Wednesday, 21st October 2020

(10.00 am)

PROFESSOR CHRISTINE ANNE LEE, on former affirmation

Questioned by MS RICHARDS (continued)

MS RICHARDS: Professor Lee, before we turn to look at HIV and AIDS, there is just one matter from yesterday I wanted to go back to first. You told the Inquiry yesterday, or your evidence may have given the impression that in the period '83 to '84 and up until the point, really, when you became a consultant, in 1987, at the Royal Free, you had very little day-to-day clinical contact with patients.

A. Yes. Obviously, I would have had contact with patients because we were in the centre, and there were some patients who needed to have blood tests. And I can particularly remember there was a patient who I described yesterday, an elderly gentleman who had an undiagnosable chest infection, that I did see. I remember that.

So I must have had an honorary clinical contract to work in the hospital, but that wasn't my primary role. The vast majority -- I spent four days a week -- I had no on call commitments, and the vast majority of my time was spent looking at notes and analysing all that.

Q. Could we just look at what you said about your work in an interview you gave in 2015 as part of the Royal College of Physicians Oral History Project. So it's THOM0000001, please, Henry. If we go to the bottom of page 26, please, Henry. I just want to show you a handful of passages and check whether these accurately convey the balance of your workload. So the very bottom of the page starts:

"Now, my primary task when I went there was to follow up patients with haemophilia who'd had their first treatment ever with a large ..."

Sorry. I'll wait until we go to the top of the next page.

"... large pool clotting factor concentrate, that's Factor VIII or IX."

And that's the research work that you've described, I think. And then if we go a few lines down, about ten lines down, you should see a passage beginning:

"So my job was to follow up these patients, and they were quite well. They just -- you know, some of them had had an operation; some of them might have had some trauma, which was why they'd had it, but I used to see them take the blood, and then that was tested, and we also used to store it."

So there's that description of seeing patients

taking bloods. And then if we go to page 28, please, Henry. About seven lines down from the top, there's a passage beginning this:

"Then alongside all this comes this strange immunological problem, and I used to look after the patients as they presented. You know, I was -- although I was a pathologist by training, I had done a lot of general medicine, and I was quite a good physician, and they were presenting with strange infections."

Then you go on to refer to the patient who I think is the one that you just mentioned. If we go a number of lines further down, so it's four lines up from the bottom of the screen as it currently is. If you leave it there, Henry, it says:

"So I was seeing these patients."

And then there's a reference to the Haemophilia Society. Then if we go to the very bottom of this page, you say this:

"So to go back to my patients with haemophilia in these two years, I was looking after them clinically, but also, I was doing the research."

So the two years there are 1983 and 1984, aren't they?

A. Yes. I think I would like to just set this in context a little. This is 2015. I'd been retired ten years,

and basically I hadn't done any clinical work for ten years. The Royal College of Physicians' history project was actually to lay down a history archive, and they were particularly interested in the position of women in medicine in the 1960s. You know, when I was at Oxford doing my undergraduate degree towards medicine, there were ten women. You weren't allowed to have any more than ten women in the whole university doing medicine. So that was the basic thrust of it.

The interviews were done by an oral historian, and I think in total in this oral history project, there were three days of continuous -- she was asking the questions, and I would dictate on the phone.

I would also like to stress that, you know, in no way was this saying under oath; I was just reminiscing in my own house. And what has happened in this Inquiry is that I've actually read all the papers and begun to remember more. It's quite -- it's really very difficult, you know, remembering back 1983, 37 years now. Then it would have been 32 years, I think.

So I think what I would say now, in memory, is that my -- most of my four days -- I would say 80% of my four days was spent looking at information and analysing information. I wasn't -- I didn't have clinical

responsibility in the sense of decision-making, but I was in the department, I was a qualified doctor, and I had an honorary contract. So if there was a problem, I could be asked to see that person.

The main clinical care, the first person of contact, as I explained yesterday, was the junior doctor rotating through the department who was a senior registrar in haematology, and he or she was there to train in haemophilia care. So if he or she didn't know anything, or had a question or a query, who would they come to in the department who knew something about this strange condition that was coming? Who in the department would they ask about the hepatitis? The hepatitis, by the time -- and I think I explained this to you yesterday -- the study was from April 1978 to March 1983, and I actually took up my job at the end of January '83. And as I recall -- and I'm just recalling, trying to remember 37 years ago -- I think there were about two patients left who still required to be seen every two weeks to take their blood sample.

So I hope that answers your question.

Q. Just two follow-up questions, if I may. The first is, in your answer a moment ago you posed a question, a rhetorical question: the senior registrars who would be seeing patients --

A. Sometimes it was a registrar. It wasn't always a senior registrar.

Q. -- and you posed a question: if they needed to speak to someone or discuss something, who would they ask? Is the answer to that question that they might come and talk to you about this strange new condition or about hepatitis?

A. They might come and talk to me, but clearly the -- and I tried to explain this yesterday. The Haemophilia Centre worked as a multi-disciplinary team. It had to because there was so much work coming and so many disciplines that we interacted with. You know, there were -- as I said yesterday, there were the nurses, there were the counsellors, family therapists, there were the junior doctors, there was the physiotherapist, there was the people that we interacted with, like the orthopaedic surgeon, the liver doctors, and later the HIV.

So when I say they came and asked me, they came and asked me probably primarily, but clearly the person who had the clinical decision-making around that patient was Dr Kernoff.

Q. Yes, and I'm not trying to requite your role in any respect with the role of Dr Kernoff. I'm just trying to understand roughly -- and I know I'm asking you

about events a long time ago -- how much time you spent seeing patients, because your answers here suggest that you did at least spend some time with patients.

A. Yes, I just said. I spent four days a week. I'm sorry to bore you with domestic things, but it just gives you an idea of the fact that I was semi-part-time. I used to deliver my children to school. I used to get to the Royal Free at ten o'clock in the morning, Monday to Thursday, and I used to leave at five o'clock.

So, you know, that's not a very big clinical responsibility. And I had a desk in the corner of one room, and I spent most of the day sitting at this desk looking through notes, looking at results and things. Just remember, we did not have a computer. It was quite detailed sorting through numbers and things that you had to do.

Q. You gave an estimate -- and I understand it's only an estimate -- that about 80% of your time was spent on the research work. So would it be fair to infer that approximately 20% of your time may have been spent in more direct patient interaction?

A. It may have been. I think the other thing that should be recorded here is that the organisation of the

Haemophilia Centre -- I did say yesterday that Dr Kernoff was really rather ahead of his time, having a management committee that managed, but what he also did -- we had lunchtime meetings.

On a Tuesday, there was a lunchtime meeting of appropriate people to discuss -- it was called a social work meeting. But essentially, it was to discuss the needs and requirements of -- as the time went on, of people with, as it turned out, HIV infection or AIDS, and hepatitis or haemophilia problems. You know, people had to have cars and sorting out that. There were little children where maybe the school was being difficult, about the child keeping -- going there and keeping treatment there. And the nurses would be at that meeting. And, you know, we had a nurse that went into the community to train the parents, and that might be something that we would discuss at that meeting because, clearly, and as many people in this room will know, it's not easy injecting yourself, and it's certainly not easy for parents to have to inject children, and it takes a lot of time to do that training.

So this meeting was a clinical meeting, if you like to call it that way. So that was on a Tuesday lunchtime. And on a Thursday lunchtime, we had almost

a total department business meeting. And the important thing about that meeting was that in the week ahead, we would be seeing people, and the laboratory, particularly if we had -- you know, we were having new referrals all the time, so somebody had a bleeding disorder in some small district general hospital in Essex. That haematologist would refer that patient for investigation in our centre.

So this lunchtime meeting on a Thursday, we would have a sheet of paper telling us what was happening next week because the laboratory had to do these tests, and we had to make sure that they weren't going to have a problem with the tests. And we also needed to know whether everything was organised for the clinics.

So it was a total comprehensive care, and a whole team were involved, not least the receptionist. You know, there was no computers, so she's got a book where she's having to book people's appointments and know who's coming or is not coming, and there are telephone appointments. So it's very difficult to -- it wasn't like -- you know, I think probably you may have a sort of idea of a regular outpatients, you know: the doctor sitting in a room and receptionist here. It wasn't like that at all. The centre was self-contained with the whole team in it, and everybody knew what was going on.

Communication was essential. We had a little ward area where people were seen. There was a children's room. There was a room where the family therapists could talk to patients. So I don't know if that helps.

Q. And where physically was Dr Kernoff located --

A. He had his own room.

Q. But as part of the -- (overspeaking) -- self-contained centre --

A. And ultimately, he had -- we all -- he had his own room in the centre. I explained yesterday, in 19 -- the last thing he did before sadly he had this huge myocardial infarction, he had got agreement from the hospital to have an extension built, and that was paid for by what was called the Katharine Dormandy Trust, which was money that Katharine Dormandy had put in trust that was given mostly by a property development developer who was the father of somebody who had severe haemophilia. And that agreement was there.

So after that extension was built, the extension -- I think I said this yesterday -- the extension provided consultant rooms, a room for the family therapist, and the notes were put in a huge area in filing cabinets, and the secretaries would sit between the filing cabinets. And at that point -- Dr Kernoff wasn't there any more, clearly. I had

a room at the back of the centre, a windowless room, and I moved to the front.

Dr Kernoff's room became the children's room, and it was set up as a special room where children could be treated. That's going forward to 1994.

Q. Yes.

A. But the period you're talking about, 1983, he had his own room.

Q. Thank you. I wanted to then move to the question of the risk posed by AIDS to haemophiliacs. You've said in your witness statement, and indeed in your Lindsay evidence, that your first awareness of this may have been a Lancet article in April 1983.

A. Sorry, what?

Q. I'll find the reference in your statement for you, Professor Lee. So if you look at paragraph 37 of your statement. It's page 28. Henry, it's WITN0644058. And if you could go to page 28, please, Henry. Bottom half of the page. In answer to the question "How and when did you first become aware that there might be an association between AIDS and the use of blood products?", you refer to your answer in your evidence to the Lindsay Tribunal, and then effectively you replicate that answer, so:

"As far as I was aware, the first indication of the

transmission by blood transfusion of what later became known as AIDS or HIV was a publication in The Lancet on 30 April."

Then you say you don't think you were aware of the publication by the MMWR of 10th December 1982.

Can I just look with you at a couple of documents between December 1982 and 30th April.

Henry, could we have PRSE0002410, please.

This is an article in the New England Journal of Medicine, 13th January 1983, "AIDS and Preventive Treatment of Haemophilia", and it's by Jane Desforges. And I'm fairly confident you're familiar with it because you cited it in later articles that you wrote.

Did you read this at the time, Professor Lee?

A. I think I must have done but I don't remember.

Q. We know also, if we have on screen, please --

A. I think this is January 19 --

Q. 83.

A. Yes. I think I'd like to go back to something you said at some point, maybe it was questioning Dr Colvin, but you rather suggested that everybody knew that AIDS was a problem in January 1983, and I don't think that is true. It is certainly true that there were cases of AIDS that had been reported in people with haemophilia, but there was still debate

about whether those cases were a problem because of the immunological damage -- that concentrates had come and made some people more sensitive to AIDS.

To go back to this paper you're referring to in The Lancet, which was actually July 16th, 1983, if that's the one you're referring to?

Q. No January 13th, 1983, the Desforges article.

A. Okay, sorry, that's this one. Okay.

Well, I just want to go back on my comment about -- there was a suggestion that, you know, it was all cut and dried, that we all knew that people with haemophilia had got AIDS from treatment. It is certainly true that there were people with haemophilia who had got AIDS, but it was still being debated about whether the immunological change in some people had made them more sensitive to getting it.

And in the original presentation of HIV, people had what was called a lymphadenopathy syndrome, and if you go back to this paper, my paper, that was published in May 1983, that -- those data I presented in a session at the World Federation of Haemophilia in Stockholm in 1983, and there was an emergency meeting in the lunch hour, an hour's meeting of haemophilia treaters from all over the world, and of course patients were there, because the World Federation of Haemophilia has

treaters, patients and doctors, and there was a huge discussion about lymphadenopathy syndrome.

When these data that I presented were shown, we had shown that people who had haemophilia A had immunological change, and that's the change -- at that time how we measured it was T4/T8 ratios. They're now known as CD4 counts, the T4 count. And when that became low, that was -- represented immunological depression.

The people with Factor IX deficiency did not have this in our patients, and we didn't know why that was, and the suggestion was it was the manufacturing process of the Factor IX. And in fact there had -- so this is June 1983, these data are being presented in a room of international haemophilia treaters. There was still debate in that presentation, because I only had two slides -- everybody was asked to bring slides so that people could understand what was happening.

So there was still debate going on about this immunological change, and certainly, in the patients themselves, they presented with immunological change.

The other thing I would like to comment on -- I think you commented on it yesterday, or maybe it was when Brian Colvin was giving evidence -- but there was a paper by Armen(?) where a child had got HIV from

platelets. That paper was passed around numerous journals and they turned it down. The head of the Transfusion Service in America actually wrote a leader, and I don't have it at hand now but I can certainly pass it on to the Inquiry afterwards, where he disputed any notion that blood transfusion could transmit AIDS and it wasn't until a paper was published in the New England Journal 1984 that definitive proof came that blood transfusion caused AIDS.

And I just -- I'm sorry to go on about this, but it's important. If you would like to get -- can I do this? Can I interrupt this?

Q. Certainly but I do want to take you back afterwards, please, to January of 1983.

A. Yes, that's fine. That's fine. The paper is -- I think this is your number, 644090.

Q. What's its called, please, Professor Lee?

A. It's called "Relationships between blood product exposure and immunological abnormalities".

Q. No, it's 06 -- so WITN0644064, please.

This is your reach with Dr Rizza; is that right?

A. Yes.

Q. Yes, we are going to come on to this, but please do make the observation you want to make.

A. What I just want to make the point is if we turn to

page 170, which is the last page.

Q. The page before that, please, Henry.

A. This a discussion -- actually, before I say that, this paper was received by the British Journal of Haematology in June '84. It was accepted on 17th October 1984 and published in May 1985. But what it's discussing here is that immune suppression produced by repeated exposure to clotting factor concentrates lowers the threshold for infection. It's throwing that out. Now, at the time this was written, the knowledge was accruing, but I can tell you that in 1983 there were big debates around lymphadenopathy syndrome.

So there was an emerging experience but it certainly wasn't cut and dried.

Q. Yes, I think -- Professor Lee, I don't think I've ever put to Dr Winter or Dr Colvin that anything was cut and dried in 1983; the proposition I put to them, which they assented to, was that by January 1983, clinicians involved in the care of patients with haemophilia should that have realised that there was a significant risk that blood products might transmit AIDS.

Would you disagree with that proposition?

A. I think "disagree" is probably a too strong a word.

I don't think it was cut and dried.
Q. Would you agree that by January 1983, there was evidence that blood products might transmit AIDS?
A. There was some evidence certainly. If you -- you know, if you have people with haemophilia having this strange syndrome.
SIR BRIAN LANGSTAFF: I don't know if it's any help but, in either your presentation on the knowledge of risk or on Oxford, you told me that in December 1982, Rizza had observed that all HCO directors knew that there was a real risk that AIDS could be a transmitted by an infectious agent carried by blood products.
That appears to have been his view at any rate at the end of 1982, if that helps.
A. I think -- I think what ...
If you turn, on that same paper, the Rizza paper, to page 168, it's the beginning of the discussion, and I'm sure we're coming on to this because you're going to be discussing the document:
"The recognition that many asymptomatic patients have laboratory abnormalities resembling those detectable in AIDS has raised the possibility that these patients may have a condition representing a prodromal or forme fruste of AIDS ... Whether or not this is the case, it seems beyond doubt that the

abnormalities we and others have found in haemophiliacs without AIDS are caused by exposure to blood products."
What that's telling you is that our experience at that stage was that a large number of people were demonstrating immunological abnormalities, but whether they were all going to go on to developing a form -- real AIDS, was debatable, and people were debating it. It's a bit like saying whether everybody who got non-A, non-B was going to get liver cancer.
And I think I would also like, while we're on that page, so that we don't -- just to give you an idea of the people here, the patients -- can we?
MS RICHARDS: Yes, of course.
A. So this was our experience at the time that this paper was submitted, in June 1984. This was clinical experience of the patients. So we had patients with lymphadenopathy, the lymph nodes, which is this idea of a lymphadenopathy associated syndrome, we had somebody with an opportunistic infection, somebody who had lost weight, somebody with septicaemia and another one with herpes zoster. What that is showing us is that there is undoubtedly immune abnormality in these patients, but as the knowledge was emerging through that time, it wasn't entirely clear whether this was

going to be something that transmitted into full-blown AIDS. We didn't know.
Q. Before we return to January 1983, whilst we're on this paper, Professor Lee, is it then your evidence that as at June 1984, when this paper was submitted to the British Journal of Haematology, you and your co-authors had still not reached the conclusion that what you're describing here was most likely to be AIDS?
A. I think you could say that at that stage.
Q. We'll come back to --
A. I just would remind you again of the patient that I saw in 1983, the elderly gentleman. He died at home. His chest X-ray was complete whiteout. And in retrospect there's no doubt that that was Pneumocystis carinii. So that was AIDS. The other thing that, you know, was happening at this time, we didn't have a test, so the definition of AIDS was very vague, in a way. There were these -- lymphadenopathy, a wasting syndrome, opportunistic infections like shingles, that is herpes zoster, septicaemia, and those things were added up and saying: well, you know, this looks like immunodeficiency, this looks like AIDS. But it wasn't really we got a test that you could definitely say that somebody had got HIV

infection and AIDS, really.
Q. If we return to January 1983, could we please have on screen PRSE0002647. These are a notes of a meeting attended by a number of Haemophilia Centre directors, not attended by you, Professor Lee, including Dr Kernoff, on 24th January 1983.
And to provide you with some context, it was a rather unusual meeting, it took place at a hotel at Heathrow Airport.
A. That is not unusual, because many meetings were held at airport hotels because it enabled -- you know, because haemophilia was rare -- this not in the context of this meeting, but many meetings were held, of European treaters or American treaters, they would fly in to a meeting and fly out. So having a meeting at a hotel is not at all unusual.
Q. If we can just turn to the last page we can see who was there. If you very quickly cast your eye down the list of names, you'll see it involved the reference centre directors at the time, including Dr Kernoff, but also a number of other Haemophilia Centre directors who were not reference centre directors, together with Dr Craske and Professor Zuckerman, and the author of these notes is Dr Boulton, we think, and then there were representatives from Immuno.

I'm not going to trouble you with the first part of the meeting minutes, just the second part, which looked at AIDS.

Can we go to the previous page, please, Henry.

So you'll see under the heading "Acquired Immunodeficiency Syndrome", there's a summary by Dr Craske of the current position. There's a description two paragraphs down of possible clinical features, including herpes viruses. And then it says:

"Up to 10 December 1982, some 800 people had been reported as suffering from the AIDS, and there was a 45% mortality."

Then it says this:

"Ten haemophiliacs in the US have been affected, five have died. The youngest was aged 7. All cases have had prolonged treatment with Factor VIII, but there is no specific implication of one particular product or batch. Other cases involving blood and blood product transmission have included platelets transfused in three cases. In one of these cases, one of the donors was a young New York man in his twenties. A second case was a 20-month year old child with Rhesus HDN who had received several units, including platelets known to have come from a homosexual donor ..."

A. That case was the case I was talking about.

Q. The San Francisco baby case --

A. When it was written, up, the paper itself, I think this is very clearly shown in the book called And The Band Played on by Randy Shilts, where he really exposes the transfusion head-up in America, but that paper was sent around various journals and turned round because people didn't believe it.

Q. Yes, but --

A. I'm saying that because there was debate. You know, people who reviewed papers are experts.

Q. The child, it's described, has developed a possible AIDS case. That's a 20-month old child.

Over the page:

"The incubation period for the syndrome appears to be six months to two years.

"In the UK, so far only one or two cases have been reported ...

"The infectious precautions include ..."

Then the various protocols in relation to donation are discussed.

Then:

"The attention of the meeting was then drawn to the two articles on the editorial in the New England Journal of Medicine ..."

We've looked at one of those.

And reference there expressly to the T4/T8 ratios, and issue about donor pools.

Now that's an update, as at 24th January, about cases in the States, including the case of the baby that you've referred to. Was that information, as far as you can recall --

A. I think you've missed out the critical line in this, actually.

Q. Which line is that, Professor?

A. The second line:

"In the UK, so far only one or two cases have been reported from the communicable diseases centre."

I presume that's talking about haemophilia people, with AIDS.

Q. No, not at that stage. And I don't think I did miss it out. I think I read it out, Professor Lee --

A. Okay --

Q. In any event -- no, not haemophiliacs at that stage in the UK.

A. Okay, well, it was during that year.

Q. Yes, it was. Would you agree that the information set out there about the numbers of cases in the States and the cases from platelet transfusion was significant information which would support the idea of a link between transfusion and AIDS? Not prove but support.

A. Well, I think -- I think, probably, yes. It's not proof.

Q. Did Dr Kernoff report this information at one of the meetings you have described? This is about the time you're joining the Royal Free.

A. This is January 1983. I'm not even there.

Q. 24th January 1983.

A. Yeah, I'm not there. I said the end of January I started there.

Q. Yes, forgive me Professor Lee --

A. I also don't -- I really don't think it reasonable to ask me about a meeting in 1988 -- what is it --

Q. January 1983 --

A. -- that I wasn't at.

Q. I'm trying to show you, Professor Lee, the state of knowledge as at January 1983 that would have been shared with Dr Kernoff, and the question is: was that information that was disseminated by Dr Kernoff amongst the staff at the Royal Free?

A. I have no idea. And what I would also say to you is to go back to -- I would like to tell this Inquiry about things I know about. I don't want to tell this Inquiry about what Dr Kernoff was thinking. I don't know what he was thinking. But what I do know what he was thinking when I was working with him.

And I would go back to The Lancet paper in May 1983. And what I can remember -- and this is a very clear memory -- is before that letter was sent to The Lancet, he had a very clear discussion with me about what this means. That's after this paper. So what I'm trying to get over to you is that that was not the only view. I would also -- it was emerging experience.

Q. What discussions, if any, did you have with Dr Kernoff, or Dr Kernoff have with you, about AIDS in the first half of 1983?

A. We were continually talking about it because we were trying to understand what was going on.

Q. So it's like --

A. But what I'm saying to you is the idea that everybody knew everything at that stage is wrong. And I'm trying to also -- you know, the memory, the really powerful memory I have, mainly because it was the first international meeting I'd ever been to in my life, was this meeting in Stockholm where it was an ad hoc meeting. It wasn't even in the programme. Because people were tying to report their ideas. And these data were presented there and accepted. And people were still discussing: is everybody going to get it? There was no doubt that some people had got

it, but it -- (overspeaking) -- certainly not clear that everybody was going to get it.

Q. Professor Lee, at no point have I suggested to you, or to any witness, that everybody knew everything in 1983, in January 1983 -- (overspeaking) -- or that everyone --

A. Well, I'm getting the feeling that that's what you are suggesting.

Q. No. I'm trying to understand, and ask you to explain, what discussions took place in the early part of 1983 at the Royal Free Hospital. You are one of the few witnesses that we can ask that question of. If you cannot remember, then you cannot remember.

A. All I can say is, I sat at this desk in my room, and I'm looking at this information and trying to analyse it, talking with Dr Rizza maybe on the phone occasionally. And Peter is in his room down there, managing the department, and he clearly would come and see me and discuss things and, "What's the findings? What have you got here?"

So, insofar as that, we were discussing AIDS. He also -- as I said yesterday, he had this collection of references, this filing cabinet that he -- anything that was a reference, he would stick the title, or have the title stuck on a file card so that he can access

references. So he would say, you know, "You ought to read this." So, you know, we were discussing it.

But I would also like to emphasise again, 37 years ago, you're trying to ask me what conversations I had with Professor Kernoff, Dr Kernoff. And as I said, I have this very clear conversation about the data that was going to be put in two slides in Stockholm because, you know, this was a memorable thing for me. And he was quizzing me on, you know, "Are we sure? You know, what's this all about?" And we were kind of batting ideas around. You know, that's the kind of thing we did.

Q. In the course of 1983, the UKHCDO Reference Centre directors met. Information was sent to directors by Professor Bloom and Dr Rizza. Information was sent to Dr Kernoff, and he was part of their decision-making process.

Was that information that, as a matter of general practice, Dr Kernoff would circulate amongst the department or summarise for the benefit of the department?

A. Well, we did have -- every Thursday morning for an hour, we had what was called a teaching session, and so that would be the kind of meeting where information was presented. So the laboratory might be talking

about a meeting they'd been to, and they'd give feedback of new tests or something. And it might have been something he might have discussed then, but I don't recall. And I think, also, it's a little unfair asking me about meetings at which I wasn't at.

Q. I wasn't asking you about meetings at which you weren't at then, Professor Lee. I was asking you what information was shared by Dr Kernoff with those at the centre.

A. About a meeting at which I wasn't at.

Q. No, about the advice that was provided by UKHCDO Reference Centre Directors. Was that information --

A. I can't remember. It's 37 years ago.

Q. One of the -- I won't take you to the documents of the meetings that you weren't at, Professor Lee, but one of the things which Reference Centre Directors asked directors to do in March of 1983 was to look out for signs of AIDS and to set up a reporting system so that that information was fed back to Dr Craske. As far as you can recall, was that something Dr Kernoff asked you to do?

A. No. Don't think so.

Q. Just before we get to the World Federation of Haemophilia meeting, which I think was 27 June to 1 July 1983, could we look at PRSE000372.

This is a document from the Council of Europe's Committee of Ministers. If you look at the heading, it was:

"Recommendation of the Committee of Ministers to Member States on preventing the possible transmission of acquired immune deficiency syndrome (AIDS) from affected blood donors to patients receiving blood or blood products".

And you can see from the italicised bit in brackets, it was adopted by the Committee of Ministers on 23rd June 1983.

So this was just a few days before the World Federation of Haemophilia meeting in Stockholm which you attended.

If we go to the second page, please. Could we zoom in on the top half of the page? This is a recommendation at national level from Europe to Member States, and you may not have seen it at the time, Professor Lee, but you'll see it says this:

"Recommends the governments of Member States take all necessary steps and measures with respect to AIDS and, in particular, to avoid wherever possible the use of coagulation factor products prepared from large plasma pools. This is especially important for those countries where self-sufficiency in the production of

such products has not yet been achieved."

Then the second is:

"To inform attending physicians and selected recipients, such as haemophiliacs, of the potential health hazards of haemotherapy and the possibilities of minimising these risks."

So you'll see there, Professor Lee, that a few days before this World Federation meeting in Stockholm, the European Committee of Ministers recommended the avoidance, where possible, of the use of coagulation factor products from large plasma pools, and that not just doctors but also haemophiliacs, patients, be informed of potential health hazards of haemotherapy.

Was that something, as far as you can recall, that was discussed at the World Federation of Haemophilia in Stockholm a few days later?

A. I don't recall it.

Q. Your evidence to the Lindsay Tribunal was that there were no changes to the approach to treatment at the Royal Free Hospital in 1983 or 1984, save that in 1984 there was at some point adopted, and you couldn't recall when, a policy in relation to deferring elective surgery.

As that was your evidence to the Lindsay Tribunal, as you've said when events were still fresher in your

mind, is that likely to be correct?

A. I think that probably is correct, yes.

Q. So --

A. I think also, as I said yesterday, the evidence I gave to the Lindsay Inquiry 20 years ago, I was a working director of the Centre and Professor of Haemophilia, and I actually had access to documents or treatment records, and so I think it was accurate. But again, I would emphasise that, you know, I wasn't in the business of the decision-making process through that period.

Q. The discussion that took place that you refer to, the debate at the World Federation of --

A. It wasn't a debate; it was people from all over the world. It was an hour at lunchtime. It was chaired by Dr Margaret Hilgartner, who was -- she's died now, but she was a famous paediatrician in New York who had looked after people -- children with haemophilia.

And before the meeting -- and this request would have gone to Dr Kernoff, not to me. He didn't go to the meeting. A request was made that if you had information to share, would you take information on no more than two slides. And the reason for that is, again, I would pick up a point that I made yesterday. You know, the dissemination of information now is

instant. Then, it was very, very slow, and so -- and you may pick up on this in a later discussion about London. You know, meetings were instant, and that was where the information was being shared. So it wasn't a debate; it was a presentation. And what I was trying to say -- that there were different views presented.

Q. Can you recall whether there was a majority view that the condition was likely due to an infectious agent?

A. It wasn't -- there wasn't a vote taken or anything. What I do remember -- one, I remember I got my slides upside down, which was -- which is perhaps why I do remember it. But I also remember, right at the back there was a Canadian physician -- I think he was called Tsoukas, T-S-O-U-K-A-S, and he talked at great length about lymphadenopathy syndrome, and that was quite a dominant part of that meeting.

Q. Can we move on to 1984 and a document that you authored. Could we have on screen, please, Henry, WITN1003018, please. This is a Haemophilia Centre publication: "Haemofact. AIDS. Release number 3". It says:

"This fact sheet contains important information concerning AIDS. 11 May 1984."

If we go over the page, we can see that the leaflet

is authored by you. Can you recall how you came to write this? Who asked you to write it?

A. I think -- I don't know, but I think the -- certainly, the request came from The Haemophilia Society. And I would imagine it must have gone to Peter, because they wouldn't come directly to me, and that Peter passed it on to me. And it's certainly -- what I wrote would have been reviewed by Peter.

Q. As far as you can recall, were you given any kind of brief from The Haemophilia Society as to the kind of message they wanted to convey?

A. No. They wanted information.

Q. If we look at what you wrote here, you said:

"The occurrence of AIDS in haemophiliac patients has strongly suggested transmission of disorder by blood products. Epidemiological studies have suggested it may be related to a transmissible agent."

Then you make reference to scientific developments in the States and in Paris. You say this:

"These reports should be received with optimism. The obvious benefits from such findings would be the provision of a blood test for both affected persons and donated blood and, in the long-term, the development of a vaccine."

You then say:

"In Great Britain, the number of haemophiliacs who have been reported with AIDS remain at two. Thus, the incidence is less than 1 in 1,000 patients at risk."

I'll come back to that. You say:

"The relationship of immunological abnormalities found in many heavily treated haemophiliacs throughout the world is uncertain."

Then refer to studies in Scotland, Australia and America. You say:

"It's possible that the immune suppression produced by repeated exposure to clotting factor concentrates lowers the threshold for infection with a putative AIDS agent."

And there is then further discussion about that. And you suggest that:

"This is a function of the characteristics of the final product and the fractionation methods used to make it. Thus, prospects for resolving these problems are brighter for haemophiliacs than for other high-risk populations, since improvements in plasma fractionation are likely to make it possible to remove or inactivate causal agents from the therapeutic products."

And you say:

"The heat treating clotting factor concentrates which have been manufactured by many commercial

companies in the NHS may be an advantage."

Then you talk about:

"The really good news."

And you refer to work at the Royal Free, Speywood, and in San Francisco about the synthesis of the gene -- sorry, the cloning of the gene for Factor VIII.

Now, it could be said -- and this is what I'm inviting you to comment on, Professor Lee -- that this is an article which has a rather upbeat, positive, reassuring tone. Was that what it was designed to convey: to reassure patients?

A. No. It was written to convey what the knowledge was at that time. And the number of cases of AIDS in people with haemophilia in the UK at that date, at that time, was two, and this had come from the Oxford records, as you said. The people had to let Oxford know for the database -- not the database; the records.

And when I write in here that the number at risk was 2,000, this was the number of people in the UK who had been treated with clotting factor concentrate at that time. So the incidence was 1 in 1,000.

And in terms of the immunological change, I think I would like you, if possible, if that's allowable, to pull up a paper. I'm afraid the number I've got is my

witness statement which is 655086 --

Q. -- (overspeaking) --

A. -- and this was published in 1996.

Q. Is that comparison of immunodeficiency -- (overspeaking) -- conditions?

A. Yes.

Q. Yes, it's WITN0644086.

A. I think the reason I want to just put this up is because the discussion around immunological abnormalities caused by blood transfusion and concentrates was quite significant in the time period that we're talking about. And indeed, it didn't go away in North America. There was a Professor Duesberg who was still promoting this idea, and we had to publish a paper in 1996 to dispel it. What we did was we compared a group of our patients who were HIV negative -- we had a test then -- and group who were HIV positive, and it was quite difficult -- it was quite difficult, sadly, to find number of HIV negative people who had had a large amount of treatment at that stage, but we did manage to find it so that the two groups could be compared. And we showed that, you know -- I'm sure we would all think that that was what would happen, that people who were HIV positive were the people who got AIDS and the

people who were HIV negative didn't.

Even so, when you read -- I don't want to prolong this here now but if you read the paper, this Duesberg is still saying -- but even so, you know, he's not believing it.

So this is published in the BMJ, so it's been subjected to peer review, so the BMJ are not going to publish a paper about something that is not an idea. And that's 1996, when we've got tests for it. In 1983 we didn't have any tests. So I don't think I should be representing somebody who is putting rose-coloured spectacles on what was a terrible health problem.

What I was doing in this pamphlet was stating the facts and the view that was held at that time.

Q. We know that --

A. And, you know, I would add that -- you keep asking me what Peter -- Professor -- Dr Kernoff thought, and did I have discussions with Dr Kernoff. He certainly would not have let this go to the Haemophilia Society without, you know, looking at it.

I would also, you know, go back to the earlier discussion we had about the paper, looking at immunological abnormality, of these strange opportunistic infections and lymphadenopathy that we were seeing, but it was still discussing about whether

it was immunological or whether we were going to see AIDS, or whether those people were going to go on to AIDS. It was still not clear.

And the other thing that's quite interesting here -- I mean, there is such a parallel with Covid-19, isn't there, because we're talking about there may be a vaccine. Well, of course there never was a vaccine because it's a carrier state. But we never had any idea of that then. So I don't think -- you know, people now, with Covid, are talking about we'll have a vaccine by Christmas maybe. We don't know. And I really do think it's important to try to understand that -- it's very easy with -- looking retrospectively, but it's very difficult to remember the confusion, the lack of knowledge, at that time.

Q. Do you think there might have been a risk that those reading this material might have felt reassured from reading it that very few people were likely to develop AIDS and that it was safe to continue taking factor concentrates?

A. Well, I guess, on the face of it, there may be. But that -- you know, that ...

Because you present facts as you know it, and that's how they're interpreted, I don't think one should massage facts, you know. These are facts.

There are two people in the UK who have got AIDS. There are 2,000 people who have been treated. Therefore, the incidence is 1 in 2000. And then I go on to talk about the immunological changes, which undoubtedly there were. We were sitting there recording them. People, because their lymphocyte count went down to less than a thousand, they got these strange things -- like shingles and streptococcal pneumonia in a young man was, you know, unheard of, but if you've got immunological abnormality it's there. And so we were learning. We were so learning.

Q. Telling a lay audience, in the sense of non-clinicians, although no doubt many of those reading this were experts in their own care, as haemophiliacs, but telling a lay audience that the incidence is less than one in 1,000 patients at risk, the number of haemophiliacs remain at two, may be factually correct, but what you don't go on to set out here is what the risk might be from factor concentrates of others, having transmitted to them what was, by now, surely thought likely to be, if not proven to be, yet, a transmissible infectious agent?

A. I would take issue with you. Patients with haemophilia are pretty expert because they go to

meetings, they have bulletins from the Haemophilia Society, so, you know, there was a lot of knowledge circulating around, so I think it's a bit patronising, really, to -- well, not patronising, but it's -- of all the patients you have, they're probably the most educated about their condition, because it's so rare, it's lifelong, and so they learn about it and they get information from the Haemophilia Society.

Q. Yes, absolutely right about their bleeding disorder. They wouldn't be experts in AIDS or viable transmission --

A. But they were getting as much information as was coming out by the Haemophilia Society. That's why they produced this bulletin: to send stuff out. They were sending anything that came their way about this strange condition.

Q. Can we just look before we break at one further document, which is the haemofact that followed up this. It's at WITN1003019. This is 24th September 1984, so the next release.

And this was at the time the Haemophilia Society's chosen method of communicating information to its members, through these leaflets.

This is authored by Dr Kernoff, if we go to the next page, and I'll just ask, if we go to the page

after that, please -- so he talks on the first page about the identification of LAB and HTLV-III, and then at the top of the page he says:

"The presence of antibodies is usually taken as evidence of immunity to infection, and perhaps one reason why the risk of AIDS in haemophilia is so low (around 1 in 1000) is that many patients are immune to it."

Now, he's not there talking about incidence; he's talking about risk.

A. Well, I think that is incidence. That's the same figure I had. I mean, I think you're playing with words.

Q. Words can be rather important, Professor Lee, when they're the means by which patients understand if they're being exposed to the risk of a fatal condition.

A. All I can say is that, in this context here, he is meaning incidents. It's the same, you know, the risk of AIDS. You know, those are words he crafted but he is meaning this as incidence, one in a thousand.

Q. He says in terms:

"... the risk of AIDS in haemophilia is so low ..."

A. Yes.

Q. What's the basis upon which he could reasonably say

that, in September 1984 --

A. Because --

SIR BRIAN LANGSTAFF: I think I've got that. What he's saying is we have seen two cases, or something of that region, of AIDS, despite the number of haemophiliacs. I think you're right, Professor, that he uses the word "risk" where he ought to have used the word "incidence", and it may be, it's a matter of comment and a matter of conclusion for me, that -- whether I come to the view that, really, the risk of AIDS is something very different from the incidence of AIDS. And it may be that somebody reading it without having, as it were, a dictionary to one side, might have said, "Well, the risk to me is very, very small."

And that might be the way that somebody might read it, I suppose. But that's a matter for me ultimately.

It's time for a break, I think.

PROFESSOR LEE: Before we go for a break, can I just comment on what's written here?

SIR BRIAN LANGSTAFF: Yes, of course.

PROFESSOR LEE: I mean, what is interesting is -- the presence of antibodies is usually taken as evidence of immunity to infection, and perhaps one reason why the risk of AIDS in haemophilia is so low is that many patients are immune to it. When we come -- well,

I don't think you'll be questioning me on this because I never gave results out and I think you have a statement being made by Dr Eleanor Goldman, who was a specialist in the centre -- and it's my understanding, I think I put this in my statement, that when patients were given the result of their HIV test, that some of them were -- or they've maybe all been told, I don't know because I didn't do it, but that they might be immune. And of course now we know that there's a carrier state. So it doesn't mean you're immune.

SIR BRIAN LANGSTAFF: It may be thought it's something paradoxical about it, there being a disease of the immune system or a disease which results in immune misfunction which also produces an immunity to something else, but that's for others to perhaps consider, and perhaps for me to consider. But let's take a break, shall we? And let's come back at ten to 12. Ten to 12.

(11.08 pm)

(A short break)

(12.00 pm)

MS RICHARDS: Professor Lee, I want to ask you next about some correspondence you had with Dr Rizza and some work you undertook with Dr Rizza in the course of

1983. If we have up on screen, just to put this in context, WITN0644065. If we just zoom in a little bit on the text, please, Henry. This a letter from The Lancet July 16 1983, and it's authored by you and others at the Royal Free.

It talks about a study of 64 patients with haemophilia A. And as I understand it, this was the examination of the T cells, in the way that you've referred to already, and consideration of whether fractionation methods were a cause. So a somewhat crude summary, but is that right?

A. Yes.

Q. At this stage, the patients that you had looked at were all Royal Free Centre patients?

A. Yes.

Q. If we could have on screen, please, 0XUH0002971_006. We can see this is a letter which you wrote to Dr Rizza in July 1983. It refers to a telephone conversation the two of you had had, and then you set out in writing the plan. And you talk about how the technician will receive specimens, and you suggest it would be helpful if some serum could be stored for future use. And then you say in the concluding paragraph:

"I look forward to receiving the specimens.

I think it will be a very valuable contribution to have these results in a population of patients treated with non-commercial Factor VIII."

Could you just explain what it was you were asking Dr Rizza to do and what the two of you had discussed?

A. I already talked about this. Basically, that letter that you just put up, in our patients we had found that patients treated with Factor IX concentrate did not have abnormalities, but the patients treated with Factor VIII concentrate did. And our speculation in that letter was that we thought it was the manufacturing process of the Factor IX and -- which was different to the Factor VIII. But there remained the possibility that it might be something to do with the donor pool.

And although we had all our patients with Factor IX NHS concentrate because they're a smaller number -- they were all on NHS -- our patients with Factor VIII deficiency had a mixture of commercial and NHS.

So what we wanted to do was to expand the population of patients with Factor VIII deficiency who had NHS concentrate so that we could compare the two.

Now, Oxford had a long tradition of making concentrates, and they had tended to keep more of their patients on -- the fractionation moved from Oxford to

the fractionation plant later. But they had a tendency all the way through to keep more patients on NHS concentrate than we did. So the idea was to expand the number of patients with Factor VIII deficiency who'd been treated exclusively with NHS product and compare the two.

Q. So an arrangement was made between you and Dr Rizza that he would take samples of serum from his patients, and those would be sent to the Royal Free, and then tested in the Royal Free's laboratories; is that right?

A. That's right. And the comment here, "Our technician here feels he can cope with five or six specimens", that was because the immunology laboratory, it's quite -- it was quite an intensive test to do. And I think they were sent by a car to the Royal Free because the other issue about these T4/T8 ratios was that it was important that the blood was tested as quickly as possible.

Q. Before we look at the rest of your correspondence or some other bits of your correspondence with Dr Rizza, could we have on screen OXUH0002245_007.

This is a letter not sent by or to you, Professor Lee; it's from Dr Rizza to the Regional Medical Officer within his own health authority. And

it's May 1983. You can see if you look at the last sentence of the first paragraph, he's saying he thinks it's important to act quickly to set up screening tests to detect patients who might be at risk of developing the full-blown condition. And then he goes on in the next paragraph to talk about the fact that Oxford has many patients who have received only NHS Factor VIII and says:

"It should be possible to find out if patients on NHS concentrates are immunosuppressed to the same degree as those on US concentrates."

So was the work that you and Dr Rizza were doing in any respect connected with what's identified here as the importance of screening tests to detect patients who might be at risk of developing AIDS, or was that something separate?

A. What was the date of this?

Q. 11 May 1983.

A. I think it probably was to do with our tests.

Q. Did you see the tests that you were doing in any respect as a means of detecting people who might be at risk of developing AIDS?

A. To the extent that we were considering whether the immunological changes were going to lead on to AIDS, yes. We had this discussion before the break. It

would be helpful, but we didn't really know for certain. And, you know, we at that stage were still speculating whether it was the manufacture of the Factor IX that had made the ratios normal, compared to the manufacture of Factor VIII. But clearly, another possibility that was being considered then was whether it was the donor pool.

Q. In terms of those patients where -- and I'll just ask you here to deal solely with the Oxford patients, not the Royal Free patients. The Royal Free patients where you identified the immunological abnormalities, was that information, to your knowledge, shared with patients at the time?

A. I don't know. I think -- we certainly were -- it was being done as a screening test so that we knew -- we did a total lymphocyte count which demonstrated immunological damage, and you'd have a count less than 1,000. But it also -- the T4/T8 ratio allowed us to identify patients who were particularly at risk. And I can't remember whether we specifically said what the result of their T4/T8 ratio was. I suppose because we didn't entirely know, but I can't remember.

Q. Okay. Then if we go on to your correspondence with Dr Rizza, OXUH0002972, this is a letter of 3 October 1983, and it says:

"Dear Dr Rizza, I'm setting out below our plan for the joint study which we discussed at Oxford and by telephone last week.

"The aims of the study are:

1. To establish the degree of immunological abnormality in a population of heavily treated haemophiliacs treated exclusively with NHS concentrate.

2. To confirm the differences in T subset distribution we have found between patients with haemophilia A and haemophilia B."

Pausing there. That's the same study that the earlier letter also refers to?

A. Yes.

Q. If we go down, we can see various blood specimens were required. And then if you go over the page, please, Henry, can I ask you to look at the last paragraph. You say:

"I hope this covers all the details, and we look forward to the study becoming a reality. I hope you're not all worn out telephoning patients your end. If we manage to obtain this data, it should make a valuable contribution and perhaps even silence the Daily Mail."

Just dealing, first of all, with the sentence in the middle of that paragraph, "Not all worn out telephoning patients your end." What was that a reference to? Are

you able to say?

A. I imagine he actually phoned patients to ask them to come in to have blood taken, I would imagine.

Q. Then you say:

"If we manage to obtain this data, it should make a valuable contribution and perhaps even silence the Daily Mail."

What was the reference to silencing the Daily Mail?

A. I can't remember what the particular headline was, but the Daily Mail was making really awful headlines about AIDS at this time, but I can't remember the specific headline. No, I can't remember.

Q. You then -- if we go on to OXUH0002971_001, this a letter from you to Dr Rizza, 16 November 1983. You refer in the first paragraph to results for a particular set of specimens, I think. Again, they're very normal. And then in the next paragraph, you say:

"It's noticeable how normal your patients are, but apart from the problem of dose, younger patients seem to be normal in other studies, so we must be cautious and look at more patients before we can find any comment about NHS concentrate."

Then you say this:

"I do have a nasty feeling that NHS concentrate is

going to turn out safer!"

What was that nasty feeling? Can you explain that comment, please?

A. Well, the nasty feeling was that I then realised that, or began to realise, that we may maybe have a large number of Factor VIII deficient patients who have got this problem. That's what it's about.

Q. The problem meaning the immunological abnormalities, or that you were realising that it was the donor pool and not fractionation methods?

A. Yes.

Q. So --

A. Yes, so this would -- the donor pool was the problem, rather than the fractionation process. That was -- and it was that realisation that -- it was the nasty feeling, you know, that we were going to have a large number of patients with illness.

Q. So --

A. And the other issue about this is that one of the things about "younger patients seem to be normal", well, later on, we found that the normal CD4 count is 800, and early on, we began to realise that, actually, children have higher counts than adults. So that's what that comment is about.

Q. Could we then just look at the published article

briefly. We've already looked at it this morning. WITN0644064, please. So this is the article ultimately published in 1985, but we can see from the dates received on 4 June 1984, and co-authored, amongst others, by you and Dr Rizza.

If we go to the last page, please, Henry. Go two pages before that. If we see in the acknowledgments, we can see:

"This study was supported by Action Research, the Medical Research Council, the Haemophilia Society, and the Armour Pharmaceutical Company."

"Supported by" presumably means "funded by"? Is that right?

A. Yes.

Q. And what was the particular interest of Armour in funding this study; can you recall?

A. No.

Q. We've looked at this already, but if we just go back to the first page, if we look in the summary -- that's the first paragraph, please, Henry. So if we just look at the last sentence of the summary:

"Fractionation procedures used to prepare clotting factor concentrates, and the amounts of concentrate used, are more likely to be causally related to these immunological abnormalities than the origins of source

donor plasmas."

I just wanted to ask you about that conclusion, Professor Lee, because you've explained in relation to the correspondence with Dr Rizza that the nasty feeling was because the NHS results, if I can put it in that abbreviated way, were coming back relatively normal, and you were beginning to sense that it was not fractionation methods but the plasma pools that were the problem. But in an article submitted approximately six months later, the conclusion is still there that fractionation procedures are the likely cause. Why was that?

A. I think that must have been the predominant conclusion at that time.

Q. Are you able to assist us with what the basis was for the conclusion that it was -- these abnormalities that you were detecting were more likely to be due to the fractionation procedures than the donor pools?

A. Well, I think it was still going back to the -- there was a question about Factor IX. I can't remember the detail of this.

Q. Can I ask you then, next, just briefly about what, if any, information may have been provided to patients about the risk of AIDS in 1983 and 1984.

Whose responsibility within the Centre would it

have been to provide information or advice to patients about the possible risk of AIDS at that time?

A. I think probably Dr Kernoff. I can't actually remember, but I would imagine. The other thing that we had in the Centre was a -- people waited in a waiting area in the Centre. And there was a wire basket with all sorts of information put in, and you put the information from Haemophilia Society or anything that -- and that would be one way of disseminating information. But I don't know.

Q. Do you know, as a matter of fact, whether information or warnings about the risk of AIDS was provided to patients at the Centre in '83 or '84?

A. No.

Q. So you don't know?

A. I don't know.

Q. Can you recall if, whether in the regular meetings you've mentioned or otherwise, whether there were any discussions within the Centre at that time on the issue of what information to provide to patients?

A. Did you say "do I remember"?

Q. Do you remember if there were any discussions?

A. No.

Q. So you don't remember?

A. No.

Q. It appears that there was some -- Dr Kernoff contemplated some trials of heat-treated products in late '83. I just wanted to ask you to look at a couple of documents and see if it was something you had any involvement with. Henry, it's RFLT0000019.

So we can see this is an application to the Ethical Practices Subcommittee, I think, at the Royal Free. It's dated 1 December, and it's signed by Dr Kernoff. And you'll see the subject of it is:

"To assess whether heat-treated human Factor VIII concentrate or PE-fractionated porcine Factor VIII concentrate are less likely to transmit hepatitis than routinely used human Factor VIII concentrates."

Then if we just skip over of the next paragraph. In the paragraph beginning "A major effort is being made", Dr Kernoff says:

"We've been offered two commercial heat-treated products for clinical assessment. Both have been granted clinical trials exemption."

And he sets out what they were: Hemofil T and Factor VIII heat-treated.

Did you have any involvement with these trials?

A. No.

Q. Do you know if they actually happened at the Royal Free?

A. I don't think the heat-treated trials took place, but what I do know is that there was a publication of porcine Factor VIII that was published, in blood, that was authored -- I think you should probably ask Professor Tuddenham tomorrow. What I also know is that I doubt whether heat-treated Factor VIII was used at this time because quite clearly stated in one of these papers is that it says that heat-treated Factor VIII was used in the late part of '84, I think. But I think the answer should be no.

Q. Okay.

Now, we know, and you refer to it in your statement, that in December 1984, the Haemophilia Centre reference directors produced an AIDS advisory document. I'm not going to ask you to look at the details of that but it contained recommendations for the use of heat-treated products. You were asked about what the Royal Free then did at the Lindsay Tribunal. I can take you to it if need be, but your evidence was that there was a phased introduction of heat-treated products over a three to six-month period, so that would have been the first half of 1985. Can we take that as being your best evidence on that point, as it was given at the Lindsay --

A. Yes, I think it's important to remember that I left the Royal Free in -- I'd started at the Queen Mary's and at Charing Cross at the beginning of November -- sorry, did you say '83 or '84?
Q. The AIDS advisory document was December '84, so --
A. Yes.
Q. So you were --
A. Yes, so I'd started working at Charing Cross and Queen Mary's at the beginning of November '84. I do remember when I gave that evidence to the Lindsay Tribunal, I cross checked with -- I think in particular, I cross checked with Sister Patricia Lilley, who was the clinical nurse specialist who had worked alongside Dr Kernoff at that time, so that's where that fact came from. But I was then working at Queen Mary's, Roehampton.
Q. Yes, I'm not suggesting that these were decisions for which you were responsible, Professor Lee, it's simply because they were matters that were looked at at the time of the Lindsay Tribunal through your evidence.
So the position, as I understand it from your Lindsay evidence -- and it's really to ensure that this information is heard by others -- there's a three to six-month period in relation to patients receiving Factor VIII where some would continue to receive

unheated concentrate, some received heated concentrates on a phased basis, and in relation to Factor IX, what you told the Lindsay Tribunal was that it was unheated NHS Factor IX until the beginning of May 1985, then Alpha heated Factor IX for a short period of time, and then, from July onwards, heated NHS Factor IX.
And that's likely to be correct, is it, on the basis that's what you told Lindsay?
A. I think so.
Q. I want to ask you next about the arrangements that were made for the testing of patients for HIV/HTLV-III once a test became available. Your statement says the last thing you did before you left in November 1984 was to take samples from all the patients and send them to Professor Tedder. As I understand it from what you've said elsewhere, Professor Lee, what was actually done was that the stored samples were used; is that correct?
A. Yes.
Q. And if we just look at the way in which you put it in the oral history project interview.
It's THOM0000001, please, Henry. And if we go to page 29, please.
If we look down about eight lines from the top, it

begins:
"And then towards -- the HIV virus was not identified until I think May or June '84 and we didn't have a test until the November of '84 and we had access in a research capacity, and I mean now it's horrific what we did but it was okay at that time -- I can't even remember -- we must -- yeah, we used gloves, but we actually decanted from our stored specimens -- because we had 600 patients we didn't know who was positive and who was negative, and we decanted the stuff into little test tubes and sent them down to Richard Tedder at the Middlesex Hospital for testing ..."
So is this right, that as a matter of fact, patients at the Royal Free, all the patients at the Royal Free, were tested for HIV without their knowledge and consent?
A. Yes.
Q. In terms of the numbers who were infected, who were HIV positive, you've told us in your witness statement, and you told the Lindsay Tribunal, it was 111 patients, I've seen reference, Professor Lee, in some places to 112; is that because there was a partner?
A. Yeah, the 112 initially -- this information was -- I think I should just expand a bit here.
Richard Tedder had a test in advance of there

being a test available. And so the reliability of that test was not entirely sure. Peter -- Dr Kernoff had obviously -- must have had conversation with Richard Tedder, and he asked me to decant samples into test tubes, and I did that alongside Dr Elizabeth Miller, who was going to take over my post. So I had no further involvement in that.
I'm sorry, I've lost the thread.
Q. You were --
A. What did you ask me?
Q. I had asked you about the numbers who tested positive.
A. Yes, sorry. The 111 and 112.
Q. Yes.
A. It was only much later that we realised that one of the 100 -- the 112th, if you like, was an individual who actually had -- he had HIV for other reasons. And not -- he had never received clotting factor concentrate.
Q. The majority of those who tested positive were those with severe haemophilia A, and I think the figures you put in your statement, and which you gave the Lindsay Tribunal, were: 101 patients who had severe haemophilia A, seven who were mild or moderate, one haemophilia B, and a possible von Willebrand's.
As I understand it, no children who had been

treated solely at the Royal Free were HIV positive?
A. That's right.
Q. And that is presumably because they had been treated solely with NHS concentrate?
A. Yes. And the individual -- one individual with -- who'd been treated with Factor IX concentrate, it was unheated NHS concentrate that he had.
Q. And --
A. And he acquired HIV from it.
Q. That was the haemophilia B patient?
A. Yes.
Q. And that was in the course of the first part of 1985?
A. Yes, because the -- there was a lag in time between the BPL treating -- heat-treating Factor VIII concentrate and Factor IX. I -- my memory was that -- I may be wrong but it may not have been until August of '85, but that's a --
Q. I think there's certainly one place in the papers where you've said July --
A. And I've expanded on this in my statement, that it was because -- and, you know, this is picked up in Mannucci's commentary -- that we still didn't know for certain whether heating would fix it. It wasn't entirely clear, and Dr Kernoff took the decision that the NHS donor pool at that stage was more likely to be

safer than the American donor pool and therefore he continued those Factor IX patients on NHS treatment. And it was a wrong decision, but it was made with the right logic, I suppose, at the time.
Q. You also told the Lindsay Tribunal that there were a few adults who'd been treated solely with NHS Factor VIII, and none of those were infected with HIV?
A. That's right.
Q. You may not be able to answer this, but was there any particular feature of that cohort of patients who had only been treated with Factor VIII? Was that just happenstance? Or was that a particular type of patients who only got NHS Factor VIII?
A. I can specifically remember two of them, why they did it. You remember yesterday I talked about a carrier lady who had fulminant hepatitis from cryoprecipitate, and she was a very -- she was very protective of her son and she had always insisted, although actually there wasn't necessarily a scientific reason at the time, but she had always insisted he had NHS product, and that was the way it was. And there was another patient who insisted on having it.
Q. Is it right that there were children who had been treated elsewhere than the Royal Free, so treated

I think specifically at Great Ormond Street Hospital, who had received commercial concentrates there and then had transferred their care to the Royal Free?
A. Yes, I can't -- I can't remember how many there were. I can remember specifically that there was a child whose parents had worked in the Middle East and he'd had concentrate, and so he was a child with infection. But I don't think -- you see, the children from -- there may have been children who'd been treated as children at the Royal Free -- is that what you're saying? The children who had been treated as children at Great Ormond Street who then came to the Royal Free because they'd had commercial -- yes.
Q. It wasn't entirely clear from the Lindsay evidence whether that was the position or not.
A. Yes, it was -- Professor Hardisty was the haematology professor at Great Ormond Street then, and they had used commercial concentrate, I think. But they came to us as adults. They didn't -- we didn't -- we had our own children and Great Ormond Street had their own children, and our children were treated with NHS concentrate.
Q. Whose decision had it been to use the stored samples, to send those off for testing without patients being told in advance? Would that have been Dr Kernoff's

decision?
A. Yes.
Q. It would have been Dr Kernoff?
A. Yes. And I think it needs a bit of qualification because there was a bit of concern about public health risk and things as well. Because, you know, although universal precautions became adopted gradually soon after that, the nurses were handling needles and doing treatments, the laboratory were handling the samples, and indeed the surgeons might be operating on these people, these patients. And so I think although basically it was to find out who was infected, there was also that issue. There was also, I think, the issue that we had, I think, about 500 patients then, and we needed to focus our clinical care on people who were infected. I mean -- you know, I'm doing what I said it's difficult to do: I'm reading Dr Kernoff's decision-making process to do that.
Q. You observed in the oral history project, in the passage that we've got on the screen, you said:
"... now it's horrific what we did but it was okay at that time ..."
And in your statement you say, in relation to this issue, the issue of samples having been tested without the patient's knowledge or consent, you say you abided

by the relevant ethical principles as you understood them to be at the time.

I'm conscious this was not, on the evidence you've just given, your decision, it was Dr Kernoff's decision, but can I just ask for your understanding of what the relevant ethical principles were at the time regarding issues of consent for testing?

A. Well, what I -- essentially there weren't really big issues of consent until HIV came along, and then we started taking consent before testing that.

I went very thoroughly through your expert document on the ethics and consent, and it's a wonderful document, but I mean the bottom line is that until the Data Protection Act in 1998, which became law in 2000, taking samples or using stored samples of blood or specimens, if it was anonymous and it was used for audit or epidemiological reasons, there was no need. This is before -- sorry, the Data Protection Act in 1998, which became law in 2000, states that you have to get ethical consent for a study, but you do not have to ask the individual patients' consent for testing stored serum or specimens, but it's recommended that you get -- if it's anonymous, you don't have to get the patient's consent but you should get ethical consent. This is

in 2000.

At this time, there wasn't any ethical issues around this, or consent.

Q. We do have various materials which I think have been sent to you in advance, Professor Lee, which are BMA Medical Defence Union materials really from the 1950s onwards. I can ask you about those if it would assist or if there's anything you want to say about them. Do you know whether any express consideration was given within the Centre at the time to the -- whether there was an ethical component to what you were proposing to do?

A. No.

Q. As in there was no express consideration or you don't know?

A. No.

Q. Sorry, forgive me, Professor Lee, but --

SIR BRIAN LANGSTAFF: There are two questions.

MS RICHARDS: Yes, it's my fault entirely.

Was there any ethical consideration or discussion -- was there any discussion or consideration of the ethical dimensions of what was proposed?

A. No.

Q. Okay.

Then, as I understand it, the arrangements for telling patients the result was the responsibility of others within the Centre, and you by this time were now back down to working only one day a week at the Royal Free?

A. I wasn't there.

Can I go back to what I was doing and what I wasn't doing? I started at Queen Mary's and Charing Cross at the beginning of November, and I didn't start going back for a day a week, I think it was, until '86.

Q. Okay, so in any event I won't ask you about --

A. But I mean I sometimes feel -- it was very difficult, I'm sure, for those who had to give results. I never had to do it ever. Ever.

Q. So by the time you had rejoined the centre as a consultant, full time in '87 --

A. '87.

Q. -- all the patients as far as you are aware had been told their diagnosis?

A. Yes. And the people concerned, I think, were -- it's in my statement -- were Dr Eleanor Goldman and I think she's making a statement for you, I imagine, and Mrs Riva Miller, who -- the family therapist, and I think the person who took over from me,

Dr Elizabeth Miller. I think they were the three people. I don't know whether Dr Kernoff himself gave results. I don't know.

Q. Okay.

Work was undertaken to try to establish the date of seroconversion at a later stage.

And if we have up on screen, Henry, please, WITN0644083.

This is a 1989 publication, "The natural history of [HIV] infection in a haemophiliac cohort", and again, you were one of the authors of this. And if we just look down the bottom of that page, we can see it says:

"112 anti-HIV positive patients were entered into the study using data from records and samples from the period 1 December 1979 to 30 November 1988."

Then you give various statistics in relation to that.

If we go over to the next page, please, Henry.

If we look first of all towards the bottom of the page, under the heading "Results", we can see there were:

"... 59 patients for whom a previously negative anti-HIV result was available ..."

So that's doing a retrospective exercise going back

to the stored samples -- (overspeaking) --
A. Yes, I think it might be helpful --
Q. The table at the top?
A. -- to take you through the diagram about --
Q. Yes. Top of the page, please, Henry.
A. This doesn't show 112 patients. And, again, actually, that's a slight mistake. It should be 111. I was telling you about the von Willebrand's -- subsequently was found not to have had concentrate.

This is looking at people for whom we had a specimen that was negative. And then we had a specimen that was positive. So each line represents a patient. And you see the seroconversion for the first patient was in 1979, and then gradually there's a transition over these years. And you see the last patient was the haemophilia B patient I told you about who seroconverted in 1985. From the beginning of 1985, as we've already discussed, there was heated concentrate, so there were no more seroconversions. And I think what is really sad is that -- well, if I look at the time that I started at the beginning of '83 and you draw a line up, most patients had actually been infected by then, and unwittingly, we watched some people being -- moving into HIV positivity, being infected. Of course, we didn't know.

The epidemic of HIV and haemophilia is short because it ended with heating, and also, in this country ... well, actually, in this country it tended to come over later in the general population, but that was when it emerged in the American population, donor population.
Q. Just so that I can ensure I've correctly understood the table, Professor Lee, the open circle, as it were, or the white circle represents the last negative --
A. That's right.
Q. -- result, the black circle represents the first positive result, and the line is a line -- and it's any point between those two points where the individual may have zero converted?
A. Yes.
Q. And for the purposes of the analysis you do in the paper, certain assumptions are made -- I don't use that word critically -- about taking a median point; is that right?
A. Well, it means the middle.
Q. Yeah.
A. You take the time here and the time there, and you take the middle point, and I think the middle point was the beginning of '83, yeah.
Q. Do you know whether the information that that table reveals about the time periods within which these 59

individuals would have seroconverted, was that information that was ever shared with the individual patients themselves?
A. I doubt whether it was. By the time this paper was written, the patients were on heat-treated concentrate that -- so we knew it was safe. And these data, I -- we wouldn't have had early on. But at the time that the people were giving the test results, we wouldn't have had these data. They were -- I mean, I think when they gave the test results, they had who was positive from Richard Tedder's results, but these data were assembled later.
Q. I understand that this would have been information available only potentially as at 1989 when the analysis was done.
A. Yes.
Q. And my question -- and it's a question that I've been asked by others, in fact, to enquire as to -- was: at that point in 1989 when for some patients -- only some; you had a time period within which they'd seroconverted -- was that information given to them at that point?
A. I doubt whether it was done routinely, no. To be perfectly honest, I don't know. I think one would have to refer back to the contemporaneous note that

was made.
Q. Can I turn, then, to the question of testing and diagnosis for hepatitis C. Before we look specifically at the hepatitis C testing arrangements in the Royal Free in 1990/1991, can I just ask you about the practice in relation to non-A, non-B hepatitis.

You -- 1986, you returned part-time; 1987 you become a full-time consultant at the Royal Free. Presumably there were patients who the Centre believed to have non-A, non-B hepatitis. Was that information, as far as you know from your patient dealings and patient records -- was that information and that diagnosis shared with patients at the time?
A. Well, the particular patients that I can remember that I was testing for -- I told you that it was the end of -- or the beginning of 1983, it was the end of the study period. Certainly, when I took blood from them, I discussed what we were doing and why we were doing it and what we understood. And before that, I imagine Dr Kernoff must have done that. But -- and certainly, if you look back in the notes when we did reviews later on, I mean, we talked about what had been shown in their liver function tests and what we understood, but -- yes.

Q. Your statement refers to then the process for testing for HCV once the hepatitis C tests became available. We'll look at a couple of documents in a moment, but before we do that, what can you recall -- you were a consultant at the Centre by now, 1990/1991. What can you recall about how testing for hepatitis C was undertaken at the Centre?

A. Well, it was my responsibility to look after the patients with HIV and hepatitis C, and by 1990, I was the kind of more knowledgeable one, I suppose. So the liver unit and the virology unit had got an in-house test for antibody, I think, and then later they had what's called a PCR test for antigen.

And we knew -- you know, the virus was identified in the world in 1989. And we had the test available, and therefore we tested our patients with -- to see if they had antibody. By that stage, the patients would have been regularly reviewed, and their liver function tests had been discussed with them, and those who had abnormal ones would have been discussed. And we sent -- I sent a letter of the results. I think we initially tested about 80, and you probably have the letter there.

Q. Yes, we'll have a look at the document. It's BART0000668. If we go to the next page first, please,

Henry. So if we look at both this page and the next page on screen together. It's just easier to see the document as a whole.

So we can see this is headed "HCV Policy Document for the Haemophilia Centre", and if we look on the right-hand side, we can see it's authored by you. It's dated 4 May 1990.

We can -- then if we look in "Introduction", you talk about the agent having been identified. And then if we go on to the heading "Studies at the Royal Free Haemophilia Centre", you say this:

"We've been able to study the longitudinal behaviour of antibody to HCV using the Ortho HCV antibody assay and testing stored sera from patients who developed non-A, non-B hepatitis following exposure to unheated pooled factor concentrates. All such patients have been found to be seropositive for anti-HCV. We have thus demonstrated reliability of the Ortho anti-HCV assay. In our haemophiliac population, HCV accounts for the non-A, non-B hepatitis seen, and in the majority of patients the antibody is long-lasting and may reflect virus replication," et cetera.

Just pausing there. This would appear to suggest that by this time -- this is 4 May 1990 -- at the Royal Free, all patients believed to have non-A, non-B

hepatitis had been tested for HCV using the particular test --

A. No, that's wrong. It may read as if that's the case. I think this was using the samples from the retrospective study that showed that a first exposure had a 100% risk. And so we were able -- because we had longitudinal samples of that and we knew the incubation period from the transaminases, we were able to use those samples. This is from the 1985 paper on non-A, non-B. Those samples -- we used those samples to test that antibody test.

Q. Okay. So just trying to understand. So is it only a subset of patients?

A. Yes, at that stage because -- I think the question was whether this was reliable or not.

Q. And that testing was undertaken on the stored sera --

A. Yes.

Q. -- so you, or whoever it was, took the sera from storage --

A. Yes.

Q. -- for this subset of patients.

A. It's the patients who are in the first exposure --

Q. Yes, which we'll look --

A. -- in that table --

Q. We'll look that up this afternoon. But you test them

for -- their samples for hepatitis C using the new test that was now available, so for that cohort of patients, is this right, that that first test was undertaken without their knowledge and consent?

A. Yes.

Q. If we then go to the previous page.

A. I mean, I just go back -- without their knowledge and consent. Of course, all those patients will have been told that they -- back in '78, '83, they will have been told at that stage that they had had non-A, non-B hepatitis, so they knew that. And every time they came for their review, they would be reminded of that.

SIR BRIAN LANGSTAFF: Just let her run, if you please.

MS RICHARDS: That, I think, is an assumption you're making because as you rightly --

A. Sorry?

Q. Is it fair to say that what you've just said, which is that patients between 1978 and 1983 would have been told they had non-A, non-B hepatitis, that's an assumption you're making because, of course, as you have told us, you only came in at the end of January 1983 --

A. Yes, okay, it's an assumption that Dr Kernoff would have told them that.

Q. And could we go to the first page of this document,

which is the letter. So this is a letter dated 4 June 1990. And it's obviously a standard form letter, but we have seen it crop up in individual patient records from the statements that the Inquiry has received.

"Dear [blank], I'm writing to tell you about the new anti-HCV test. Many haemophiliacs who have been treated in the past with unheated clotting factor concentrates or other blood products have been exposed to the non-A, non-B hepatitis virus, so-called because it's unrelated to hepatitis A and unrelated to hepatitis B. One agent responsible for non-A, non-B has now been identified. We have a new test available which measures antibody to HCV (anti-HIV) and shows past infection. Your anti-HCV was positive on [blank]."

Now, the way this letter might read or might be said to read is: it's simultaneously telling the patient about the new test and telling them their result, which would suggest they hadn't been told in advance of the testing that the testing was going to take place.

A. Yes.

Q. Is that what happened then for all patients? Was this the model that was adopted for all patients --

A. I think ultimately, yes, and I go back to what I said before, that the patients will have been regularly

reviewed through all these years. And we had a review pro forma, and one of the sections that patients were asked about and discussed was hepatitis. So the transaminase results would be discussed through all those years. And as information emerged, it probably would have been more detailed information about what the nature of non-A, non-B was, as far as we knew it.

Q. Do you think there's a difference between the process of telling a patient that -- what their liver function test results are and whether they have elevated transaminases and so on and actually testing them for hepatitis C once the test was available?

A. Yes.

Q. Is there any particular reason why patients were not called in and asked whether they consented to being tested for hepatitis C before the tests were undertaken?

A. I think it wasn't thought necessary at that stage, you know, it wasn't -- you see the last paragraph of this letter, that the idea was that it would be discussed at their next review, and if they wanted to come and discuss it, they were very welcome to, earlier than that.

Q. Was this the method by which patients were intended to be given their hepatitis C result? It was by letter?

A. At this stage -- I mean, I think the original -- the first batch -- I had 80 patients to do this. It's, you know, the context of a busy department doing all sorts of other things. If we had pulled up 80 patients at that stage it would have been very difficult. And the reason for putting that last paragraph in is to encourage patients who wanted to come and discuss, to come and discuss earlier. And the whole working of the Centre also was that people could walk in if they wanted, they could phone up, they could phone directly to nurses, the consultant haemophilia nurses.

Q. Do you think -- and whether with the benefit of hindsight or otherwise -- do you think that was the right way to tell patients that they had tested positive for hepatitis C, known to be a serious disease with long-term consequences?

A. Yes, because they had been -- it had been discussed over many years about non-A, non-B transaminitis, so I do think so.

Q. And the onus then was on the patient to make the appointment to come in and be given further information if they sought it?

A. Yes.

Q. Can we go back --

A. I can't exactly recall the number but it was a handful that did that. Most patients discussed it at their next review appointment.

Q. Can we go on to the second page again, Henry.

Can we look at the paragraph below the paragraph we looked at previously. It begins:

"We have also demonstrated a 7% seropositivity rate in 30 female sexual partners of 30 anti-HIV and anti-HCV positive haemophiliacs. In other studies the seropositivity rate amongst sexual partners has been higher. It seems likely, therefore, that sexual transmission of HCV may occur."

I just wanted to ask you about that first sentence. How was it that the Centre had stored sera for the sexual partners of a number of its patients?

A. I think very early on there had been a concern that, you know, because people were treating at home and sometimes families were -- it wasn't necessarily the patient; sometimes other members of the family, like a wife, might be doing it, I think that's how that had been there.

I think Dr Eleanor Goldman had looked at this, because the other issue that is sort of slightly tied up about this was that hepatitis B itself, not non-A, non-B, but hepatitis B, was very -- you could easily

get it from people you were living with. And I have a feeling that there was a study to look and see whether family members had abnormal transaminases, but -- this -- there is a publication from this, I think.

Q. The 30 female partners whose sera has obviously been tested if one looks at this, would it be right to infer from this that their sera had been tested also without their -- them expressly being told that that was happening?

A. It's very difficult to remember precisely. I think there is a -- we did look at HIV seropositivity in partners, because obviously, you know, it's very important. And of course what we haven't talked about here at all is that everybody who has -- who got infected with HIV must have been infected with hepatitis C either at the time they got infected with HIV or before, and I think this may have been the blood samples from looking at whether spouses were infected with HIV. I can't remember.

Q. That might be why you had the stored samples --

A. Yes, I think that was probably why. I can't actually remember. And -- yeah, well, that's ...

Q. Is it right to understand from this that, as with the patients themselves, the samples were tested and only

after that the patients were informed of that fact?

A. I think --

Q. That the partners were informed of that fact?

A. I think the -- I think we -- I would have to go back to the publication of the HIV in partners, because I think by that -- that's published, by the time that was done, you know, consent for HIV testing came in quite early. I mean it's something that really moved, consent for that, I think in '85. And so those partners would have consented for the HIV test. And those specimens would have been stored. But the partners weren't asked, I don't think, for their anti-HCV, no.

MS RICHARDS: Okay.

Sir, I note the time. Is this a convenient point to stop?

SIR BRIAN LANGSTAFF: Yes, it is. So let's meet again, then, at five past two. Five past two.

(1.04 pm)

(The Short Adjournment)

(2.04 pm)

MS RICHARDS: Henry, could we have up, please THOM --

SIR BRIAN LANGSTAFF: Just one moment.

MS RICHARDS: Sorry.

SIR BRIAN LANGSTAFF: We're just a little early.

MS RICHARDS: Sorry.

SIR BRIAN: No, it's my fault for starting just a bit early, don't worry. Right.

MS RICHARDS: THOM0000001, please. This is the oral history project again, Professor Lee. Could we go to page 27, please, Henry. Just look at the bottom six or seven lines. You're talking here about hepatitis C testing -- sorry, hepatitis C testing coming in, and you say:

"So in the context today, we have patients going around saying, 'They never told us anything. They never told us, but they knew, and we weren't told until 1991.' And, of course, it's very difficult for them to remember. We would have been talking about them having abnormal liver function tests, but we don't know what this means, and you're very well, and all the rest. And it wasn't until 1991 that we actually told them, 'Well, actually, you've been infected with hepatitis C'."

Now, were you saying there that the information given to patients, as far as you know prior to the availability of the hep C test, was about them having abnormal liver function tests, rather than specifically being told they had non-A, non-B hepatitis?

A. Yes.

Q. If patients were told prior to 1991 at the Royal Free

that they had non-A, non-B hepatitis, would you expect that to have been recorded in the notes of the relevant individual patient consultation?

A. Probably depends who saw them. Mostly I saw them, but not always. Some reviews were done by other doctors. I think I probably would have done. I don't know.

Q. So that I understand this correctly, if you had been seeing a patient and telling them about non-A, non-B hepatitis -- so this is before the HCV test. If you told them that you had, or you believed they had, non-A, non-B hepatitis, you would have expected that you would have recorded that in the notes?

A. Probably, yes.

Q. And you can't necessarily speak for others?

A. No. Of course, the importance of the review and the importance for considering the abnormal liver function tests and the liver was because if we sensed there was any abnormality or things were not going well -- and then, of course, when we move on and we've got treatment, we can actually refer them to the Joint Liver Clinic which I established when I became the director, where I think they roughly, or about every month, it rather depended when the consultants, hepatologists, could come down to the clinic. And I would sit alongside him in that clinic, to review

him from a hepatological point of view.
Q. When did that start?
A. I think it was after I became -- there were joint liver clinics, but I set a regular basis certainly when I became director.
Q. So that would have been after April 1991?
A. It was probably before then, because the first liver consultant that I worked with was somebody called Dr Geoffrey Dusheiko who ultimately was the world authority on hepatitis C. And he came in 1988 from South Africa. And what I remember is that we tried very, very hard at that stage to try and access treatment for these patients, and that was before I was director.
Q. Yes. Just sticking with the question of notes and what you might or might not expect to be recorded in the notes, if at a patient consultation a patient was given information about the risks of non-A, non-B hepatitis or the risks of AIDS, would you expect that to have been documented in the notes of the consultation?
A. What do you mean by "risk"?
Q. If there was any discussion, whether by Dr Kernoff or a registrar or anybody else in, say, 1983 with a patient, in which that patient was given information

about what was known about the risks of AIDS at the time, would you, based on your knowledge of practices and looking at the notes of subsequent years, would you expect that to be recorded in the notes of the consultation?
A. I don't know about '83. I can't remember. I don't know.
SIR BRIAN LANGSTAFF: You've said that Peter Kernoff was a careful and meticulous, I think --
A. Yes.
SIR BRIAN LANGSTAFF: Looking at his practices, as far as you knew them -- you may not have known -- would you expect him to record it if that had been said by him? If you can't answer it -- (overspeaking) --
A. I can't answer it.
MS RICHARDS: Then, again, just while we're talking about notes, as the '90s went, the issue of vCJD arose. I'm not going to ask you in detail about that because that's fairly well documented in what you've provided to the Inquiry and in some of your publications. But in terms of any conversations you had with patients specifically about vCJD, would you have recorded that in their notes?
A. Yes.
Q. Going back to the question of the testing for

hepatitis C, 1990 onwards, we've looked at that letter, the model letter, June 1990. Over what period of time, as far as you can recall, did the process of testing the Royal Free's patients for hepatitis C take place?
A. I don't know precisely, but I would have thought within a few months.
Q. Of the test becoming available?
A. Yes. We actually had -- I mean, on records, sadly some people had died. We had 310 patients who'd been infected with hepatitis C, and I think 30 of them had actually cleared it. So you're talking about a vast number of patients that we had to tell, although some of those patients would have been -- have died by 1990.
Q. And so the -- did you test the samples of every one of the patients who you knew had received blood products?
A. Ultimately, yes.
Q. When you say "ultimately", was that a -- (overspeaking) -- period that took some time?
A. Well, the Natural History paper published in 2000 was clearly dependent on that and, because we had such meticulous records, we could actually look at the dates of when patients had a first infusion, and we knew from the previous study that if you had a first

infusion, there's 100% chance of getting infected. So in that paper, the 310 were identified because we looked back at the records to see when they had a first infusion, and we tested those sera. Some of it had already been tested, so that was how they were identified.
Q. So for the purpose of that study, perhaps we should just briefly look at it, it's WITN0644088.
A. I think the other thing that perhaps I ought to just interject is that -- is the convention of writing papers.

This paper, which is the first author, Dr Yee, because she analysed the information. And she actually, I think, put this in towards an MD on the side effects of therapy. She has the first author here. My name at the end here is because I was in overall in charge of the study. And Anja Griffioen is an epidemiologist who looked after the data. And by this stage, although the Data Protection Act actually hadn't kicked in, we had already moved to a position that all data on computers was totally anonymised with codes. And Caroline Sabin, who is now Professor Caroline Sabin, was also an epidemiologist, and by this stage she had moved to the AIDS unit, looked at their epidemiological issues. And I've

already talked about Dr Dusheiko, who by then was beginning to be the most eminent hepatologist looking after non-A, non-B.

Q. So every patient should -- well, presumably before this time should have been told their hepatitis C results?

A. Yes.

Q. There is a reference, if you just go to page 2, under the heading "Results", on the bottom half of the page, right-hand column, you refer there in the last sentence of that paragraph, under the heading "Results" to there being 27 individuals who were consistently HCV PCR negative, they:

"... were thought to have cleared the virus and were studied in more detail."

Are you able to say what the more detailed study entailed?

A. Yes. The specimens were re-tested, I think, to check -- when -- what you're dealing with here is you've got a positive antibody test, and they're PCR negative, so they haven't got the virus anymore. And -- so they had cleared it.

And interestingly -- I think this is important -- those patients who were co-infected with HIV, there were three out of that 100 who had cleared

hepatitis C, which is quite extraordinary.

Q. The reference to further study there is a reference to repeat testing, is it?

A. I think so, yes.

Q. And when you first saw a patient after they tested positive for hepatitis C, whether it was because they'd asked to come and see you on receipt of a letter or at a consultation that was taking place in any event, what information do you recall providing to patients at that stage in the early part of the 1990s about their diagnosis?

A. Well, it would be the equivalent message that was in that letter, really. We would explain that the virus had been identified in 1989 and it was now possible to actually test and confirm that they had this infection. And I think certainly if I saw such a patient, I probably would have referred to the fact of when they first got it, that they got it when they would have had their first infusion.

And of course, when we reviewed them, we had the hospital notes, we had all the notes in the Centre, and we had the separate folder with the treatment records in, so you could find that out very easily.

Q. And what was the system in place at the Royal Free for the ongoing monitoring of patients with hepatitis C?

A. Well, the patients -- all patients with severe disease were ideally reviewed every six months, but of course that depends on patients coming. Sometimes patients didn't take up their appointments, so -- mainly they did. And certainly at their review they would have had liver function tests done, and we -- when we had the facility for doing testing, we would -- when we could do the antigen testing, we began to look to see who had the antigen and probably those people, once we began to have treatments, would certainly be reviewed at the Joint Liver Clinic. And more importantly, we became available -- became available to actually do the genotype of the hepatitis C, and again, this was information that would be passed with some -- when they were seen in the Joint Liver Clinic. And the significance of that was that once we had treatment, if you had what's called genotype 1, it was more difficult to treat. If you had genotype 3, it was more easy to treat with interferon and, later, with combination therapy. So that information was very important for the hepatologist and that would have been shared with the patient.

One of the other things I would say, and I don't -- I can't precisely say the date that I started doing this, but it became my habit to copy the letter that was

sent to the GP to the patient. The letter from the Liver Clinic, there was a dedicated secretary who did the special clinics, she would -- the doctor, the hepatologist who was doing the clinic would review the patient, talk to the patient, and he dictated the summary then, and that summary was sent to the GP, and I would countersign it to make sure that, you know, there hadn't been any mistakes. But I don't know at what stage I started copying it to the patient. And I also did that for reviews.

Q. In terms of the Joint Liver Clinic, was that something that would be offered to all patients in the early 1990s who had been diagnosed with hepatitis C, or only a subset of those patients?

A. No, it would be -- as soon as we had treatments available, all patients would be referred, because hepatitis C essentially could be cured. But this was very slow. I mean, it wasn't really -- we had in the interferon first of all, which didn't have such a good, success rate for genotype 1. It was quite good for genotype 3.

And later, when we had better treatments, the people who hadn't succeeded in clearing would be referred. I think that's the answer.

Q. In terms of treatment with interferon, you referred in

your statement to a study of 20 patients, and you said -- well, sorry, I don't want to misquote you so let's see exactly what you say. It's paragraph 97. No, it's not that paragraph but there is a question about that paragraph.

If we can have up on screen WITN0644058, please, and if we could go first of all to page 56.

So this is paragraph 95 of your statement. And you refer, third paragraph, to two studies, the second which is a controlled trial of interferon, in 20 haemophiliac patients where you say this:

"... where we describe side effects as:

"Fever malaise, lethargy, poor concentration and irritability were reported as troublesome in 16 (80%) of patients."

Then you said this:

"But we found these tended to lessen as treatment continued and were generally tolerable when paracetamol was taken with the interferon injections".

Then you go on to say:

"Side effects were very common as is usual with interferon, and although only temporary in the majority of cases, may deter patients from adhering to the demanding treatment regime."

Was your assessment, that the side effects of

interferon were generally tolerable when paracetamol was taken, based on the trial of those 20 patients or your broader patient experience?

A. I think at the time this was written, it was in the context of that trial. I think it's important at this point to say how we were able to access this treatment. When Dr Dusheiko came to the Royal Free, it began to be more common to treat patients -- as soon as we had a test, to treat patients with interferon, but it was extremely difficult to get interferon for people with haemophilia. There was a reluctance to treat such patients. Before this trial we had an experience with interferon, because -- we had a patient who had HIV infection. And, as I said earlier, everybody with HIV infection had hepatitis C. And one of the fairly rare complications of HIV infection is thrombocytopenia, a very low platelet count, and you can imagine a low platelet count in somebody with a bleeding disorder is pretty hazardous. And there had been a case report, I think in the American literature, that intravenous interferon would be successful in treating such a side effect.

And this patient who's actually reported in the British Medical Bulletin review -- and there's a picture

of it -- was started on intravenous interferon, essentially for the low platelet count. And what is quite extraordinary, he went on with this -- it was given intravenously which also was unusual because that was done because he had haemophilia. People who didn't have haemophilia were given interferon by, I think, subcutaneous injection. And his platelet count became normal, and ultimately, he actually -- he was a carrier of hepatitis B e-antigen. He converted, and because -- there's another agent called delta agent that you get only if you have hepatitis B e-antigen.

So he had HIV. He had HCV. He had HB -- delta agent. And all those were cleared completely with the intravenous interferon. And he went on having that for some time. And I think, you know, it's anecdotal, but there's no doubt that -- well, maybe it's anecdotal so there is doubt, isn't there. His CD4 count didn't decline at the rate of other people with HIV infection, so that was one experience we had.

The lady who I keep describing, who had cryoprecipitate and got acute fulminant hepatitis C and was on ITU and things -- a carrier of haemophilia -- she -- I looked after her, and I think it was Dr Thomas who suggested that perhaps she would benefit from interferon. And she was on a really tiny, tiny dose for

many years, and her transaminases went to a very low level. Ultimately when, you know, learning in hepatology, they realised what was the right treatment, she had a proper course of interferon. She had type 1, genotype 1, and she cleared it completely. And her son, who had severe haemophilia, who'd actually been somebody who had NHS concentrate, so he didn't have HIV -- and he cleared it.

So that was the previous experience going into when we tried to get this trial going.

Q. You'll appreciate that the experience of many patients who subsequently were on interferon was that the side effects were very far from tolerable and were intolerable.

A. Were very what?

Q. Were very far from tolerable --

A. It depended who the patient was. Some people did tolerate it; some people had very bad side effects. It was -- I mean, I think what is written here was the experience of these patients, some of whom were Dr Colvin's patients. It wasn't -- you know, you can see the results of the study. It wasn't at all successful, but we needed to move into a situation of trying to provide some treatment.

Q. Can you recall what information or advice you gave

your patients in the 1990s about the potential side effects of interferon?

A. Well, this study had ethical approval, and I'm sure there was a patient information sheet for it, talking about the side effects.

And the patients, when we moved on and the treatment of hepatitis C was really clarified within the hepatology world and we could genotype the patients and it was really under the direction of hepatologist, the person administering the interferon -- the patients would come up -- was a nurse specialist, and she would discuss the side effects.

Q. Can we just go to the next page of the statement, please, Henry. The first main paragraph. You talk there about your natural history of HCV paper and what you describe as:

"The lethal combination of HCV and HIV co-infection."

And then you said this:

"Hepatitis C is a very slowly progressive disorder with a 3% progression rate to a liver-related death from the time of HIV infection in 1985."

I think you probably didn't mean HIV infection in the last --

A. Sorry, what?

Q. If you look at the extract of the statement -- (overspeaking) --

A. Yes, I've got the paper here. It comes from --

Q. It's not the paper, I'm sorry. I wanted to ask -- (overspeaking) --

A. No, it comes from the e-paper. This is from the e-paper.

Q. Could I ask you just to look at the statement for a moment, though.

A. Yes.

Q. It should come up on the screen in front of you. That last sentence there says:

"For those HCV positive individuals without HIV infection, we have shown that hepatitis C is a very slowly progressive disorder with a 3% progression rate to a liver-related death."

Then it says:

"From the time of HIV infection" --

A. Yes.

Q. -- that must be HCV infection?

A. No. The significance of this is that we were demonstrating that HIV is a lethal combination. We had already, in 1994, identified that the people who had progressed to cirrhosis were the -- the majority were co-infected. There were one or two people who

drank a lot of alcohol, but mostly it was co-infection.

This paper here is analysing the progression rate after they've got HIV infection to compare the people who were co-infected with the people who were not co-infected.

Q. So for those who had hepatitis C but not HIV --

A. Yes.

Q. -- that's the cohort in respect of whom you're saying there's a 3% progression rate to a liver-related death?

A. Yes.

Q. Can I --

A. This is 2000, so you're talking about the time they would have got ... the HIV was sort of '70/'90, '83 -- '85, sorry. So that's why the analysis was done from '85, if you're comparing these two groups of patients.

Q. Can I just ask you to explain, and again, this is a question I particularly have been asked to ask you: what's meant by the "3% progression rate to a liver-related death"? How should that be understood on a year-by-year basis?

A. You mean why is -- how do you explain what 3% means? Is that what you mean?

SIR BRIAN LANGSTAFF: If I may just intervene for a

moment?

PROFESSOR LEE: Sorry?

SIR BRIAN LANGSTAFF: If I may just intervene. A rate depends -- (overspeaking) -- on what you're measuring against --

PROFESSOR LEE: I think probably could we look -- I think in order to explain this, the question you're asking me, could we look at the Yee paper which -- well, my number is 655088. Or the other number I've got is 655084. I'm not sure which one it is do you have got.

MS RICHARDS: I'm not sure either.

A. It's the 2000 paper from --

Q. So it's WITN0644088, yes. Thank you.

A. If you turn to the fourth page. I mean, this becomes rather like the familiar graphs we keep seeing on our television screens from Covid. It's epidemiological, the way epidemiologists present data. And if you look at the lower graph ... if you look at the lower graph, that's progression rates to liver-related deaths from 1985 in HIV positive and negative patients, and you see this very slow progression. Then the top one is any death; do you see --

Q. Yes.

A. -- at the top? And, of course, the difference between those is that, very sadly, those individuals who are

HIV positive have died from -- a lot of them have died from the HIV, although they might have been co-infected with whatever they were co-infected with; HCV.

Is that clear, or have I completely bamboozled everybody?

Q. We probably have enough evidence from elsewhere, including from the Inquiry's own experts, about the progression --

A. Yes.

Q. -- so I don't need to ask you more about that.

Can I move to a slightly different topic and just ask you briefly about the use of porcine products.

We looked earlier at an application that Dr Kernoff had made in December 1983 for a clinical study in relation to porcine products. I'm not going to go back to that or go to, I think, your own publications on it, but you did, I think, for example, publish a case report about successful long-term treatment with porcine Factor VIII.

A. Yes.

Q. At the Royal Free, was porcine used solely for those with inhibitors?

A. Yes. Yes.

Q. Was consideration -- would it have been suitable at

all for use for those without inhibitors, and do you know whether consideration was ever given to that?

A. I don't think so. I go back to what I said yesterday. The reason for making porcine in the beginning was: in Oxford, they realised there wasn't enough human blood to make human Factor VIII at that time, and Macfarlane suggested making bovine Factor VIII. They made bovine Factor VIII. They took, I think, three patients' teeth out, and they got antibody afterwards. So he said, "Oh, we can't make bovine Factor VIII," so I think they made sheep Factor VIII, and they made porcine Factor VIII. And subsequently, it became an extremely useful drug for treating inhibitors, not only in inherited haemophilia, but there is a condition called acquired haemophilia that predominantly occurs in elderly people, and it's an autoimmune condition, and it proved very valuable for that group of patients. And then subsequently, as time went on, Professor Ulla Hedner did a lot of research work, and ultimately, there was a product called recombinant VIIa, which was a most wonderful drug, extremely expensive, that we used to treat inhibitors. And it was first used in the war situation, or it was used in the war situation in Iraq, because you could use it to treat haemorrhage

from wounds. And it slowed down the time to -- you know, if you had people killed in the -- injured in the battlefield, you could give them a shot of recombinant VIIa, and then get them to the field situation. It was a very, very useful drug.

So the answer to your question, we used -- we used then at the Free porcine Factor VIII for inhibitors, and then latterly, we moved to a recombinant product.

Q. And could, in principle, porcine have been used for the treatment of haemophilia more broadly in those who did not have inhibitors?

A. I suppose it could have been, but you might have got inhibitors from it.

I think probably tomorrow, you're questioning Professor Tuddenham, and Dr Kernoff and Professor Tuddenham published a paper in blood describing the first use of porcine Factor VIII, and I think he can answer these questions --

Q. I'll take that up with him.

A. -- better than I can.

Q. Can I just ask you briefly about Factor XI deficiency, not what it is; the Inquiry has heard evidence in relation to that. But the Royal Free, as I understand it, treated more patients with Factor XI deficiency probably than any other centre in the United Kingdom.

A. Yes, and this reflected our situation in North London where there's a large Jewish population, and it has a very high frequency in Jewish people.

Q. And in the '70s and 1980s, how -- if we take it up to about 1985, 1986, what were the treatments that were used for those with Factor XI deficiency?

A. Initially, fresh frozen plasma was used, and then a plasma-derived Factor XI concentrate was used. It was produced by BPL and it was heated, so there's not an issue about transmitting viruses.

One of the issues which -- I don't think you were planning to talk about variant CJD, but it's relevant to CJD. When this became an issue, the thing about Factor XI deficiency, it -- you don't tend to get spontaneous haemorrhages. It particularly affects women with their periods and post-partum haemorrhage. And we looked after women who were pregnant with Factor XI deficiency. And sometimes it -- we used to do it -- I worked -- we had another joint clinic which was a joint women's clinic for inherited bleeding disorders which I started in 1995 with an obstetric gynaecology colleague.

And for the pregnant women with Factor XI deficiency, she would write a birth plan, and that birth plan would require Factor XI to be given, maybe.

And the ladies, quite rightly, said they didn't want it because it was plasma-derived; they might get variant CJD. And we did a study which is in my list of publications in my CV, we covered them with recombinant VIIa to stop their risk of bleeding.

I think it's important to know that, you know, Factor XI doesn't go up in pregnancy, but Factor VIII does, and von Willebrand factor does. So it was quite a serious problem for those ladies.

Q. The practical arrangements that were made at the Centre for the treatment of patients with HIV, when you rejoined full-time in 1987, what was the position in terms of how patients with HIV at the Centre were being cared for?

A. I had a weekly clinic on Monday morning, and depending on their medical condition, I would either review them monthly or sometimes it might be weekly when -- if they were becoming ill. And just to kind of give an idea of what that clinic was like, it wasn't until we'd identified that people were getting opportunistic infections when their CD4 count reached 200 -- the normal is 100 -- we realised that that was when they were getting the opportunist infection, so we were able to give septrin to cover Pneumocystis, we were able to give drugs to stop the Candida in their mouth,

and drugs to stop CMV, and that was done on a -- and also for tuberculosis. So that prophylactic treatment was done when they reached the CD4 count of -- of 200.

So basically, I suppose the way I reviewed those patients and I saw those patients, and I can tell you, it was such a sad period. Between 1987 and '92, on average we were losing about eight young men, and sometimes not so young, a year. And the only thing we could offer these people was prophylactic treatment, to begin with. So we would use the CD4 count as a monitoring. And you don't want me to digress, but the way we arrived at that information was from the 1989 cohort study where we realised that the CD4 count goes down in a line, and at the line of 200, that's the point at which these opportunistic infections are coming in.

So I was seeing patients and clearly, if somebody was ill, supposing, for example, they'd -- I don't know, recently had a septicaemia or something, you would put the closer follow-up.

Q. You described how in relation to hepatitis C care there was -- there were arrangements for joint clinics --

A. Yes.

Q. -- with liver specialists. What, if any, arrangements

were there for bringing in others to the care of those with HIV? Did it fall to you to start with?

A. Well, Professor Margaret Johnson, who ultimately headed up the Ian Charleson Centre, the AIDS unit at the Royal Free, because there was an enormous AIDS unit eventually, but in the beginning, she was appointed in 1989, and she was a senior registrar in chest medicine, because initially the problem that was hitting was Pneumocystis carinii, and patients were given pentamidine inhalations, and she, as a senior registrar, was beginning to develop that.

In terms of expert input, to begin with there was a joint ward round on ward 11, which was where non-haemophilia patients were admitted, and the patient care was discussed. As soon as she became a consultant, we set up her coming to do a joint clinic with me, but clearly, you know, we had a communication, and if I needed advice or help, I got it from her. But there was a formal -- she would sit in on the clinic.

There was another person who was very involved, and his name appears on some of the reports, and he's now Professor Seng Lim, in Singapore, a professor of medicine there, and he was employed as a research student under me, paid for by the MRC to run the concord trial of zidovudine. And he became quite involved and

quite expert clinically with these patients. So ...

Q. The Inquiry is aware from other information that there were patients at the Royal Free who were filmed during consultations, certainly consultations with Mrs Miller and Dr Goldman. Was that something you were aware of?

A. Yes, I was aware of it. Dr Goldman, who I would emphasise is 90, and Riva Miller, who's dead, were both trained family therapists in the Milan method, and they did video consultations with signed consent, and occasionally I would sit in, but not always. I think you are going to have a statement from her, probably, and I imagine you will ask her those questions.

Q. The consultations you undertook, as a consultant, would not have been filmed?

A. No, I didn't initiate it. It wasn't my practice. But I had sat in on consultations.

Q. You referred in your evidence yesterday several times to the balance of risk, and the choice between what you described as "life-saving treatment and the risk of infection with non-A, non-B hepatitis". It was during the course of your evidence yesterday afternoon when I was asking you about risks of non-A, non-B hepatitis.

A. Yes.

Q. What would you define as "life-saving" treatment?
A. Well, I think perhaps I would like to give you an example. Can I do that?
Q. Yes, of course.
A. The very first treatment with Factor IX concentrate, which was developed in Oxford, was in 1961. And a little boy of four, who I think came from Edinburgh, had had a venipuncture, blood taken from his arm. And he developed a haematoma, big, big clot. And that got infected. He then got osteomyelitis and gangrene of his fingers, this is in 1970 -- 61, and he was going to die unless he had an amputation. And the only way he could have an amputation was to have Factor IX concentrate.

And fortunately for him, Dr Rizza, who got phoned up because he'd been a senior registrar or something in Edinburgh I think, where this child was, the child was flown down, and he was given this Factor IX concentrate and had an amputation. The reason I know this so well is because when I was the editor of The Journal of Haemophilia, I ran a series of historical accounts to show how the treatment of haemophilia had developed and improved, and I asked Dr Rizza if he would write a commentary. The description of the amputation had been extremely successful, he survived,

and it was published in a journal. And Dr Rizza wrote a commentary about this case. And, you know, Dr Rizza was another one who was very thorough, very detailed. He phoned up the patient. And the patient I think by then, you can check in the publication, was about 36 years old. And he was very well. He must have had hepatitis C unless he'd cleared it. And he played one-armed golf very successfully. That's really such an illustration of risk of the treatment versus the ability to save a life, really.
Q. Would you accept that there will be many bleeds experienced by an individual with haemophilia where that bleed couldn't be described sensibly as endangering their life?
A. Yes.
Q. And the balance might -- could potentially be struck differently in such cases?
A. Well, it depends what you call a risk. And I know that you criticised me yesterday for quoting Carroll Birch and said, "Oh, this is 37 years ago, what kind of relevance has that got". I've got a facsimile of that original book, or report, which I had copied from the British Library, and I tell you, to look at that book is horrendous. The poor disabled people, it's awful. And, you know, you're not -- you

don't have a life-threatening thing if you're disabled and have to limp and you have painful joints, but I think, if you talk to patients, they would have appreciated, perhaps, having regular treatment and having treatment as soon as a bleed occurs.

If you don't treat a bleed, it damages the joint, and ultimately the joint becomes bent. And, you know, we still had one or two patients at the Royal Free whose life was in a wheelchair. And with the advent, the gradual advent of concentrate, not only could you let the little boys have prophylaxis so that they didn't get deformity as they grew older, but you were able to offer some patients joint replacements, so that instead of being in a wheelchair they might be able to actually walk. And certainly their pain would be reduced.

So when you're talking about risk, it may not just be, you know, stopping a cerebral bleed; it may be giving somebody a better quality of life.
Q. I just wanted to ask you a little more about how decisions about risk would be taken or might be taken. Your evidence to the Lindsay Tribunal -- and I'll just call it up -- find the reference.

It's LIND -- you're ahead of me, Henry, thank you.

Go to page 28 and 29. If we look just almost halfway down the page -- well, we pick it -- thank

you, Henry.

There's a question there:

"You indicated this morning that the parents of children with haemophilia opted for [NHS] concentrate as a preferable treatment option. Was that a matter that would have been discussed with their medical advisors as to the appropriateness of [NHS] product versus commercial product at that time?"

"No."

Then your answer went on to say:

"It wasn't at that time the practice to discuss the treatments that were being used with patients. Times have changed."

And I just wanted to ask you a little about the approach to patient involvement in decision making.

As I understand that evidence, but please correct me if this is wrong, the decision as to which form of treatment would be used was regarded at the time you're describing there as a matter for the doctor rather than for the patient.
A. Yes.
Q. You're there talking about the choice between NHS or commercial product, but the same -- would that apply, whether it was a choice between concentrate or cryoprecipitate, or concentrate or DDAVP, it would be

the doctor's decision, not the patient's?
A. No, I think what was more relevant is the type of concentrate. So what we're talking about is plasma-derived versus the high purity concentrate. I mean, that's -- that was the decision that had to be made. And it was -- when I'm talking about type -- it depends, of course, what period you're talking about. I'm now talking about type when I'm there, and the type was defined by the doctor according to the recommendation of UKHCDO, actually. You would go by guidelines. The type. That's not talking about the manufacturer.

And of course, by then, they're all heated anyway.
Q. I don't know whether you can answer this because it's a time before you're director that I'm really asking you about, but to the best of your understanding, what would -- or what would your expectation have been of the patient's involvement, in the late 70s, first half of the 80s, in decisions about their treatment?
A. I can't answer that. That was not my decision making.
Q. Would you agree with this, and this is a general question rather than specific to any particular point in time or any particular centre, that the question of -- that that balance of risk question that you identified, may depend upon the level of risk that the

individual patient is willing to accept?
A. Well, when we look at risk, it's partly how much the patient is going to accept, but it's also what is likely to happen after this first presentation, what the risk is going to be. And often, you don't know.
Q. The clinician's role may be to advise, to inform the patient, but ultimately, the judgment as to whether the patient wants to run the risk of being exposed to a virus or run the risk of sustaining damage to their joints or limitations upon their lifestyle, would you agree that that ultimately is a decision for the patient?
A. I can't answer that question.
Q. If we move, then to -- sorry, just in relation to stored samples. The practice of storing samples at the Royal Free began, you've told us, in 1978, obviously well before you arrived there. But your statement explains why you say those samples were stored. And if we look at the bottom of page 5 of your statement, sorry, Henry it's WITN0644058, page 5, you say towards the bottom of that page:

"I think it is important to add that samples used were stored for clinical management and treatment of patients with haemophilia."

We know they were used subsequently for testing,

analysed for the purposes of the research studies you describe. What did you mean by "clinical management and treatment"? In what sense were the stored samples used for those purposes?
A. Exactly what you've just said. You said -- say what you just said again.
Q. The samples were ultimately, a number of years later, used for testing.
A. Yes.
Q. Was that the purpose, in part, of storing them, in case a virus was identified in future?
A. I think it depends how you define "clinical management".
Q. But they were your words, Professor Lee --
A. Well, I know, clinical management was everything to do with managing that patient, and knowing what was happening was part of that management.

The only way that we could find out about the fact that 100% of people got hepatitis following exposure to concentrate, and that commercial concentrate was just as much risk as NHS concentrate, the only way we could find that out is by using stored samples. I would think that knowing that information was part of clinical management.
Q. Okay. And do you know, as a matter of fact, what

information was provided to patients about the purpose for which the samples were being stored, or indeed the fact that the samples were being stored?
A. They were told that the samples were being stored. The blood -- in the Centre, we had a dedicated four-bedded area, and the people who took the blood were the nurse specialists, and they would say that that was included in the spectrum of samples that were taken. But you can't say what it's going to be used for when, for example, it was ultimately used to tell the genotype or the fact that somebody had got an antibody to hepatitis C when in this stage you don't even know that there's going to be hepatitis C identified.

I would also say that the clinical management of our patients with HIV was tremendously helped by the fact that there were stored samples. So, uniquely, we could find out when people had been infected, and later on, with the help of the regular assessments we made of immunology -- that wasn't for research, that was to know whether somebody needed drugs or were getting ill. The studies on that information was so valuable to clinical management, not just for people with haemophilia but dare I say it, and it may sound terribly conceited, but worldwide, because I think nowhere in the world did we

have such a regular assessment of patients. And we were able to demonstrate that the opportunistic infections came at a CD4 of 100 -- 200, and later, in one of the papers that I've submitted to you, in 1994, we were able to show that some 25% of people, 20 years after they got infection, would not have AIDS. And that was an important, you know -- clinical management is not just doing drugs and things, it's important to tell people their prognosis.

And again, that was information that was important for the patients that we were caring for. It was also terribly important in the world, you know.

And the other thing about knowing that when the CD4 count reached the 50, patients were very vulnerable. So, you know, we might bring them up more regularly to follow them up.

So I think -- you know, clinical management is the whole patient and what is happening physiologically and pathologically to them.

Q. Were those samples ever made available to anybody or any organisation outside the Royal Free Haemophilia Centre?

A. The only thing I can think of was, after I retired, they were offered outside. And I'm sure you've got all of the references to that. I was very cross about

it.

Q. Yes, and that's an issue I will explore with Professor Tuddenham tomorrow.

A. Sorry?

Q. That's an issue that I may need to explore with Professor Tuddenham tomorrow.

A. I think, in all fairness, since I'm here, could I comment on it?

Q. Yes, certainly.

Yes, I think --

A. Could we have it up?

Q. Are you talking about the newspaper article?

A. Yes, the newspaper --

Q. Let me see if I can find the reference.

A. I think I've probably got the number.

Q. You've got it somewhere, WITN064410 -- no, that's the wrong one, forgive me.

HSOC0021123. Is this the newspaper article you were referring to Professor Lee? "Patients' fury over blood test 'betrayal'"?

A. Yes. And the date of this is 3rd June 2007.

Are you going to talk about variant CJD today or not?

Q. It rather depends on how much time we have, because there are some other issues I need to cover first.

A. Well, In that case, I'll just very briefly make a comment.

This is 2007. I retired in 2005. The Data Protection Act was 1998; made into law in 2000. So the first thing to say is that I knew you needed ethical approval for any kind of thing like this. That was the first thing to say.

The second thing to say is that, because of my involvement with the Transfusion Transmitted Working Party -- and as you know, for two years, I had constant contact with the Department of Health in order to get a multi-centre ethical approval for follow-up of patients -- I actually became quite knowledgeable about variant CJD.

The National Institute for Biological Standards and Control was almost as it says: in the world of haemostasis, you need control bottles of Factor VIII so that, you know, a lab in London is measuring the same thing as a lab in Edinburgh, so controlled samples.

A scientist from the -- as I understand it, a scientist from the National Institute of Biological Control was at a meeting in America about variant CJD. And somebody, I don't know who, at that meeting must have heard him say, "Oh it's possible to access specimens at the Royal Free," and he'd had

a conversation with Professor Tuddenham, presumably.

I was very cross about this because, you know, although you might say, you know, I was testing people without consent in 1983, things have changed, and actually, we're being told not to do that now. But even more importantly -- it's more important than the consent -- was: there wasn't a reliable test. We didn't know whether people were at risk or not at risk. So here's these specimens being offered to this scientist at the National Institute of Biological Control to do tests that then you're going to tell a patient that they've had this test. You don't know whether it's really sensitive. You don't know what it means.

I get phoned up. I was -- I feel very cross about it. I was actually on holiday in Ireland. I got phoned up by a journalist saying, "You've been testing patients," and it was wrong, and that's why.

Q. And you're reported in this newspaper article as saying:

"You can't just go around just grabbing stored samples."

Then it says:

"Acknowledging that she had used samples for hepatitis and HIV infection research at an earlier time, she said that attitudes had changed. You can't go ahead

and test people without their knowledge. It's just not on."

Is that an accurate summary -- (overspeaking) -- of what you said?

A. Yes, it is, and you know, that's what I said. But I think it's more than that. When those patient -- well, it's not more than that, actually, because the initial tests for HIV, we didn't necessarily know that they were sensitive or whatever.

So we're talking about from 1984 to 2000 and, you know, 2007, actually. So what's that?

SIR BRIAN LANGSTAFF: 23 years.

A. Yes.

MS RICHARDS: Sir, I note the time. I still have some further questions for Professor Lee and also, obviously, recognise the legal representatives' need to have the opportunity to suggest any further questions that they would wish to have asked. So is it sensible to take a break now? It might mean, obviously, sitting a bit late tonight in order to complete Professor Lee's evidence.

SIR BRIAN LANGSTAFF: Well, let me ask Professor Lee.

I saw you looking at your watch a moment or two ago, so I suspect you could probably do with a break.

PROFESSOR LEE: I think so.

SIR BRIAN LANGSTAFF: Very well. Let's do that.

MS RICHARDS: And sir, can I suggest, so that I have time to consider any questions proposed, that we take 40 minutes, 45 minutes?

SIR BRIAN LANGSTAFF: Yes. Well, let's say we'll come back at four o'clock, shall we? That gives us plenty of time, I would hope.

MS RICHARDS: Thank you.

(3.09 pm)

(A short break)

(3.59 pm)

MS RICHARDS: Professor Lee, hepatitis B briefly, if I may.

How frequently did you come across cases of hepatitis B infection at either St George's or the Royal Free?

A. I think, when we did that study in 2000 -- so we that the data -- there were actually six patients who'd had it. I particularly was involved with one patient who sadly went on to get hepatitis -- cancer. Cancer of the liver. So there were six altogether, but I was particularly -- actually, two, because I talked about the one who had the interferon.

Q. What was the severity of the hepatitis B cases? Or was there a spectrum with some mild, some severe?

A. Well, two were severe. The other thing I should add is that we did immunise patients against hepatitis B as soon as a vaccine became available, and that was 1984. And when we did that, clearly, we had to see who'd had it, and many people had had it, so they had antibody.

Q. Parvovirus. We've got two documents from you. I'm not proposing to display either of them, but we have a letter from you to The Lancet in 1995 which was a case report about a life-threatening human parvovirus B19 infection in a haemophiliac, and then a more in-depth study that you've provided to the Inquiry.

Can I just ask you perhaps to deal with parvovirus more generally. How frequent an occurrence was it that patients treated with blood products developed parvovirus?

A. I think it was probably very rare. The whole significance of that was to point out that, in spite of having what we perceived was safe plasma products, they weren't entirely safe, and indeed, one of those patients had quite a severe septicaemia, and the other was a woman carrier who had parvovirus. So it was more we were disturbed that parvovirus had caused illness in these patients.

Q. Were patients routinely tested for parvovirus at the Royal Free?

A. No.

Q. So they would be tested only if there was a reason to believe --

A. No, it was only -- it was only two cases that we encountered. But it was important to point it out to people because of the fact that they received what was perceived to be safe plasma dried concentrate.

Q. And that was then part of the debate that you were involved in, in relation to the funding of recombinants?

A. Yes. And you sent me last week the correspondence within the Department of Health that I had not seen before and many of the comments from the Chief Medical Officer about that case.

Q. Yes, and I'm not proposing --

A. No, okay.

Q. -- to ask you, not least because there's a lot of documentation which tells the story --

A. Yes.

Q. -- in relation to recombinant and the various efforts that you made in that regard.

A. Yes.

Q. So we have the correspondence for that.

Pharmaceutical companies and relationships with pharmaceutical companies. You mentioned advice that you received from your husband about involvement with -- or not getting involved with contracting arrangements or tendering arrangements. What precisely was the advice; can you recall?

A. Well, the advice was not to. And so what I practised was advising on the type of product, you know, whether it was plasma derived, high purity, recombinant, whatever. And that would be -- accord with what guidelines were around. But I was not involved in financial matters, apart from -- I think this is important for the Inquiry to know about, actually, that the financial matters when we moved to becoming a Trust became extremely difficult. And when I first took over as director -- you showed earlier these concentrates that had been commercial that we had used so that there could be a product licence got. So many people had to be treated. They were safe, but so many people had to be treated for them to get the product licence, and I think there was £1.5 million worth.

And when we became a Trust, that had happened, and they tried to -- they put their budgets in order, and they suddenly realised that they were this money short. And haemophilia was regarded as a liability within the

Trust. And it was being said that -- they had a new management structure of specialty groups, and it was being discussed at specialty groups that: oh, well, the Trust, you know, is not doing so well. It's 2 million over, but that's because of haemophilia. It was a very difficult time.

And I think this is important for the Inquiry to know. You have the document, and I won't -- you don't need to go into it, but we were being challenged about the amount of concentrate that was being used, and a tremendous amount of increase was for two main reasons: one was prophylaxis in children, so giving regular treatments, and the other was doing operations, where we need a lot of treatment. And then there was one particular case of a patient with von Willebrand's disease who had his tonsils taken out, and he had horrendous, horrendous bleeding afterwards. So there was an enormous amount being used.

And I'm -- maybe I shouldn't do this. There was one particular manager who said -- she'd been on a management -- an MBA course, and she said, "Well, I think what we should do is ask the manufacturers", and she actually went to ask the manufacturers, or somebody went to ask the manufacturers -- the bottles

used to come in 1,000 units or 500 units, and you would probably need maybe 2,000 units to treat a bleed, maybe less. And she said, "If we had bottles of 250 units, they wouldn't use so much."

So I had terrible trouble trying to explain to her, number one, they needed -- patients needed to use the amount that was right to stop the bleeding or to prevent the bleeding or whatever it was, but also, the idea that instead of having two bottles, you got four bottles. It was absolutely extraordinary.

And I don't know whether you're going to talk about this today, but that moved -- you've got all the documentation. That moved on into the recombinant era. And the -- and it's partly tied up with the parvovirus, as well, that we as Haemophilia Centre Directors, we really wanted to have our patients on recombinant Factor VIII.

And you'll be talking to Professor Tuddenham tomorrow. We had had the first patient in Europe in 1988 have recombinant Factor VIII in a trial. And when the trial finished, it had to be stopped. He had to go back on plasma-derived concentrate.

We didn't get a -- the negotiations were going on about recombinant, and we said, "Well, you know, at least we should have our children on it." And the

parting shot of this particular manager, as she left on maternity leave, was, "No way are those boys going to get recombinant."

In my papers, there is a newspaper article, and there was a child who needed his grommets repaired. And, you know, I had been lobbying the Chief Executive explaining about the safety of products, and, you know, we never know, there might be an unknown virus, and I use the parvovirus also as a marker. Anyway, this little boy who was having his grommets done, his mother, who was a very nice articulate lady, she said to me before he was having it, "I assume he's having recombinant?" And I said, "No, I'm sorry. We're not allowed to treat our children with recombinant."

And she naturally was upset about it, and I did something -- perhaps I am a little bit indiscreet. I had written a very clear letter to the chief executive, explaining why people should have recombinant, and in particular children should have recombinant. And I said, "Well, look. This is a letter I sent to our chief executive." Really to inform her. And she said to me, "What do you want to do about it?" And I said, "I don't mind what you do about it." But I just wanted to explain that I wanted this child to have the right treatment.

Anyway, I was actually away at a meeting at the time that it struck, but this lady had a friend who was a journalist on The Independent, and she showed her the letter. And there was this double page spread in The Independent with photographs of the child and the mother, and actually, it was -- you know sometimes journalists don't write well, but it was a very good article about the problem, with many quotations from my letter.

Now, it didn't do me much good, but I'm pleased to say that overnight a decision was made to let the children have recombinant.

Q. You've said in your statement that you personally received no money directly from any pharmaceutical companies. Did the Haemophilia Centre receive funding from pharmaceutical companies from time to time?

A. Yes, I think -- we had a charitable trust, as I told you, called the Katharine Dormandy Trust. And particularly when we were building the extension, which was opened in 1994, we needed -- it sounds extraordinary, but it only cost half a million then, but we were fundraising for it, and I think pharmaceutical companies paid into the Katharine Dormandy Trust for that.

And if we did -- if we did trials -- for example,

I know in detail about the Kogenate -- the first Kogenate trial of recombinant. There would be a medical advisory board, an international medical advisory board, and I was on that. I didn't receive specific payment, but clearly -- you know, mostly the meetings were in the United States, so they would pay the airfare. And I think -- and maybe Professor Tuddenham can talk about this more. As we were moving forward with the Katharine Dormandy Trust, he became the chairperson of it. He left the Royal Free in 1986, and he went to set up his MRC research unit at the Royal Postgraduate Medical School. But he came in as the chairman of the Katharine Dormandy Trust as somebody who knows about haemophilia, and, if you like, was sort of objective; you know, away from what was going on.

So they moved into a process of trying to raise money for gene therapy, which eventually was developed at the Free and jointly with UCH. And I think there were commercial monies that went into the KD Trust to help support research people.

Q. I will ask Professor Tuddenham about that, then.

In terms of your own practice, did any pharmaceutical companies ever suggest to you that you should use specific products for specific cohorts of patients, or try and influence your clinical

decisions?

A. No.

Q. And did pharmaceutical companies ever request you to provide particular data or information to them about patients?

A. Well, I suppose in the context of the -- not the patients, but certainly in the context of the Kogenate study, I was asked to lecture in Greece. And I was also -- and that was, I think, a meeting that was sponsored by the manufacturers of the recombinant product.

Q. I wanted to ask you, in terms of your research, just about the study that you've referred to a number of times in your evidence but we've not looked at in detail. It's PRSE0003439. So this is your 1985 paper with Dr Kernoff: "High risk of non-A, non-B hepatitis after first exposure". And I just have a handful of questions relating to that study.

You've already pointed out that it was a study that took place over a five-year period from April 1978 to March 1983, so I'm not going to ask you -- I'm going to try not to ask you, in any event, questions about how it was set up because you weren't obviously there in 1978. But do I take it from the fact -- well, your name appears second on the list of authors. So it's

Dr Kernoff, then you, then two others. You would have had a role at least in the writing up of this paper?

A. Yes, I -- when I went there in '83, I was handed a graph paper strip about this long, and what Dr Kernoff had done from the time he became appointed in 1978 -- because he had come from Oxford where he realised about hepatitis, every time any patient needed treatment for the first time, he wrote down their name, what was the problem, and what they had. So I was handed this sort of long scroll.

And what I did was go to the notes and find out, you know, who they were, what they presented with, look at all the liver enzymes and try and work out -- and whether they were symptomatic when they had this treatment, and to try and work out -- you could work out the incubation period from when you got the peak of the transaminases. And that's -- the information that is in the table, which is on page 474, which is EPU --

Q. The sixth page, please, Henry.

A. Page 6, isn't it? All that information on the table -- it's got the concentrate they had, the batch, the dose. And then this previous exposure, the significance of the previous exposure, this previous exposure in cryoprecipitate. And then you see the incubation period, the peak ASTs, and whether they

were symptomatic or not. So this long scroll of graph paper that I'm telling you about, the reason it was presented to me like that was, you can imagine, if he's busy in a centre and he's just started, he had a lot of other things to do. So he was just, you know, writing this down.

And that was compiled from the hospital notes of these patients. And then, you know, this graph of incubation periods was calculated from this information.

In terms of authorship and writing this paper, the reason Dr Kernoff's name goes first is clearly it was his idea, and his enthusiasm for it. The reason Howard Thomas's name goes at the end is because in practice, that's usually the author who's a sort of lead person, and what was happening here was Howard Thomas had been very advisory about liver disease with Dr Kernoff at that stage, and -- he was working under Professor Sheila Sherlock -- and clearly, he saw the drafts of the paper and things, but actually, he did nothing to get the information.

Peter Karayiannis was the laboratory scientist with Professor Thomas, Dr Thomas, and they were trying to get an assay of the antigen of non-A, non-B and it was totally unsuccessful. But because he had been supportive and was in the group, that's why his name was

on the paper.

My name is on the paper because I looked out all the information and also I wrote quite a lot of it. My recollection is that I wrote the patient's methods because that's factual, that's not opinion.

I think Dr Kernoff wrote the introduction, and I think the conclusion, he -- I think -- I can't remember whether I wrote the first draft and he, you know, corrected it or something, and of course I would have researched the references, which was not -- it was quite a difficult task in those days, as you know, getting the precise title of the patient and the volume and all the rest of it, you had to go to the library and look it all up and everything.

Q. The bit I want to ask you about particularly is under "Patients and Methods", I'm hoping you can help. It's really just some factual questions. So if we go to page 2 please, Henry -- it's page 470 if you have the paper copy -- and we go, towards the bottom of the page, "Patients and Methods". Just enlarge that so people can see.

It describes 58 patients receiving 60 first exposures. I'm not quite sure I follow that but I don't think that matters. And then 31 of those exposures prospectively studied, 29 retrospectively

studied. And then you go on to say, towards the bottom of the page, or you and your co-authors:

"... a minority of the patients were 'virgin' ..."

So there were a handful of patients who had not previously received any form of blood, plasma or cryoprecipitate therapy.

Do you have the page I'm looking at?

A. No, I'm fine.

Q. Then:

"... most [of them] needed infrequent treatment ..."

That would tend to suggest, would it, that they were predominantly not severe haemophiliacs but non-severe?

A. Yes, I was just looking if, in this table, it actually -- in some way it describes who they were.

But -- I thought it was on this table. Hang on a minute.

There were a number of people who had von Willebrand's disease. Surely it's here somewhere.

Q. Yes, if you go to the next page.

So it's page 471, or page 3 please, Henry.

A. Yes, here we go.

So 13 had haemophilia A, four had haemophilia B, ten had von Willebrand's disease, three were female

carriers, and the three female carriers, two carried haemophilia A and one carried haemophilia B. And it says five patients, six exposures, were less than 5 years at the time of exposure.

Yeah, okay.

Q. And then of the patients with haemophilia A or B, seven were classed as haematologically severely affected.

A. Yes, I would think the most -- likelihood is -- I can't remember now, but, it's most likely they were the children.

Q. Those who were severely affected?

A. Yes, because the adult patients who were severely affected most likely were -- certainly would not be untreated -- you know, virgin patients ... I don't -- the term "virgin" is awful, and I make apologies for that. It was the convention at that time to call people "virgin patients". Unfortunately the North Americans found it wrong, which I think is right, it was wrong. And so it was changed to "previously untreated patients" which I think is rather a more respectful way of talking about patients.

Q. Just in the same paragraph it says in the last two sentences:

"Duration and dosage of therapy, and choice of

therapeutic product, were influenced by clinical circumstances, local availability of products, and departmental policies which operated at the time treatment was given."

What's meant there by "clinical circumstances"? Are you able to cast any further light on that?

A. I think it was the nature of the bleeding episode, whatever it was.

Q. Um --

A. I mean, I -- because I had a Rule 9 request, I know certainly, I think, three people had von Willebrand's disease, and there was epistaxis. I thought the clinical thing was listed here, but I can't see it at the moment. So that's what it means, it's: what was their bleeding? Why did they need to have this treatment?

Q. Okay, and "local availability of products" I think is probably self-explanatory.

"Departmental policies which operated at the time that treatment was given."

That is going to, presumably, predate your arrival, and I've asked you already about what you were able to assist with in terms of policies.

A. Yeah, I mean, it's what you've been discussing about Dr Kernoff and what he was using. I mean, these

patients were being treated from the time he started in '78, through that time.

Q. And it says, and again it was really whether you can throw any light on this, it then says:

"These policies changed over the period of the study, as it became appreciated that the risk of [non-A, non-B] hepatitis after concentrate therapy was very high."

A. Well, I think that possibly relates -- well, I don't know, because it wouldn't have DDAVP in it. I don't know what specifically that's referring to. It may -- I don't know.

Q. And in terms of the products that were used for the purposes of this, we can see those, can we, from your table on page 474?

A. Yes.

Q. Then if I can just ask you to look at the bottom of page 472 -- it's page 4, Henry -- under the heading "Ethical and legal considerations", if we just -- it's the last paragraph, Henry.

A. No, I've got it.

Q. It's just for the benefit of others to be able to see.

It talks about ISG and Kryobulin-G being used on a named patient basis. I wonder, could you just explain what they were used for.

A. This was the only part of the study that needed ethical approval because it involved giving a product immunoglobulin, and the idea was to try and see, if you gave immunoglobulin, whether it would protect against non-A, non-B.

It's a bit akin to the Covid-19 patients -- and indeed, Donald Trump, I think -- having antibody given. And because it was a commercial product and it was done in the context of a study, it had to have ethical approval.

This was completed before -- the accrual of the information was completed before I started. And as it happens, one person did not get non-A, non-B. But it was only five -- out of five people, so whether that was significant or not, I don't know. I think it's shown in the table there's a -- yeah, the cross in the table --

Q. Page 6, Henry.

A. -- indicates the ones that had the immunoglobulin, and you see this person with Factor IX deficiency -- I have a feeling it was a carrier, actually -- where, with Factor IX deficiency, it was NHS Factor IX deficiency, did not get non-A, non-B. And I think in the paper we speculate why that might have been or why ...

Q. If we just go back to the bottom of page 472, sorry,

Henry, back to the bottom of page 4, what's said there under "Ethical and legal considerations" about information to patients and Ethical Committee approval, do I understand that that relates solely to the use of the ISG and the Kryobulin-G?

A. Yes, yes.

MS RICHARDS: Sir, before I turn to another project, was there anything you wanted to ask Professor Lee about this paper?

Questioned by SIR BRIAN LANGSTAFF

SIR BRIAN LANGSTAFF: Well, I do just want to ask a couple of things.

If I can just go back to the point which raised your own eyebrows, Ms Richards, at the very start, under "Patients and Methods" --

A. Yes.

SIR BRIAN LANGSTAFF: -- it says:

"58 patients with congenital divisions of coagulation factors VIII or IX received 60 first exposures ..."

Now am I right or wrong in thinking that first exposure generally in this paper meant first exposure to concentrate? Or did it mean first exposure to any treatment?

A. Yeah, I think -- I agree, yes, it's rather confusing.

I think possibly it refers to the two -- no, I don't know. I'm sorry it's a bit pathetic, isn't it, but I --

SIR BRIAN LANGSTAFF: There may be some help. If you go back to the table at 474, one of the curiosities, if you look at the numbers which go down from 1 to 30, 16 appears twice, between 24 and 25, and in its normal numerical order. And it may well be the same person, a baby or infant, at 19 months and at 27 months, I don't know, but it's the same patient number.

A. I'm lost, I'm sorry. Oh I see, okay. Ah, I know --

SIR BRIAN LANGSTAFF: Does that help?

A. Yes, it does. I think it's ... I think, yes, it's -- it's because they had -- I think it's because they had cryoprecipitate on two occasions, probably.

SIR BRIAN LANGSTAFF: But that's what I thought might be an answer, but then I don't really understand if you go back to page 470 -- so we go back to that, please, Henry.

Right down at the about of the page, page 470, you say this:

"Only a minority of the patients were 'virgin' ..."

You've rightly put that in inverted commas.

"... although most needed infrequent treatment, a majority had received blood, plasma or cryoprecipitate

that way before."

A. Yes, um --

SIR BRIAN LANGSTAFF: So it's a definition of what a previously untreated patient was --

A. I think in the context here it was, strictly speaking, the concentrate. But what you -- you raise a very, very interesting point here, because if you look at the NHS patients, there are 12 of them, and ten get hepatitis and two don't, and as it happens, the two that don't had had enormous quantities of cryoprecipitate. And I think we speculate in the discussion that maybe they were immune. And indeed, that is so, because when we were able to have the test, they actually had antibody.

And I think -- yeah, I think that underscores the fact that if you have vast amounts of cryoprecipitate, you could have got it.

SIR BRIAN LANGSTAFF: The next thing which I would just like to understand is you set out, in previous exposure, going back to the table -- I'm sorry, Henry, to ask you to dodge around -- back to page 474.

A. Yeah, I've -- now I've got it.

SIR BRIAN LANGSTAFF: Under "Dose", well, that's international units per kilogram, presumably.

A. Yeah.

SIR BRIAN LANGSTAFF: And that would prefer to dose of concentrate, would it?

A. Yes, yes, that's right.

SIR BRIAN LANGSTAFF: And that explains why, when we come down to the 22 to 25, the unit is donor units, and donor units are donations?

A. No, this is the bags of cryoprecipitate.

SIR BRIAN LANGSTAFF: Yes.

A. So I think what we've done there -- I can't remember, I don't -- no, we wouldn't have -- what you could do with cryoprecipitate is you could actually take a sample out and measure it in the lab, but I don't think that was done. I think what was done was looking at the literature of where people had done that, and so, you know, the average bag of cryoprecipitate contains this much. And then you can put the unit --

SIR BRIAN LANGSTAFF: Well, just stopping you there, because I think the unit of comparison is rather different.

One is international units per kilogram, the other is donor units. And I think in the text you define donor units as the number of donations.

A. Yes, but I think -- no, it -- let's take patient number 22, who's the female aged 40, and she has

NHS cryo. And we've got this column saying, "Units per kilogram", and she's had -- so that essentially is 84 units per kilogram. And that's been worked out from the -- I think the average content of a cryoprecipitate bag is something like 30 units, something like that, or 60 units. It's very variable. And so that -- so you'd multiply 70 by 60. Would that work it?

SIR BRIAN LANGSTAFF: No, it wouldn't.

A. Well, she weighs --

SIR BRIAN LANGSTAFF: 84 is 12 times 7.

A. Yes, but she weighs 50 kilograms, doesn't she?

SIR BRIAN LANGSTAFF: Ah, yes, so that then might. But then the 70 donal units is -- (overspeaking) -- dose per kilogram.

A. -- (overspeaking) -- 70 times 60 is -- you're better at maths than me, 4,200. If you divide that by 50, does that work out? Yes, it does.

SIR BRIAN LANGSTAFF: Yes.

A. Yes.

I'm sorry, I --

SIR BRIAN LANGSTAFF: No, no, that's fine. I just wanted to understand what it was telling me, really.

A. If you'd asked me this in 1983 I probably would have been able to tell you very quickly.

SIR BRIAN LANGSTAFF: Please understand I don't expect people ever to remember every detail of everything that happened years ago. I couldn't, I'm sure nobody here could, unless they're remarkable. So don't worry about it.

So if we're looking at the paper, on one view, it would say -- in the summary, at the start of the paper, please, Henry.

There are 21 patients who had Factor VIII concentrate. It's describing nine out of nine British patients treated with USA commercial products developed non-A, non-B. And ten out of 12. There's 85% treated with NHS products, non-A, non-B.

A. Yes.

SIR BRIAN LANGSTAFF: So that could be read as suggesting that the British product was less likely to develop non-A, non-B.

A. It could be read like that but, as we say in the discussion, these two patients had had vast amounts of National Health Service cryoprecipitate. But you're right, I mean, it could be read like that. But subsequently, when we were able to measure it, there's no doubt that these people had been immune.

SIR BRIAN LANGSTAFF: Part of the significance, perhaps, of this study and the Fletcher study which came before

it, is that until this time, 1983, 1985 -- 1985 for this study, it had been, I think, thought that those who received cryoprecipitate and nothing -- no concentrate, were much less likely to develop hepatitis, and that NHS concentrate was much less likely to develop hepatitis because the donor pool was less infected. And you were showing, I think, you say --

A. No, the data at the time which came out of blood transfusion data in a cardiac surgery unit, I think in Scotland, was that the infection rate of hepatitis C, or the transmission rate, was 1 in 300 of the population. And so, in theory, once you reached 300, it was almost inevitable you would get hepatitis C.

SIR BRIAN LANGSTAFF: Just dealing with the donor pool size, do you know how the donor pool size for commercial concentrate or NHS concentrate worked?

A. I think at that time the NHS product -- I'm a bit sort of rambling numbers -- but I think it was something like 4,000, 5,000, and the American pool size was sometimes bigger.

But the other thing that happened in America much faster than here, and I'm sorry I can't give precise dates, was that in America, in order to get plasma, they used the technique of plasmapheresis, where they

would use a single donor and that donor, you know, in a year would give more donations.

So that inevitably, you know, cut down the risk of it, didn't it, because you've got one person giving, say, four --

SIR BRIAN LANGSTAFF: Unless he was giving infected -- (overspeaking) --

A. But I think it's right to say that the commercial products were a bigger pool size, but I think the significance of this was, one -- I mean, I understand your criticism, but the significance of this was that non-A, non-B hepatitis approached 100% in NHS, when previously it had been suggested it was safe.

The interesting thing is that it probably is right to say that the viral load might have been bigger in the commercial concentrate because if you look at the lower diagram and you look at the days to the incubation period, you see for the commercial Factor VIII, it's about three weeks, whereas for the NHS Factor VIII it's about seven weeks. And it has been suggested that that might be because of viral load.

And the other thing that I think came out of this study which is true is that when you look at the symptomatology, there were many asymptomatic people, but

there were some who were quite symptomatic, and I think they were more likely to have received commercial Factor VIII, and that may have been because it was a bigger viral load. The analogy to Covid-19 is quite remarkable, actually.

SIR BRIAN LANGSTAFF: I was just wondering if -- let me put it in context. The Inquiry is yet to have a full report -- given to me, anyway -- of the relative pool sizes that were used for commercial concentrate on the one hand.

A. Yes.

SIR BRIAN LANGSTAFF: -- and NHS concentrate on the other. Such information as I currently have, and it is by no means the last word, suggests that NHS concentrates were initially made from very small pools.

A. Yes.

SIR BRIAN LANGSTAFF: And very little infectivity was reported. It may have happened, but very little was reported. And later on, particularly around about the 1980s, they became very much larger. Now, that may or may not be right, but it does give rise to the question whether infectivity may, in part, be a consequence of the pool size, or whether there's any -- you weren't studying that at all in this paper, were you?

A. No, um, but what I can say is, in all my experience of looking after this large number of patients during the years, I never encountered a patient who had not got infected.

SIR BRIAN LANGSTAFF: I see. Well, that's all I --

A. I think the other thing to say about this. I don't know ... I don't know. Probably not, actually, because what I was about to say is whether any of these -- no, they wouldn't have been. I was thinking about co-infection. No, forget it.

SIR BRIAN LANGSTAFF: That's all that I wanted to ask you about this paper. Thank you very much.

Further questions by MS RICHARDS

MS RICHARDS: Professor Lee, just a very small number of final questions, I've been asked to ask you.

You referred in your evidence yesterday to a Haemophilia Centre profiteering through the purchase of commercial Factor VIII concentrate. Do you know which pharmaceutical company or companies were involved in relation to that?

A. I don't know. I suspect many.

Q. And which Haemophilia Centre?

A. I don't know. I feel a bit bad about -- I know who it is. I know the centre, but I feel rather embarrassed about saying these things. I don't --

SIR BRIAN LANGSTAFF: I don't think you should be embarrassed. We are, after all, inquiring into something which is of public importance, so the public interest, I think, suggests that you should tell us --

A. Can I then rehearse what I was saying yesterday?

The contracting at the Royal Free, when I was director, was that the managers did that contracting. And as I understand it, whatever they paid for the concentrate was passed on to whoever was buying it, and the people who were buying it would be sometimes a district, but I think there were occasions when there was a GP practice that had a patient who were the contractors.

And there wasn't profiteering, if you like, but at St Thomas', where Professor Geoffrey Savidge was the director -- and he was a very good physician. He'd trained in Sweden. He looked after his patients extremely well. And I am not suggesting for one moment that he put any money -- he took any money. I don't think he did. But what he did was he would negotiate, and I think he did the negotiation. He's died now, so he can't confirm this or not. He would do the negotiation directly with the drug company, and he would -- so they paid A plus B for a unit, or whatever it was. When the purchasers -- when the contract was

made with the purchasers, there would be added on, I don't know, 2p a unit or 3p, I don't know, but there would be added on something. And that meant that St Thomas' Hospital had all this extra money because they were buying the factor at one price, and then they were getting money from the purchaser at another price.

And I hope I'm not being libellous or something because -- I mean, this is -- this is the truth as I know it.

Within the hospital, he became a kind of king, if you like. And I'm not criticising any way he treated his patients. He was excellent, and I think he cared very much for the organisation and the clinical care of his patients, but he was making a kind of profit for the hospital. And it almost became that he was kind of handing out, you know, money to different departments, or different departments were benefiting from this.

And I'm certainly not going to tell you the detail of something further I know, but there was a difficulty relating to Professor Savidge, and I knew the details of this difficulty. And I spoke to the Dean in the medical school about this difficulty, and he said he would investigate it. And he didn't investigate it because he said, "I can't investigate this because the hospital is benefiting from this finance."

But again -- I would say, again, that Professor Savidge I don't think was benefiting himself; he was benefiting the hospital. And I think also it benefited his centre. His centre was very nice. But it wasn't the right thing to do, I don't think, but it was a consequence of this change in contracting. And it was this change in contracting that, in the end, compelled me to start looking at the economics of all this, because it seemed wrong that the Royal Free was a wonderful hospital, and it had got this wonderful Haemophilia Centre who were very expensive. And, you know, they needed this expensive treatment, but a tenth of the haemophilia treatment of the UK was being -- having to be provided by the Royal Free in this purchaser-provider contract. And in that leader that I wrote for the BMJ, I was saying, look, there needs to be some kind of national thing here.

I think -- I haven't really kept up with what's happening now, but I think that has largely happened. It's -- you know, it's the problem of a very expensive specialty which is of low volume.

MS RICHARDS: So that there's, I hope, no great mystery about these matters, the difficulty you refer to about Dr Savidge, Professor Savidge, was not connected to his care of patients?

A. No. It was nothing to do with haemophilia. Well, it kind of was, but it wasn't.
Q. I'm not proposing at the moment to ask you any more about that.
A. Indeed, I think -- I don't know if there were other centres that that's how the money was managed.
Q. Again, arising out of some evidence you've given either yesterday or today, you talked about the convenience and practical advantages of concentrate over cryo. I'm not asking you to go over that again, but you talked about how the patients wanted concentrates for that reason.

As far as you know, from your own direct personal knowledge only, was any patient actually presented with that choice at the Royal Free in the first half of the '80s?
A. I don't know.
Q. And then we talked about the storage of samples of serum. I don't need to ask you about that again.

Was there ever any storage, to your knowledge or understanding your directorship, of any other tissue samples from haemophiliac patients post-mortem?
A. Yes, I -- the only thing I'm particularly aware of, the post-mortem samples were stored anyway -- biopsy specimens were stored -- and I think it wasn't until

the Alder Hey problem that these all had to be destroyed.

In 1998, we did a study jointly with Edinburgh and Oxford and the CJD unit in Edinburgh, because there was a big, big worry and concern as to whether patients with haemophilia were going to get variant CJD. And what we did, we knew that we had a group of patients between these three centres who had had nothing but NHS concentrate for the years. You know, the BSE came into cows in '80, and the idea was that people ate the meat, and they were blood donors and contributed to the concentrate which then went into people with haemophilia.

It was really a big concern, so we identified a number of patients. And, actually, I was going to send the paper and I never got around to it -- it's here -- but I will do it after the Inquiry. And this is before 1998, so it's -- and 2000; it's before the Data Protection Act. And there were specimens from the brains of people who had died, and they were looked at in the new variant CJD centre in Edinburgh. And there was no prion agent, and this was very reassuring.

The only -- the only evidence that there has been transmission is that there was a post-mortem done in Oxford much later, I think, 2003, for other reasons in

a patient who had haemophilia, and prion agent was found in the spleen. That's the background to that, and I will send the paper.
Q. Thank you. There's just one further matter. I wanted to ask you about two comments that you made. Again, I've been asked to ask this question, Professor Lee. I'm sure you'll understand why.

Could we have up on screen, first of all, Henry, JEVA0000009. This is an interview that you and Dr Rizza conducted with Dr Biggs, and if we could just go to page 4, please.
A. This was done for the historical --
Q. Yes.
A. -- Wellcome Trust document, and because Rosemary Biggs was quite elderly then, that's the reason it was done as an interview.
Q. Yes, I understand that. It's the highlighted passage towards the bottom. And if we just pick it up before the highlight with -- can we go to the passage just above that, Henry, to put it in context. This is -- Dr Biggs, says:

"The next thing that started to crop up was that patients started to get jaundice, and we felt at the time that they were better alive and having jaundice than dead with haemophilia."

And then your response was:

"That's the most amazing comment. I get very irritated with patients now who are demanding compensation because they have got hepatitis C from concentrate, but they wouldn't be alive to make those kind of complaints if they hadn't been treated."

That's the first I wanted to draw to your attention. I then want to draw another, and then I want to just ask you a question if that's okay.
A. Yes. You kindly sent this to me last week. And when I read that now, I think perhaps the word "irritated" is strong, and I think what I was really trying to express there was frustration. And let me explain.

Dr Biggs made this quite brutal statement -- I've written about this; it's in many of my papers -- that patients were better getting jaundice than being dead. And it's presented very harshly, but what it's saying here is what we've been discussing throughout. And it's a balance between risk and the necessity of treating. And what she's got on her scales is the risk of treatment, which at the time she was working, there were patients getting jaundice, and the -- if you don't give the treatment, there's the possibility of dying.

And I would take you back to the fact that in 1937,

the life expectancy was 20 years. Towards, I would say, the beginning of the '90s, it was approaching 70. And this was at the cost of having hepatitis and sadly, for many people, HIV. But the other side of it was people lived a life. And, indeed, as early as 1981, there was an anonymous leader in the BMJ, which I think was probably written by Peter Jones who I think you may be talking to, and he makes the point that the side effect of hepatitis may be the cost of having this very effective treatment.

I think the other issue that I would just say about this is that I think the Macfarlane Trust and the Skipton Fund are wonderful. And I believe, truly believe, that any patient who has any treatment for which they have really severe side effects should have financial support, as far as possible, to make their life better.

But what I don't like about the idea is the idea of compensation because what compensation does, it suggests liability. And I truly believe that people at that time were doing what they thought was the best, and the side effects were really not at all clear, and certainly HIV was a tragedy that nobody could have foreseen. And of course, the other thing that we haven't really talked about at all is this lethal combination of HIV and HCV.

And many of our patients, and I think this probably goes for the majority of patients who had HIV, they progressed so fast to liver failure, and we couldn't have seen that.

And, you know, it's very easy retrospectively to say, "Ah, but the HIV all came from America," and that's true up to a point, but there were occasional donors who had it here. The infection in Britain came much -- sort of a year later. We were lucky we could do de-referral at Blood Transfusion Centres which mitigated it to a certain extent.

So I suppose, you know, when I read this, when you sent it, I just felt that my choice of words was not very good and rather insensitive in this interview. But I think what I was irritated about was that I had not been able to explain or convince people that what we did was in good faith, if you like. For the knowledge at the time.

Q. The second document is from the oral history transcript. It's THOM0000001. If we go to page 35, please, Henry. If we go to the bottom half of the page, so it's about ten lines down from what we can see on the screen. You say this:

"And in the context of today, you know, there are one or two haemophilia patients who are really driving.

They want compensation, and their argument is that Britain didn't get self-sufficient. Well, that is an argument we didn't get self-sufficient, but, on the other hand, they were -- in the period that -- well, self-sufficiency would not have done anything about hepatitis. From the point of view of HIV, the fractionation and the ability to provide enough was just not there, and the patients were actually champing at the bit. You know, they wanted it. They wanted to have regular treatment. They wanted to be able to have their operations, and that's kind of forgotten. You know, you may be able to blame America for HIV. You certainly can't blame it for hepatitis, and personally -- but, you know, maybe I would say that, wouldn't I?"

Then you say this, Professor Lee:

"Personally, I don't think that faults can be attributed. And, you know, that recent Inquiry in Scotland, the Archer Inquiry, fortunately did kind of sort of come to that kind of conclusion, but it kind of rumbles on. I mean, cynically, I think the patients, the few patients driving this, are probably after money, actually. There was quite a big payout. Not payout, but there is a fund -- I've forgotten the name of it for a minute -- for hepatitis [that's obviously Skipton], and it's done on health grounds and also on whether

you've got families and dependents."

I asked to show you that, Professor Lee, and I just wanted to ask you whether, on reflection, you stand by those comments, or if there is anything further you want to say about them?

A. It's a similar reflection that I have for the Rosemary Biggs comment, or in that article, in that I really supported and continue to support the idea of the Macfarlane Trust and Skipton Fund which is giving support for hardship, but I think that compensation is the wrong thing to do because it suggests liability.

And I think at the time I conducted this, I was feeling particularly aggrieved, and I think it would not be appropriate to tell the Inquiry who this patient was. But I was particularly aggrieved because a patient had received -- and this was hearsay of the amount -- but had received £80,000 as an out-of-court settlement. And I had been asked about this case, and I had seen the notes, and if that had gone to court, I think it wouldn't have shown any liability. And I was feeling aggrieved that the National Health Service had paid out this money really that was unjustified.

I think it is right for any medical treatment, if it causes harm that wasn't expected, the patient should be

supported. But I think the idea that we would -- anyone who is a decent person would give somebody some treatment that they knew would cause harm is frankly ridiculous. And it's actually quite hurtful for those people, those many people who cared for patients.

I mean that's -- I really don't want people to think I'm a hard, unsympathetic person because I'm not. These people have always been part of my life. They're still part of my life. I sometimes see them. I've got an ornament of a Greek student who died on my shelf. Every day I see that, and I see this man. It was the saddest tragedy of all, but I think to suggest culpability is wrong. Sorry, my speech is over, but I -- it does upset me.

Q. Professor --

A. I've been -- one of my rule nine things said I killed somebody, and that is -- it is so hurtful. And then I think, well, I'm pathetic. These patients have got far more problems than me. Why should I feel pathetic about that? But I do. I feel totally hurt.

Q. Professor Lee, those are the questions I have for you.

Sir, are there any questions that you have?

SIR BRIAN LANGSTAFF: No, I don't, thanks. I've asked all that I've needed to, and you've answered the others with what you've been saying.

MS RICHARDS: Is there anything else, Professor Lee, that you wanted to add?

PROFESSOR LEE: Yeah, I would just like to say something. We've talked a lot about risk, and, you know, whether treatment was necessary, but one of the things I was thinking a lot about last night is: one of the things treatment has done for a lot of patients is it had given them some better quality of life. And I just want to give you three examples of patients who I cared for, where the concentrates really had made a dramatic difference.

One had had prophylaxis. He'd had prophylaxis since a child. And he'd studied music, and he became a composer who won a prestigious prize and performed a work at the Barbican; somebody with severe haemophilia. And that is just amazing.

And another patient, when we were -- when our orthopaedic surgeon was able to do -- he did double knee replacements because it -- there was less concentrate, and also the patients actually said that it was less frightening having one operation than two. He had double knee replacements, and his walking was completely transformed. He had HIV and, of course, hepatitis C, and he went on the Trans-Siberian Railway. And on the border between Mongolia and wherever on this railway, he

said he was stopped at three o'clock in the morning, and they had some kind of inspection. And he said, "It was really weird because they looked at passports, and then they asked was I HIV positive?" They didn't know he had haemophilia or anything.

And he said, "I thought it was probably better to be economical with the truth at three o'clock in the morning."

But, you know, that was somebody who had this wonderful experience, and then the third patient that I remember -- sadly he has died -- was an Egyptologist, and he'd done most of his researching and things at the British Museum, but when he got concentrates, he could go to Egypt.

So that's to just reflect on the fact that life was improved for a lot of people. And my personal first memory of haemophilia was when I was doing orthopaedics in Oxford, and there was this ward of little boys with joints strung up to all sorts of things. So, you know, it -- treatment has been good, despite these terrible side effects.

And the last thing I really want to say is I want to pay tribute to Dr Kernoff. It's very difficult. He left treatment as a doctor when he was 40 years old, I think, with this heart attack. He was the most

amazing organiser and he cared for his patients, and he set up a very good centre which I was lucky enough to inherit. And I just think that he just needs to be remembered. I think that's enough.

MS RICHARDS: Thank you, Professor Lee.

Sir.

SIR BRIAN LANGSTAFF: Well, it remains for me to thank you, Professor, for coming to give your evidence. You've given us a much greater understanding of what life was like at the Royal Free, in particular, when you were there, and what it was like to practice as a researcher and as a clinician during the late 70s and through the 80s, and since. And you'll understand -- I know you understand -- how important it was that we hear from someone with that experience in your position.

And I'd particularly like to thank you because it's common knowledge that concern about the -- "the virus", as we might now call it -- Covid has been heightening, particularly in London, and it makes me all the more grateful that you were able to come in person, as I want you to, but you did, and so thank you for that.

PROFESSOR LEE: Thank you.

MS RICHARDS: Sir, tomorrow we have Professor Tuddenham,

so we continue with the Royal Free at ten o'clock tomorrow. And Professor Tuddenham is also here in person.

SIR BRIAN LANGSTAFF: So, ten o'clock?

MS RICHARDS: Ten o'clock tomorrow.

(5.09 pm)

(The hearing adjourned until 10.00 am the following day)

I N D E X

F:

F:

F:

F:

F:

F:

F:

F: