding to low sodium fluids or because simply too much low-sodium fluid had been administered; is that right?

A. Well, that's a possibility. I mean, in the context of Claire's illness, of course, there was a brain problem.

Whatever it was, she had a brain problem. So that would help to trigger the inappropriate ADH rather than just the intravenous fluid itself contributing.

THE CHAIRMAN: When you said "she had a problem, whatever it was", do you say that because it's not quite possible to say exactly what it was because of the limited autopsy?

A. Yes.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Thank you.

I had, in fact, just referred you to one paper, but Professor Young goes on to refer to a number of them: the 1999 Bhalla et al paper; he refers to the 2001 Kirkham review; then the Hoorn et al from Toronto in 2004. Is it your view that those papers are all along the same theme, in other words dealing with the increasing appreciation of low-sodium fluids in and of themselves producing hyponatraemia and requiring to be addressed as opposed to the compromised brain, vulnerable brain, responding in a certain way?

A. Well, of course Dr Kirkham's paper is considering the management of coma, non-traumatic coma. So it's specifically addressing a comatose child. If you have a coma, you have a problem with your brain -- unless you've been given an anaesthetic of course, but I suppose that's a problem.

The Kirkham paper does counsel against the use of low sodium fluid, although the wording is not specifically "hypotonic", and I've commented on that. It's a matter for elucidation at some time, I think.

Q. Let's pull up the particular bit that has been cited by Professor Young. 178/2 at page 7, and if we can pull up alongside it the next page. It starts at the bottom, which is her review on non-traumatic coma. If we pause there: was your position that this is in a different category from the Arieff et al line of papers?

A. Yes. She does refer there to cerebral salt waste, which is another issue, and I can come back and comment on that.

Q. Does this capture the element where you say she has addressed matters but perhaps not as clearly as you think it might be done or has he not extracted the part that you would wish to rely on?

A. Well, Dr Kirkham does mention hypoosmolar fluids as being contraindicated because of cerebral oedema. But actually, both of the fluids which she quotes as examples are not hypoosmolar. The 5 per cent dextrose has been classified by the NPSA report in its table as iso-osmolar. And the 10 per cent dextrose is hyperosmolar. Both are absent of sodium and both are hypotonic. So I think there's a bit of confusion about

that message. I think she's right to say that fluid management can be very difficult, she's right to say it needs to be tailored to the individual child's needs. But she does say that fluid restriction is potentially harmful, and that -- by that time, there was an active debate because fluid restriction up to that point, and indeed beyond ... Certainly in the textbook of 2003, fluid restriction was the norm for acute encephalopathy with hyponatraemia. It was the normal practice.

So what she is doing here is to say, "Well, is it safe?", and I think that's a very reasonable question because, in the 80s and in the early and mid-90s, I think we were overrestricting fluid. We were also overresorting to hyperventilation, and both of them we realised, I think, can cause damage through different mechanisms. So the idea of shifting the severity of the fluid restriction was in the direction of reducing it, and Dr Kirkham goes as far as almost advising against it.

Q. She's doing that also in the context of salt wasting. Maybe this is the point to address that because that produces the hyponatraemia, but by a different mechanism --

A. Yes.

Q. -- in the sense that you lose sodium; would be I right

in saying that, putting it simplistically?

A. Yes, it leaks in the urine. It's something which has been addressed, but only slightly so in the major textbooks. So in 1996, cerebral salt wasting was not high in the thinking processes of controlling hyponatraemia. And my own take on that and my own understanding of it when you brought it up with me this morning is that it is more a feature where the coma has been prolonged, where you have somebody with a very severe head injury or a brain injury of some other kind, like neurosurgery -- I don't want to insult my neurosurgical colleagues, but what I mean is that when they operate, very often the brain is disturbed for a while, while they're recovering -- and after subarachnoid haemorrhage, for instance. Then under those circumstances, when the patient has been comatose for a day or two or more, then there appears to be this attempt by the body for whatever reason to leak sodium.

Under those circumstances, though, the biochemical profile is different from that which Claire had in the sense that the potassium level was less than it was on admission, whereas in salt wasting, it goes high.

And there is a tendency to hypovolemia, that's underfilling of the circulation. In a girl like Claire, that would be manifest by tachycardia -- high pulse

rate -- she didn't have a high pulse rate. I mean she had a slightly high pulse rate at 100, but nothing like what would happen in hypovolemia, where it would be about 130 upwards. So I don't think she had cerebral salt wasting, so that is why I wanted to address it.

THE CHAIRMAN: I gathered from Professor Neville's evidence that, acknowledging the various mistakes which he said had been made, that Claire's condition was, to be fair to everyone involved, actually quite difficult to manage; is that something you would agree with?

A. Yes, I think any acute encephalopathy is difficult to manage. I think her presentation was somewhat unusual in the sense that she seemed to slip down slowly rather than go rapidly into a deep coma. And yes, I think all acute encephalopathy is difficult to manage, but there are some cardinal rules and those are to do a lot of tests, which I've dealt with, and to manage the hyponatraemia when it occurs. That's relatively straightforward in the sense of the guidance of the time. The debates about fluid restriction have emerged in the early 2000s. But that difficulty about fluid balance in acute coma continues. I was focusing on 1996. That difficulty is manifest in the College guideline on acute loss of consciousness in children because they there do not address fluid management in

the early stages. They don't envisage anticipatory care and they only suggest hypotonic fluid removal if there are symptoms or signs of raised intracranial pressure.

THE CHAIRMAN: Am I right to get the sense from you that, by definition, acute encephalopathies are difficult to manage?

A. Yes.

THE CHAIRMAN: And that is why you have anticipatory care, that's why you do the tests?

A. Yes.

THE CHAIRMAN: That's why you consider whether fluid should be restricted or a different type of fluid might be given?

A. I think when you have hyponatraemia, even if it's just a low sodium outside the normal range, you're triggered into maintaining homoeostasis. That's why 132 is a signal that you may have a problem.

THE CHAIRMAN: Right, thank you. Mr Fortune?

MR FORTUNE: Sir, can I take you back to Professor Young's witness statement, 178/2 at page 5, in which he refers to the paper, Arieff et al, published in 1992? Bring us up to 1996, and if I've understood Dr MacFaul's evidence correctly, he was saying that he expected the contents of the Arieff paper to be known amongst paediatricians and also paediatric neurologists. Can I take you back

to the evidence of Professor Gross on the afternoon of 9 May this year and, in particular, to the transcript for that day at page 126 and on to page 127? If we could have them up side by side, please.

You'll recall that it was at this stage that we were discussing, on line 20, the Arieff article, as it was called. Then on page 127 at line 5, you take a hand, sir:

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

Professor Gross disappointed you when he said:

"I was no longer in Denver, I was back in Germany."

And then he went on in this way:

"I am afraid I would have to say, even though this turned out to be a landmark article, very important article, it was not widely known. I think it was known to many nephrologists because they're reading this kind of -- electrolytes is considered to be the field of nephrology and endocrinology. It's my experience that the knowledge in the field of electrolyte disturbances, hyponatraemia amongst them, with anaesthetists is better than with many internal medicine people. Whether anaesthesiologists would have read this article, I kind of doubt. I think it probably was -- I'm sure it was not well-known amongst internists in Germany. I think

it was not very well-known amongst nephrologists in Germany. I think it was not very well-known amongst anaesthetists."

Sir, if that was the state of knowledge in Germany -- and you'll recall that Professor Gross had extensive experience of practising outside Germany -- then what does Dr MacFaul say about those comments?

MS ANYADIKE-DANES: Sorry, Mr Chairman, just before he does, could we have page 128, please?

MR FORTUNE: Any particular line?

MS ANYADIKE-DANES: Yes, I think it'll be obvious. Line 5. Moving on to -- if you go to 20:

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

THE CHAIRMAN: Well, in essence, this was evidence, doctor, which was given in the course of the hearings about the death of Adam. What's being highlighted for you is whether you can be so sure about the extent of the knowledge of the Arieff paper and the extent of the knowledge that comes from that in the mid-1990s.

A. May I just ask if the opening remarks of your question can be read back when you suggest specifically that

I made reference in my paper that the Arieff paper should be well-known?

THE CHAIRMAN: Yes. What Mr Fortune said -- this is Professor Young referring to the Arieff paper.

A. Ah.

THE CHAIRMAN: At 178/2, page 5, in which --

A. Yes, I can see that.

MR FORTUNE: "This was the first paper which may have been noticed by a wide readership as it was published by a significant UK journal."

THE CHAIRMAN: Yes.

A. I misunderstood the beginning of your question because I thought you had said that I had said in my report that this paper should be well-known to paediatricians.

MR FORTUNE: No, you said in evidence -- and if necessary we'll have to go back in the transcript -- that you expected it to be known amongst paediatricians and -- perhaps to a different extent -- paediatric neurologists.

A. Well, can we go back to the transcript? If I can clarify it: I did not mean to say that the Arieff paper would be well-known to paediatricians; in fact, in my response to Professor Young, I say something to the effect that if it was known to the paediatricians ... Because what we're talking about with the Arieff paper

is where hyponatraemia has been caused by -- and cerebral oedema -- caused by the intravenous infusion of low-solute fluid. That in, say, a child with appendicitis, I don't think is well-known and was not well-known. What I was trying to establish was that it was well-known as a complication of acute encephalopathy. I will stand to be corrected. I don't think I say in my report that the Arieff paper should have been well-known to paediatricians, and I don't think I said it this afternoon. But I stand to be corrected.

THE CHAIRMAN: I've got one reference to what you said earlier, in which you said -- this is at page 83, line 16 of the [draft] transcript:

"The Arieff paper, in my view, is highlighting an occasional complication, which is not very frequent, and the complication that can arise from fluid administration in a child who was previously conscious and where the fluid administration has caused a brain oedema, that's a completely different kettle of fish, if you like, to a child who already has a disease of the brain, which is likely to develop cerebral oedema and then using a therapy which can actually make it worse."

That's the contrast you were drawing between the type of scenario which the Arieff paper was

investigating and what you are describing to me as the more established and better recognised scenario of having to be careful about and pre-emptive about the administration of fluids in children who have acute encephalopathies.

A. That reflects what I was aiming to say. I don't think the Arieff paper was well-known in paediatric or anaesthetic practice for that matter.

THE CHAIRMAN: Okay.

MS ANYADIKE-DANES: Can I help by drawing up the bit where you do deal in your report -- I know you've been trying to find it there as you are responding. It's 238-004-006:

"I agree to some extent on his comment on the relevance of the Arieff et al 1992 paper to Claire."

And then there's a bit. Then you give your comment:

"My comment: this paper mainly focussed on causation of encephalopathy with IV fluid use rather than management of an existing encephalopathy, but knowledge of its content should have influenced fluid choice had the clinicians been aware of it."

So I don't think you have there said who would have been aware of it. I think you have simply said that if they had they been aware of it, it would have been an aid. But in any event, you're making the distinction

between it causing the encephalopathy rather than managing an existing encephalopathy.

A. That's correct.

THE CHAIRMAN: Sorry, one second, Mr McAlinden.

From what you have just said a few moments ago, do I understand that the phrase at the end of that page, "had the clinicians been aware of it", is an acceptance on your part that it is not a paper which was terribly well-known among paediatricians --

A. Yes.

THE CHAIRMAN: -- and paediatric neurologists?

A. Probably so because it relates really to giving intravenous fluid to otherwise healthy children, often.

THE CHAIRMAN: Okay. Mr McAlinden?

MR McALINDEN: Mr Chairman, just to explore the witness's evidence in relation to the knowledge of the need to restrict fluid in children suffering from acute encephalitis or encephalopathy.

Would the witness be able to comment on the paragraph WS178/2, page 7, starting with the words "in 2001" and, in particular, from the words "all the children had received hypotonic fluids" --

THE CHAIRMAN: This is about halfway down the paragraph?

MR McALINDEN: Yes. Just basically that section of the paragraph where Professor Young has highlighted that:

"Even by 2001, the use of hypotonic fluids remained routine even in acutely unwell children with encephalitis."

Perhaps he could comment on that.

THE CHAIRMAN: This is obviously something you've read because you've responded to it, doctor, but just take your time to read that paragraph. You're being invited by the Trust to respond to that proposition.

A. Well, it is clear that it's documented there that they had received hypotonic fluid even though they had encephalitis. If that is what is being referred to ... "13 had developed in the post-operative period", "15 referred to critical care". Where does it say encephalitis was a risk factor? In this paper, highlighted --

MR McALINDEN: Professor Young -- and I'm sure you have already read this in detail -- is saying in this paper that:

"The authors, in contrast to earlier publications highlighted above --

In other words, this is the first time in which, in the series of papers that he has referred to, any learned author is referring to the complication of encephalitis. He's saying:

"In this paper, the authors, in contrast to earlier

publications highlighted above, identify, as a risk factor, disturbances of the central nervous system, meningitis, encephalitis."

And then it states:

"In their conclusion, they recommend that the currently used guidelines for maintenance fluids in children admitted to hospital must be changed because they do not take into account the unpredictability of vasopressin secretion."

Highlighting the fact that in 2001 the use of hypotonic fluids remained routine, even in acutely unwell children with encephalitis.

So the point that he's making is that, contrary to the suggestion that you're making that this was old knowledge at that stage, he is saying that the reason why this paper was published in 2001 is that it wasn't old knowledge; this was new research that they were looking at and giving guidance in relation to encephalitis.

A. Yes. I think when I scanned that, I didn't see in there that this was relating to encephalitis. It mainly seemed to be focused on post-operative. But I would come back there to you and refer back to the information that is present, not just in research papers, but in the textbooks of the time, which raised this as an issue and

raised it as a particular issue in acute encephalopathy management. So I think that that's an interesting observation, but it doesn't get away from the fact that it was usual practice to address acute encephalopathy where there was a low sodium or an out-of-range sodium with fluid restriction plus or minus the adjustment of the blood sodium.

MR FORTUNE: Sir, Dr MacFaul ought to be reminded that he can look at the actual "Lesson of the week". It's to be found in WS178/2 at page 43. It's a very short paper and, indeed, Dr MacFaul might want to take a little while just to look at it to comment upon its content.

MS ANYADIKE-DANES: We can pull the next page up as well because that's the page that has the discussion.

I don't think we can get all three on at the same time, because there are three pages to it, but if we pull up 44, you'll get the discussion part of it, and then there's a third page, but maybe that's enough to start the process. I don't know if that's clear enough for you to read.

A. No. Could you blow up page 44, please?

Q. Yes.

A. Thank you. (Pause). Is there a following page?

Q. You might want the next page as well, which will continue the discussion. Page 45. That might help.

(Pause).

A. Well, without going through it in detail, may I point out that it seems to be focusing on the management of hyponatraemia and caution being given to too rapid a change of the sodium in treatment. It post-dates the event in 1996.

THE CHAIRMAN: I think that's the point that's been made, against you, against your proposition. I think if I understand Mr McAlinden correctly, it is that is Professor Young not right to say that this shows that it was only around 2001 that this issue was being flagged up for an increase in the saline content?

MR McALINDEN: If one looks at page 43 in the coloured box, it specifically lists a number of causes of vasopressin release. And in relation to that, you'll see specifically mentioned:

"Disturbances of the central nervous system, meningitis, encephalitis."

A. Yes.

MR McALINDEN: So this is research as of 2001, where you're saying that the knowledge base was well in existence before then.

A. Yes. The knowledge base is present in the textbooks, which I have referred to, as well as in other textbooks.

THE CHAIRMAN: Sorry, how could this be breaking news in

2001 if it was in the textbook which Professor Young is criticising Dr MacFaul for using from the 1980s? Sorry, if I understand the proposition, you're saying, this shows that Dr MacFaul is wrong because it's only in 2001 that this is emerging. As I understand your response, you are saying it can't be right that it is emerging for the first time in 2001 because this is what was in the textbook which you referred to in your original paper?

A. Yes, and other textbooks, all of which address the problem of being managed with fluid restriction and other books address how to deal with a low sodium in terms of adjusting intravenous rate. Some go straight to 0.9 per cent saline, which is normal. Some, including Nelson, state that if the symptoms are serious -- this is the current Nelson for 1996 -- it goes on to say that if there are symptoms associated with hyponatraemia such as coma or convulsions, that they should have hypertonic saline. Others would say if the child is not dehydrated and the sodium is low, to give 0.9 per cent. My own practice was to give 0.9 or 0.45.

The maintenance of homoeostasis is the issue here. One is the inappropriate ADH, but the maintenance of homoeostasis is to find a low sodium, and if a low sodium is present, to stop making it worse by giving

a low-solute fluid, and then the intravenous fluid that you're using to increase the sodium content. That was the practice and that is what's envisaged and set out in various portions of textbooks that are applicable to 1996.

MR FORTUNE: Let's go back to page 45, please, sir.

THE CHAIRMAN: I just notice by the way that one of the co-authors of this paper is Desmond Bohn, who is one of the peer reviewers to this inquiry, which unfortunately means he's not a witness.

MR FORTUNE: Page 45, please. Doctor, would you please go to the paragraph under the heading "Study limitations"? Read that to yourself because the authors refer to the currently used guidelines for maintenance fluids in children. (Pause).

A. Yes.

MR FORTUNE: Why should the authors in 2001 be recommending that:

"The concentration of plasma sodium should be measured when starting an intravenous infusion, taking account of the currently used guidelines for maintenance fluid, that there must be a change because they do not take into account the unpredictability of vasopressin secretion"?

Something that must have been known about, on your

basis back, in the 1980s or even the 1990s; is that correct?

A. It is correct, and I have said so in my report, that the routine intravenous fluid used in general paediatric practice was fifth-normal saline. And that is the case. Routine. And it is good guidance, obviously, to do electrolytes first. The point is that in an acute encephalopathy, you are not in a routine situation, and you anticipate -- or the ideal practice is to anticipate a development of inappropriate ADH secretion, but I have accepted that the ideal practice would not necessarily apply to the admission and overnight.

But by the middle of the next day, when a paediatric neurologist was involved, it was already known that her blood sodium was outside the normal range. Further information was not sought. Under those circumstances, while waiting for a repeat sample, the appropriate steps following guidance would be, particularly with a signal of a low sodium, to restrict fluid, and that's well documented in many texts, and to add sodium. Why add sodium? To stop donating free water, which is another complication. At that point, when you have a low sodium, you do not know whether it is water overload or whether it is inappropriate ADH or a combination of the two. And therefore, logic dictates that the maintenance

of homoeostasis is to attempt to restore the blood sodium from outside the range to within the range. And that is done by adding sodium concentration to the intravenous fluid.

There is a caution against doing it too rapidly because of causing brain damage, but it should be done in stages. And indeed, that caution is thrown away in certain texts, including Nelson, when a child is having fits and in coma.

THE CHAIRMAN: Thank you.

MR FORTUNE: [Inaudible: no microphone] as you know, I'm going to deal with the state of knowledge amongst paediatricians.

MS ANYADIKE-DANES: Thank you.

A. On that point, may I say that, amongst paediatricians, of course, the advice was fluid restriction in meningitis, bacterial meningitis, which is a common condition for general paediatricians to treat.

MR McALINDEN: Sorry, just in relation to that point, where you say that the advice on bacterial meningitis was fluid restriction, could you comment then on the Kirkham review, which is referred to in Professor Young's statement at the top of page 8? Where she says:

"There is considerable controversy over fluid restriction, which has been shown to be potentially

harmful in patients with subarachnoid haemorrhage and meningitis. The syndrome of inappropriate secretion of ADH for which fluid restriction is indicated is relatively rare. Instead, cranial diabetes insipidus may require careful management. It is essential that the systemic circulation is well filled and that large volumes of hypoosmolar fluids are not given."

THE CHAIRMAN: I thought Dr MacFaul had spoken about this earlier on when he said that Professor Kirkham was highlighting an issue which was that, in the 1980s and through most of the 1990s, I think you said, there is, looking back on it, an issue about whether there was an overrestriction of fluids; is that right?

A. Yes. Fluid restriction was normal practice in the 1990s and, to a certain extent, in the 1980s, in the face of hyponatraemia, where syndrome of inappropriate ADH secretion was considered a contributor. There's no question about that; it's in the textbooks. But it was probably overdone.

What is concerning here is that children who come in with an acute encephalopathy may not have adequate perfusion and they may be dehydrated. And under those circumstances, the priority is to ensure that the brain is perfused. So you make sure that the blood pressure and the blood volume is maintained because the priority

is to ensure that the brain is perfused. And that is why there is this debate. Once that has been achieved -- and this is a particular problem, may I say, in the bacterial meningitis caused by meningococcal disease, because meningococcal disease produces shock, it produces low blood pressure, for a lot of reasons. Therefore, the priority is to keep that blood pressure going even in a child who is fully conscious, where you know that by doing so -- that is giving loads of actually resuscitation fluid -- you are likely to generate brain oedema. You do that because you know that's the priority, even if you then have to manage the brain oedema by elective ventilation, mannitol, shrinking and so on.

So that is why there has been this concern about overdoing two things in the 1990s: one was fluid restriction, the stringency of it was wound down, less stringent; and, secondly, to avoid hyperventilation. Because in the days when we were doing intracranial pressure monitoring, you could observe how quickly the pressure change occurred if you overventilated. The pressure would go down within half a minute of overventilating. And so many thought: what we'll do is overventilate patients. That did them harm because it produced too much fluid perfusion problems of the brain

because of vascular restriction. So these are cautions and I have alluded to this change in my report when I state that the fluid restriction regimes became less stringent in the 1990s and early 2000s. I concede that. But in 1996 it was conventional.

MR McALINDEN: There's just one further point.

In relation to the next paragraph where you'll see that Professor Young refers to an article written by Albanese and others in 2001. Their main focus was on distinguishing syndrome of inappropriate ADH production from cerebral salt wasting:

"However, of note, there was no recommendation to routinely restrict fluids or avoid 0.18 saline in 5 per cent dextrose."

So that's a paper in 2001 dealing with this issue in the context of patients with acute cerebral insults, yet there is no recommendation for the routine restriction of fluids.

A. Yes. I agree with that, and I've referred to that in my own paper and commented on it, saying that there's no mention of the use of hypotonic fluid. It's a fact, but it doesn't get away from the fact that in the 1990s, 1996, fluid restriction was conventional in the context of a hyponatraemia in a child who was well perfused, where syndrome of inappropriate ADH could have been one

of the contributing factors.

THE CHAIRMAN: Thank you.

A. And I have produced a table in my commentary on Professor Young's report, which indicates that.

MS ANYADIKE-DANES: Just as you have mentioned a number of times that you believed that it was known at that stage, the particular issue that you're dealing with, and I think you have also said that not only was it known then, but although the wording is different, the underlying effects of it carried on being known even though the wording changed in the third edition to the fourth edition; is that correct?

A. That is correct, yes.

Q. If we pull up 238-004-012, maybe we can have 011 alongside it. This is from your report. You're citing there at page 1112 and then you go on to deal with the chapter on salt and water metabolism and deal with the page 771; which edition is that coming from?

A. This is my response to Professor Young. It comes from the fourth edition.

Q. So if you see there, it says:

"In the chapter on diseases of the central nervous system, on the management of acute encephalopathy, page 771, infection accounts for approximately one-third of cases presenting with acute encephalopathy and coma."

Then one goes on to 013, having established that. Then you have your section on raised intracranial pressure. Then if you see at 013, just under page 782:

"The sick child may show the syndrome of inappropriate ADH secretion and an inability to excrete water overload. Water intoxication with oedema and hyponatraemia may result if intravenous fluids are given at the normal rate."

Is this the sort of wording that you're saying doesn't make it quite so clear-cut to piece together what they're talking about, but what they're talking about is a need to reduce the rate of the fluids?

A. Yes. I think that the fluid restriction is more clearly set out in that edition than it is in the warning against hypotonic fluid. What I would say in critique of that edition -- and it has to be said in mitigation of some of the things that I'm saying -- is that the approach to maintaining homoeostasis, although it commends that that is what should be done, is not set out. So the clinicians are left to judge how to do that and they are left to do it from a variety of sources, including first principles of adding more sodium and reducing the donation of free fluid from hypotonic solutions. And it is a pity, in my view, that that has been the case.

Q. In any event, do you remain of the view that it was known that there was that relationship between the brain, which is compromised in some way and therefore vulnerable to developing an oedema, and the use of low-sodium fluids, which would exacerbate that --

A. Yes.

Q. -- and the need therefore to address that?

A. Yes.

Q. And while we're on that, I think, unless I'm going to be corrected, that the doctors have conceded in this case that Claire was a candidate for electrolyte imbalance.

A. Yes.

Q. So far as you're concerned does that mean there's no issue that Claire was vulnerable, if I can put it that way, to the very action that you're describing?

A. Yes.

Q. In any event, and not only was she vulnerable, but her neurological presentation was such that one should have concluded or reached a view, at least from the point of view of a differential diagnosis, that she was vulnerable in that way, even if you had not had that confirmed at any given time; is that correct?

A. Yes.

Q. And I think when you were answering the chairman, that as matters went on from the evening of the 21st right

down to when Dr Webb comes at 2 o'clock in the afternoon of the 22nd, if anything, that should have been confirmed that that was her condition and her problem?

A. Yes.

MR GREEN: My learned friend invited correction in the event that she turned out to be wrong about the proposition that doctors conceded that Claire was a candidate for electrolyte imbalance. Dr Webb actually made that concession.

MS ANYADIKE-DANES: Thank you very much indeed.

THE CHAIRMAN: Is that a way of saying that Dr Sands doesn't make it?

MR GREEN: Yes.

THE CHAIRMAN: Thank you.

MS ANYADIKE-DANES: Oh right, okay.

Mr Chairman, I'm looking at the time.

THE CHAIRMAN: There was once an aspiration to finish Dr MacFaul today. That won't happen. You'll be back tomorrow, I'm afraid, doctor.

We'll start at 10 o'clock. We should be able to finish Dr MacFaul tomorrow, should we? Yes? I hope so. We will then -- tomorrow is Wednesday.

MS ANYADIKE-DANES: It is.

MR FORTUNE: Sir, I'm not sure that my learned friend shares your optimism about finishing Dr MacFaul tomorrow.

MS ANYADIKE-DANES: I don't know why you consider that; I'm going to do my very best to do that thing.

MR FORTUNE: It was the look on your face.

MS ANYADIKE-DANES: I am going to do my best.

THE CHAIRMAN: She never shares my optimism!

MR FORTUNE: Be that as it may, if my learned friend doesn't finish Dr MacFaul tomorrow, are we sitting on Thursday to finish him? Because we cannot have a parade of witnesses --

THE CHAIRMAN: Who are not complete.

If absolutely necessary, are you available on Thursday?

A. Yes.

THE CHAIRMAN: Thank you very much.

That is not an invitation to anyone to extend your questioning into Thursday. That being the case, Dr MacFaul is the last witness this week. If we are approaching the end of his evidence tomorrow at 4 or 4.30, we'll sit on, provided Dr MacFaul is up to it, rather than bring everyone back for an hour or so on Thursday morning.

(4.28 pm)

(The hearing adjourned until 10.00 am the following day)

I N D E X