Wednesday, 26 January 2022

(10.00 am)

SIR BRIAN LANGSTAFF: Professor Barbara, can you hear me?

THE WITNESS: I can indeed, Sir Brian.

SIR BRIAN LANGSTAFF: You can see me?

THE WITNESS: Yes, I can.

SIR BRIAN LANGSTAFF: Good. In a moment or two, Ms Fraser Butlin will start asking you some questions after Mary has asked you to take the affirmation. But, first, you can tell me you are at your home, are you?

THE WITNESS: I am, Sir Brian.

SIR BRIAN LANGSTAFF: At home there's, what, your wife and others?

THE WITNESS: Yes, and my dog --

SIR BRIAN LANGSTAFF: And your dog, right.

THE WITNESS: -- who is being kept securely out of the way so that he doesn't join in.

SIR BRIAN LANGSTAFF: I was just going to ask that. You are talking to a room here in Aldwych, in which there is a select and small group of people. We have no more because of the restrictions we are observing because of the current virus. Beyond this room, however, there are something in the region of 100 or so people who will be listening to everything that you

have to say today. So that's the audience that you have, the public that you are speaking to.

Now, Mary would you invite Professor Barbara, please, to take the oath.

PROFESSOR JOHN ANTHONY JAMES BARBARA (affirmed)

Questioned by MS FRASER BUTLIN

SIR BRIAN LANGSTAFF: Yes.

MS FRASER BUTLIN: Thank you.

Professor Barbara, can you see and hear me?

A. I can indeed, very clearly.

Q. Good. Professor, you hold a PhD in microbiology; is that right?

A. Yes, yes.

Q. So it is right, isn't it, you are not a medical clinician, rather you are a scientist?

A. No, I'm a consultant clinical scientist.

Q. In 1974 you became the head of microbiology at the North London Blood Transfusion Service?

A. Yes.

Q. That was about the same time as when Professor Contreras also joined the North London service, isn't it?

A. Yes, I have it in my mind we joined on the same day but I can't be sure of that.

Q. The Inquiry has heard evidence, as you know, from

Professor Contreras that she became director of the centre in 1984. When that happened, is it right that you and Dr Patricia Hewitt shared responsibility for the scientific and clinical microbiology work at the centre?

A. Yes.

Q. Would it be right that Dr Hewitt took on the clinical aspect of the work and you the scientific aspects?

A. Yes, she was dealing with the medical side and I was dealing with the clinical science.

Q. Just to complete the overview of your career, you then became microbiology consultant at the National Blood Authority in 1994?

A. Yes, and reported to Angela Robinson at that time, as well as to Marcela Contreras.

Q. You remained in that role until 2001?

A. Yes.

Q. At that point, you became an emeritus consultant?

A. Yes, I went back to London two days a week for just over five years.

Q. You remained there until about 2005; is that right?

A. Yes, about that, 2006.

Q. I just want to talk with you, to begin with, about your role at the North London centre. You had responsibility for everything to do with microbiology;

is that right?

A. Yes, including the provision -- the detection and provision of high titre immunoglobulins, like for HBIg, hepatitis B immunoglobulin, things like that.

Q. Can you tell us more about what a microbiologist works entails?

A. I suppose the call was the screening of the 1,000 or so donations a day that we would receive for an ever-increasing number of microbial agents, and then the confirmation of any reactives, the dealing with the donors who were found to be positive and, because our honorary consultant was Dr David Dane at the Middlesex, who had discovered what was known as the Dane particle, the actual infectious particle of hepatitis B virus, with his advice, I was able to set up counselling for HBsAg positive donors.

And we also did any follow-ups of possible post-transfusion infections. We did bacteriological screening of plasma before it was returned to the plasma -- before the red cells were returned to plasmapheresis donors, and we did a variety of projects associated with the development of microbial aspects. I kept registers of infected donors. We followed up -- I think I have already said -- things like jaundice enquiries and, eventually, I was

a founder member of Serious Hazards of Transfusion.

And so it was quite a wide-ranging set of remit, also developed automation, computerisation, because obviously when you are dealing with 4,000 or 5,000 tests a day, the more you can streamline it, the more you can ensure an error free pathway and proper quality control, then the better.

Forgive me for sniffing. I'm afflicted with catarrh. I think it is the slight change in the weather.

Q. Please, don't worry.

I can see that something's happened with the picture. I just want to check that you can still see and hear us and that we have not lost you?

A. Yes, perfectly.

SIR BRIAN LANGSTAFF: We had a message that the bandwidth was low. That's Professor Barbara's bandwidth is low. I hope that will be rectified.

MS FRASER BUTLIN: Indeed.

A. I fear that we are at the end of the line in the country.

Q. Would it be fair then from that description of what your role entailed, would it be fair that you were then focused on the science involved in the testing of blood and in securing the best data in the issues of

testing that you were dealing with?

A. Yes, that was mainly the thrust of things but there were a whole variety of associated projects. I was -- because of our help from Dr Dane, I was able to set up panels of infected donors, which other centres didn't do. We were able to, for example, plasmapherise high titre HBsAg positive donors so the plasma could be used for a project that was started by Professor Arie Zuckerman, to derive a British plasma derived hepatitis B vaccine, which didn't actually come to fruition. But there were those -- a lot of those sort of things.

Q. But would it be fair that your focus was on the science rather than the clinical side of the North London centre?

A. Yes.

Q. And you weren't, in your role, charged with making decisions about the broader policies that were in play in relation to which tests to introduce and when at the centre? That would have been for the director?

A. Yes, with the input from the evidence, the data we collected, the kit assessments. But the decision would be from the director, yes.

Q. It is right, isn't it, that the North London centre was the only Regional Transfusion Centre to employ

someone with a PhD, with a doctorate, in microbiology, to lead their microbiology laboratories?

A. Yes, that is correct. This was initiated by the previous director, Dr Tom Cleghorn, who very early on engaged Dr Dane as honorary consultant.

I think in my statement I may have described Tom Cleghorn as visionary because he recognised that, rather than being a nuisance that you had to get round, the problem of transfusion-transmitted infection, if anything, would grow bigger. So he was very far sighted, you know. And along came the whole range of agents that I have listed in one of the slides from my lectures that I have appended.

And I think Dr Cleghorn, with Dr Dane's advice, decided to have a bespoke microbiologist, or virologist, to head the department.

Q. That visionary view of Dr Cleghorn must have been in the early 70s, because your appointment was in 1974. Did he ever talk to you --

A. Yes.

Q. -- about what it was and why it was he felt this was something that was coming down the road?

A. Yes, in various conversations, the details of which of course I really don't remember, I became aware that he was certainly aware that the microbial aspect of

transfusion was not going to be limited to bacterial screening of blood before it was returned to plasma donors, the syphilis testing that had been in place for donkey's years and, in the early 70s, the newly set up hepatitis B surface antigen testing. I think he was aware that blood was an ideal portal of entry for blood-borne infections to be transmitted to a patient, with blood being infused directly into their bloodstream.

Q. I have also been asked to ask you whether it would be fair that, because you were the only head of microbiology with a doctorate, that made you something of an anomaly within the Blood Service?

A. In that respect, I guess I was anomalous, yes. I didn't think of it that way.

Q. And, therefore, whether you were effectively working alone, with somewhat limited peer-to-peer interactions and engagement?

A. No. The people who headed the other blood centre, what used to be called AU testing labs, Australia antigen, were senior technical staff, initially known as technicians but then more appropriately called medical laboratory scientific officers, and I was in close contact and had various projects and derived a lot of good information and ideas from these

colleagues and all the other 13 or so centres around the country.
Q. Are you aware of any discussions with other Regional Transfusion Centres about them also employing postdoctoral microbiologists?
A. No, not in the early stages at all.
Q. In terms of the physical location of the North London centre, it was physically very close to the PHLS, wasn't it?
A. Crossed a fence, yes.
Q. And also --
A. To PHL.
Q. -- the CDSC?
A. Yes.
Q. How much interaction was there, firstly, between you and the PHL?
A. A lot. And it grew considerably as the list of potential microbial risks of transfusion also grew. So I would be across the fence a lot and they would be across the fence to us. The Public Health Lab, the Communicable Disease Surveillance Centre, a lot of interaction. That was one of the benefits of being where we were.
Q. And those interactions, were they informal discussions about things you were looking at or were they more

formal meetings?
A. It was a mixture. There was lots of informal kicking ideas around. There were also formal committees, which I know you have got listed and I have forgotten most of the ones I was on or chaired, and there were joint projects that we would set up. For example, the kit evaluation group involved Dr John Parry, now Professor John Parry, in doing the seroconversion panel assessments that I have described in my statement.
Q. Before we get there, I want to discuss with you a little bit more about some of the things that were set up in the North London centre. At some point in the centre you set up an archive of serum samples.
A. Yes.
Q. Do you recall when that archive was established?
A. Not exactly. It was reasonably early on and it was facilitated by automation, automated samplers, and the use of a microplate, 12x8, a 96-well microplate -- again, there is a slide in my package of stuff -- which meant that we could store large amounts of samples securely but in a small space, and with data retrieval because they were barcode labeled. So it was quite early on and it did serve as a model, I know, for other centres. The Scots had started it

around the same time.
Q. Just so that those who are listening understand what a microplate is, it is a plate with wells in where a small sample of serum can be put into each well and labeled and then frozen down?
A. Yes. And the archive microplate I spotted in the manufacturer's catalogues, what was called a deep well microplate which took one millilitre, 1ml, and of course that was a godsend. And you could cap it as well.
Q. Did the North London centre seek to encourage other centres to set up a similar archive of samples?
A. I don't think there was formal encouragement. There was a lot of interaction and contact between the microbiologists at the centres, and we would set up seminars and we would discuss things like serum archives. So I think that ideas did catch on. And it was a two-way thing: we took on ideas, other centres took on ideas.
Q. Moving on then to post-transfusion hepatitis and the screening test for hepatitis B.
In the late 1970s, if your lab identified a donation as testing positive in a screening test for hepatitis B, can you talk us through the process that was then followed? Just broadly.

A. Broadly, okay. Do stop me if I get carried away by my pet subjects.
You would do a test for HBsAg and if it was initially reactive -- we never called them "positive" until they were confirmed -- if it was initially reactive we would repeat on that sample in duplicate and if one or both of those were also reactive, we would get a snippet of bleed line from that pack and test the bleed line as well to make sure that we got the right sample for the right pack. Then, those samples at that time, before we had our own reference lab set up, they would go to the Middlesex Hospital for confirmatory testing. There would be neutralisation tests done that would confirm that, by ablating the reaction with specific anti-HBs, you could say that that reaction was definitely HBsAg. You would do anti-HBc testing and you would check if it was of the IgG or IgM antibody class and if it was IgM, you would know it was a recent or an acute infection.
Q. At some point in that process would the donation be held so that it didn't leave the centre?
A. As soon as there was a repeat reactive result, which came within the same day, that donation would be held. No donation would be released until my lab would have

cleared them and any initial reactive would not have been cleared. A repeat reactive would have been held and my staff would collect the donation from the blood bank and store it securely in the fridge.

Q. Thinking back to the archive sample, once that archive was available, if a donation tested -- was repeatedly reactive, would you then go back to the stored samples to check them for the same donor?

A. No.

Q. Why not?

A. No, we would save the stored sample for any future use. If there was a discrepancy between the test sample and the snippet from the bag, then we would go back to the archive sample.

Q. Sorry, Professor Barbara, that was my question that wasn't clear enough. If you have a donor who is a repeat donor and the current donation has tested --

A. Sorry, not a repeat reactive?

Q. You have got a current donor who has tested positive, would you go back to their historic prior donations in the archive? Apologies, my question wasn't clear.

A. Forgive me. I thought you were meaning a repeatably reactive sample, I'm sorry.

Yes, absolutely we would go back to the archive sample and we'd, first of all, re-test in my lab in

case we had made an error and I'm happy to say -- another topic -- but we could show that we made very few testing errors. If we couldn't find any HBsAg, we would send the archive sample to the Middlesex and they would, in the early days, have access to radioimmunoassay, which subsequently we were able to develop for our own use, they would have that more sensitive test, they would check it for anti-core and anti-core IgM and then, if there was any hint that it was infectious, I would inform the hospital and we would -- I or Dr Hewitt -- and we would request samples from the recipient of that donation, preferably samples, rather than them testing it, because our reference laboratory had a good range of specialist tests to get to the bottom of the problem.

Q. Thinking about the reverse situation, Professor Barbara, the centre also operated the J system that the Inquiry has heard about, didn't it, in relation to post-transfusion hepatitis reports?

A. When you say "J", there was "JH", jaundice history, and "JE", jaundice enquiry. So, yes, we operated a JE system.

Q. So if a clinician reported that a recipient of a transfusion had post-transfusion hepatitis, you would then make enquiries into what had happened?

A. Yes. We would ask the clinician if they wouldn't mind sending us a sample. We would also ask if they got a pre-donation or pre-transfusion sample because, sometimes, we would be following up a potential case only to find that the recipient had a pre-existing HBV infection and then, when we received those samples, we would do the battery of tests I've described to check whether they were infected, and because the serology for hep B was so extensive, had been worked out early on, we would be able to say what stage of the infection that recipient was in.

Q. When you say the "battery of tests", that would include a hepatitis B core antibody test?

A. Yes, IgG, IgM and HI antigen and anti-HBe. They would also do ALT and AST.

Q. Could we turn, then, to WITN6989011, please.

A. Sorry, will this come up on my screen?

Q. It should come up on your screen, Professor Barbara. Just give it a moment and please say if it doesn't.

A. Forgive me, I will take my glasses off so I can read it more easily.

Q. This is a document, an article, that you wrote with Moya Briggs. It is dated at the top September 1982, headed "Post-transfusion hepatitis in North London in 1981; a review", and we can see in the first

paragraph:

"At the hepatitis workshop held in Scotland last year we presented a review of post-transfusion hepatitis in North London during the previous ten years. The present report provides details of our 1981 PTH enquiries."

Then, in the next paragraph, we can see that there were 16 PTH reports received in 1981.

A. Yes, "Two of the 16".

Q. Then, if we turn onto page 6 of the document, and we have the heading "Conclusion" --

A. Yes.

Q. -- could we just zoom into the conclusion part. We will just zoom into the conclusion so it is a bit easier to see.

It reads this:

"Our enquiries into PTH during 1981 illustrate the diversity and complexity of this work."

You were referring there, weren't you, to the number of tests that were involved and the difficulties in tracing what the root cause of the PTH was, weren't you?

A. Yes, and the differentiation of which agent may have been responsible, if at all.

Q. Then we see this, at the bottom of the page:

"We are trying to encourage the hospitals we supply to report all PTH in the hope we can get more information about non-A, non-B as a cause of PTH in the UK."

A. Yes.

Q. Is it fair then, Professor Barbara, that in 1981 you recognised that not all cases of post-transfusion hepatitis were being reported to you?

A. Yes. If I could amplify slightly, I think even earlier we were aware of two things, that not all post-transfusion hepatitis was due to hepatitis B and also that not all cases were necessarily being reported and so with Marcela -- with Professor Contreras and Dr Hewitt and other colleagues, we did set about quite regularly updating, doing seminars for our hospitals, to impress on them the value of telling us about any possible cases, in part so that we could prevent any infectious donor from infecting other recipients.

And, if I could also add that you asked about my remit earlier, this aspect of education and this aspect of R&D, for example, to analyse the cases of post-transfusion hepatitis B reports, that was an important part of what I did and, of course, this was helped by being a bespoke virologist or

microbiologist.

Q. We saw there a moment ago a reference to a study that looked at the -- at a longer period of post-transfusion hepatitis reports. I just want to go to that document.

CBLA0001301, please. If we zoom into the top of it, so it is a little bit easier to read on the screen, we can see that it is a short communication in the Medical Laboratory Sciences journal 1981 by you and Moya Briggs again.

Then if we go down to the body of the text on this page, please, we can see that you are presenting:

"... preliminary results of [your] approach to the examination of the extent of post-transfusion hepatitis of the non-A, non-B type in the region served by the North London Blood Transfusion Centre."

If we go further down in this paragraph, we can see it reads:

"The numbers of cases of post-transfusion hepatitis reported to us during the last 10 years are shown in [Figure] 1."

A. I'm afraid I don't have that.

Q. It is okay, we are going to just turn the page, where we see Figure 1, which is at the top of the page, which is rather difficult to read, Professor Barbara.

So if we carry on to page 3 we have a table which is much more helpful, I think.

A. Okay.

Q. If we can look at that table, we can see the totals across 1976, 1977, 1978, 1979 and 1980, the totals of the post-transfusion hepatitis reported cases?

A. Yes.

Q. It is right, isn't it, Professor Barbara, that this study depended on the post-transfusion hepatitis being reported to you?

A. Yes, absolutely.

Q. If we then go down to the next paragraph, please, below table 2.

A. May I add that that, of course, was why we encouraged hospitals to report anything that they were suspicious about, so that we would get a clearer picture of what the situation actually was.

Q. We can see in the paragraph highlighted:

"Most of our cases of post-transfusion hepatitis are based on reports of clinical jaundice: of 15 cases reported during 1977-80 this was the presenting factor in 13. In two of the cases where the patient was not jaundiced one had chronic hepatitis and subsequently became [hepatitis B surface antigen] positive, while the other recipient 'felt unwell', and had raised bilirubin

and liver enzyme levels. In two cases where blood from donors incubating hepatitis B was transfused, both recipients became [hepatitis B e antigen] positive 'inapparent' carriers of [the surface antigen] though with raised liver enzymes."

Just pausing there. It is right, as well, isn't it that, as well as being entirely dependent on post-transfusion hepatitis being reported to you, those reports would only be made if someone had clinical jaundice or something else to cause the clinicians to suspect there was an issue?

A. Yes.

Q. Then if we go to the final paragraph of this paper, we can see that the conclusion in the last paragraph is:

"The clinical importance of chronic aspects of non-A, non-B hepatitis is not yet clear, and much chronic non-A, non-B hepatitis resolves itself within 2 years. Probably post-transfusion hepatitis B is more important than the non-A, non-B variety, since not only does it appear to be the more severe infection but, if transmitted to a patient in hospital, it may be the source of more obvious infections among the staff. Even with sensitive screening methods currently available for testing donor blood, the hospital reports of post-transfusion hepatitis are still important for the

prevention of further cases caused by the same donor."
A. Yes.
Q. Your conclusion there, that post-transfusion hepatitis B is more important than the non-A, non-B variety, was that based on the number of post-transfusion hepatitis cases that were being reported to you, where it wasn't hepatitis B?
A. Sorry, I couldn't quite catch the last part of your question.
Q. Let me re-phrase it. Your conclusion here is that post-transfusion hepatitis B is probably more important than non-A, non-B hepatitis. Given that you were reliant on reports of post-transfusion hepatitis and given that the majority of those post-transfusion hepatitis reports indicated hepatitis B positivity, was that part of the reason why you concluded that hepatitis B was more important than non-A, non-B?
A. Yes, I understand what you are saying now.
Yes, I suppose the clinicians were more -- would more readily report a possible post-transfusion hepatitis if their laboratory had found hepatitis B surface antigen. So there might have been a bias in terms of the number of cases of post-transfusion B reported. I think also that non-A, non-B, as we knew it then, would often generally be milder and might not

have been picked up, unless they were doing liver function tests.
And certainly there was quite a general feeling in Blood Service circles that hepatitis B was more severe, could kill you, you could get fulminant hepatitis because of the very vigorous antibody response that, paradoxically, would prove fatal in a small number of cases. So I think those were the factors that made us feel that B was more severe than non-A, non-B.
Q. It is right, isn't it, that asymptomatic non-A, non-B, at least initially asymptomatic non-A, non-B cases, would be missed entirely or potentially missed entirely by this study?
A. Yes. The American studies would have funded prospective work, some brilliant work by Dr Harvey Alter, who would follow up recipients with liver enzyme studies. So they would see the evidence for non-A, non-B.
Q. You have said in your statement that, at that time, you didn't consider non-A, non-B as something that was particularly serious in terms of the clinical condition; is that right?
A. Not as serious as hepatitis B, yes, I felt that.
Q. Do you have any sense of the timeframes when your view

about the seriousness of non-A, non-B shifted?
A. I can't pinpoint that. Forgive me, my memory is not that clear. What I would say is that as the first serological tests became available from Chiron and Ortho, although their specificity and predictive value in a low incidence and prevalence population was poor, in a patient proportion it had more predictive value and one was able to read reports of hepatitis C being detected in patients with hepatocellular carcinoma or chronic liver disease. So it was latterly that the awareness of the significance of what we knew as non-A, non-B became clearer.
Sorry, does that make sense?
Q. Thank you, Professor. Can I take you to a paper that the Inquiry has looked at a number of times, PRSE0003622, just to see if this assists.
It is a paper that was published in The Lancet on 16 September 1978 by Professor Preston, headed "Percutaneous Liver Biopsy and Chronic Liver Disease in Haemophiliacs", where it was reported that there had been systematic screening of 47 haemophiliacs in Sheffield and liver biopsies had been carried out. We see in the middle of the paragraph that:
"A wide spectrum of chronic liver disease was demonstrated, including chronic aggressive hepatitis and

cirrhosis."
Was that a paper that had crossed your desk that you were aware of in the late '70s?
A. It may well have. I have to be honest, I didn't recall it until I saw it in the documents that you kindly sent to me.
Q. So you are not sure if, at the time, that was something you were familiar with, Professor Preston's work?
A. It was very likely I was but there were an enormous number of publications that I would have been involved with and reading, so I certainly was aware of Professor Preston and the work he was doing and I have a feeling, a sort of recollection, that there was a general -- I had a general awareness but I can't say definitively that I read that paper; I probably did.
Q. I'm asked to ask you whether, with the benefit of hindsight, do you think that the North London centre or you were too slow to recognise the seriousness of the non-A, non-B hepatitis?
A. I think that once a specific test -- and I say specific in the general term -- was able to identify the virus in patients significantly affected, I think we were pretty quick to be aware then. I think beforehand, yes, there may have been a feeling that it

was less important than hepatitis B.

And, I have to be honest, in terms of severity, chronic condition, carrier state, sexual transmission, transmission from mother to child, I still feel that hepatitis B is a more aggressive virus.

Q. Can we then look at some of the testing that the centre was doing in relation to post-transfusion hepatitis. If we look at NHBT0000030_007. It is a short communication in Vox Sanguinis in 1983 that you wrote in Dr Contreras and Dr Moya Briggs.

A. Moya didn't have a doctorate, although she would have deserved one.

Q. Apologies.

It is headed "A Donor Implicated in Two Cases of Post-Transfusion Non-A, Non-B Hepatitis". I will read some of it and then I'll pause and ask you questions about it:

"In the absence of specific tests for non-A, non-B hepatitis viruses, evidence for their involvement in [post-transfusion hepatitis] can only be circumstantial."

The report then discusses a particular donation:

"A UK-born male had donated one of only two units given as whole blood to a patient who became jaundiced 6 weeks after transfusion. Serum taken from this

patient at the time of jaundice was negative by RIA for [hepatitis B surface antigen, hepatitis B core antibody and hepatitis A], but weakly positive for [hepatitis B surface antibody]. After notification of this case of [post-transfusion hepatitis], the two donors involved were resampled ... Serum from the other donor was negative by RIA for [hepatitis B surface antigen] but positive for [hepatitis B core antibody] (IgM class negative) and [hepatitis B surface antibody]. This donor had also mildly elevated enzyme levels."

You give the figures for the ALT and the AST:

"Although the implication of the second donor was only presumptive, he was asked to refrain from blood donation until further notice ..."

You then explain that, because of an error where the donor was told he was safe to donate again, he then donated seven months later, and the recipient of that donation was identified as having post-transfusion hepatitis.

Then if we go over the page you set out the testing of that subsequent donation and you conclude at the bottom of the page:

"Nevertheless, the situation described (a donor who is both anti-HBs and anti-HBc positive and is suspected of a possible link with PTH) justifies, in our

opinion, the permanent exclusion of this donor from donating blood."

A. Yes.

SIR BRIAN LANGSTAFF: You add at the end of that --

MS FRASER BUTLIN: I'm going to come to that in a moment, sir.

SIR BRIAN LANGSTAFF: You are going to deal with the last sentence?

MS FRASER BUTLIN: I will, absolutely.

SIR BRIAN LANGSTAFF: Very well.

MS FRASER BUTLIN: Before we get there, I want to deal with one other point if I may, sir.

Just before we look at the last sentence, I just want to be clear, Professor Barbara, that when you were dealing with post-transfusion hepatitis, situations like this, you were testing samples for hepatitis B core antibody, hepatitis B surface antigen, ALT and AST; is that right?

A. Yes.

Q. And would it be fair --

A. That would be -- sorry.

Q. Go for it.

A. I'm sorry. I was going to say, because there was, if you like, a higher level of potential -- sorry, I'm tongue-tied for the moment.

Because it has been a report of post-transfusion hepatitis, there was, if you like, a smoking gun that made it more indicative to do these supplementary tests, which is why we would have done those. They wouldn't have been tests that we would have done on any donor, it was just because this donor had been one of those, and potentially implicated in a case of post-transfusion non-A, non-B in a recipient.

Forgive me for interrupting.

Q. No, I think there is a slight delay on the system which is making it slightly difficult, so please don't apologise for interrupting at all.

Essentially, what we see here is, isn't it, that you are using those tests as a surrogate for non-A, non-B hepatitis testing?

A. Yes.

Q. Then, if we come to the last sentence of the article, you note that:

"This procedure is reminiscent of measures taken for the prevention of PTH B before HBsAg tests became available."

A. Yes. I of course wasn't in service then, but I was aware from the literature and from the discussions with Dr Dane and Dr Moya Briggs that -- just this kind of thing, that an indirect surrogate approach would

have been the only thing they had when investigating post-transfusion hep B. Or likely hep B.
Q. But it is right, isn't it, that these tests weren't used by the centre across the board to screen routinely on a surrogate basis?
A. That is right. Because, if you like, the report of possible transmission or infection of a recipient enabled you to focus on a very small number of donors that would benefit from these indirect non-specific and surrogate tests.
Q. I want to pick up that point a little bit later because it comes later in the chronology again. And we will address it again in relation to hepatitis C a little bit later today.
Can I move now to your understanding of HIV.
You have said in your statement that your understanding of HIV shifted over time, but that at first you didn't link it to blood transfusion. Is that right?
A. That is correct. Do you want me to expand on this slightly?
Q. Yes, please do.
A. When an immune deficiency syndrome was initially described with reports in MMWR, Morbidity Mortality Weekly Reports from the US CDC. It was very difficult

to know -- to make sense of that. It appeared to be eventually due to an infectious agent. Certainly it was indirectly picked up by CDC because of the increased incidence of pneumocystis carinii and the increased dispensing of pentamidine, which was a drug used to treat pneumocystis.
Initially people wondered whether it could be some sort of agent like swine fever that had gone rogue or whether it was due to the use of recreational drugs called poppers, or even if it was due to a suppression of the immune system in the passive partner in a male homosexual relationship, being exposed in delicate mucosal areas to large amounts of the active partner's semen.
Q. Your view shifted somewhat, didn't it, Professor Barbara, when you received a call from Dr Roger Dodd?
A. Yes.
Q. He was the head of American Red Cross transfusion infection laboratories?
A. Yes, he was sort of my "oppo", my opposite number over there. And I knew Roger very well, he was an expat Brit, a virologist, and we had worked together and he was somebody that I knew and could talk to freely. And he phoned me one day to say that

they'd heard that two haemophilia patients had developed AIDS -- or I think it was called GRIDS at the time, gay-related immune deficiency syndrome. And I immediately said, "Well, I suppose they are homosexual men", and he said, "No, don't think so, married with kids", and the chill realisation that this was a virus and, as such, would have been transmissible by blood and even by fractionated products, because the process of fractionation to make Factor VIII would inactivate parasites and bacteria but didn't inactivate the acellular virus particles, any acellular virus particles. So it was, I think -- I don't know, about 1983, that -- this was a phone call that absolutely stuck in my mind, and still does. I don't remember the date. I'm not good on dates I suppose.
Q. Can I try and assist you with the date, Professor Barbara? The Inquiry has heard evidence of that CDC report being on 16 July 1982.
To the best of your recollection, do you think that Dr Roger Dodd phoned you fairly swiftly after that CDC report.
A. That seems very likely.
Q. So it is more likely to have been around in July 1982 than the 1983 that you have taken a guess at?

A. Yes. Looking at it with the report data, yes.
Q. And so at that point I think your evidence is that you understood HTLV-III, AIDS, the name that it was given at the time, to be transmitted by blood to the best of your understanding?
A. To be potentially transmitted by blood, yes. It seemed a real possibility. A frightening possibility but a real one.
Q. Moving forwards in time. You were involved in the drafting of the first AIDS leaflet together with Dr Tom Davies; is that right?
A. Dr Davies, yes.
Q. Could we have NHBT0020668, please. We have a letter from Dr Wagstaff from July 1983 enclosing a copy of the final form of the leaflet. If we then go over the page we have a copy of the leaflet.
Is this the version of the leaflet that you were involved in? As far as you recall?
A. Can I have it a bit bigger, please?
Q. Of course.
A. Yes, I believe I was involved in this, and more specifically in the little fold-over leaflet, the smaller-sized one, about, I don't know, eight or nine inches by four inches, that continually evolved. But, yes, I would have had involvement in this.

Q. If we look under the heading "Who is at risk from AIDS?", we see three groups of people who are said to appear to be particularly susceptible:

"(1) Homosexual men who have many different partners.

"(2) Drug addicts, male and female, using injections.

"(3) Sexual contacts of people suffering from AIDS."

Firstly, in relation to the first category, "Homosexual men who have many different partners", was that something that you were involved in drafting, that wording?

A. Yes, I would have been involved. That wording wouldn't have been down to me. There was a reluctance amongst RTD transfusion centre directors to be prying, if you like, into donors' sexual habits.

And as an aside, I must say that, since the majority of transfusion directors had a haematological background, they didn't, as it were, think like a microbiologist or a virologist, where sex and drugs were something that I would have always been aware of as potential routes of transmission of agents, and, you know, the overlap of sex and drugs and blood donation I was aware of. So there was a reluctance to

pry too deeply. And also it made sense at the time to recognise that, on a statistical basis, the more partners you have the more likely you are to encounter a partner who is infected with HIV.

Q. But it is right, isn't it, that this might suggest to a man in a stable partnership with another man, that they were still eligible to donate?

A. Yes.

Q. With the benefit of --

A. Yes, I --

Q. Sorry, go ahead.

A. Sorry. No, I would certainly have had that feeling myself, yes.

Q. And whether at the time or with the benefit of hindsight, do you think it would have been better for this to have said "Men who have had sex with men"?

A. Yes, I do.

Q. If we then look at the second group "Drug addicts, male and female, using injections", might this also suggest that those who have perhaps injected drugs once or twice, would similarly not be caught by this definition?

A. Yes, I think -- again, with hindsight -- intravenous drug users, either male or female, would have been the cover-all, but at that time one was able to use just

the word "drug addict", male and female, and I agree the phrase "using injections" implies now or currently or recently. So, again, with hindsight, I would have preferred something stronger. But as I've said, I would have been a part input into this document, which would have had a lot of input from various people, often more senior than myself.

Q. Then if we go down to the heading "Can AIDS be transmitted" --

SIR BRIAN LANGSTAFF: Just before you do that. We picked up yesterday when Dr Wagstaff was giving evidence that the first of those categories, "Homosexual men who have many different partners", is just as you have described in respect of the word "using", it is talking about now, as opposed to the past. It is in present tense.

So also, arguably, is sexual contacts of people suffering from AIDS. From your perspective, would the risk actually be not just those who are currently engaged in sex with (a) many different partners, men -- being homosexual, those who are currently using injections, and those who are currently a sexual contact of someone who has AIDS, but people who have been, to their knowledge, in the past?

A. Absolutely, Sir Brian. I think I would have preferred

to have that element strengthened. Of course, going back to a time when we now know that HIV was circulating from the early 80s, in smaller numbers probably even earlier, but for me it wasn't a current or a recent because a past event might have caused the damage of infection. So yes, I totally agree with you.

SIR BRIAN LANGSTAFF: It was a question really, rather than an observation, but it is, I suppose, also an observation.

Thank you.

MS FRASER BUTLIN: If we can go down to the heading "Can AIDS be transmitted by transfusion of blood and blood products?"

The answer there is given:

"Almost certainly yes."

The Inquiry has heard evidence from Dr Walford about the original draft of the leaflet that she and Dr Gunson prepared, just before you worked on it, and they had answered that question simply as "Yes, it can". Can you recall why that wording was changed from "Yes, it can" to "Almost certainly yes"?

A. I have to be honest, no, I can't recall that at this stage, or even whether I was involved in changing that particular aspect. I'm sorry.

Q. Again, with the benefit of hindsight, or perhaps from your understanding at the time, it would have been more accurate, wouldn't it, if it had simply said, "Yes, it can"?

A. Yes. I mean, you might have couched it as "There are reports of transmission by transfusion".

MS FRASER BUTLIN: Thank you.

Sir, I'm about to move on to another topic and I note the time. I wonder if now is a good time to take a break?

SIR BRIAN LANGSTAFF: Yes. We will do that and come back at 11.40 am.

Can I just say, Professor, what I say to all witnesses at this stage which is that you are giving evidence, what you may not do is talk about the evidence you have given or anything which you think it you may yet be asked about in evidence with anyone, whoever it is, you can talk about anything else you like. But I hope you have a satisfying break and we will be back at 11.40 am, if you please.

A. Yes, sir. I understand that. Thank you very much.

(11.10 am)

(A short break)

(11.40 am)

SIR BRIAN LANGSTAFF: Yes.

MS FRASER BUTLIN: Thank you, sir. Could we have NHBT0017448_004, please. This is a letter, perhaps if we can just zoom into the top half, thank you. This is a letter from Dr Craske to you dated 12 May 1983. Then if we go down to the second and third paragraphs, please, we can see that Dr Craske tells you that:

"From a recent review of the literature I have made, I am doubtful whether the use of a screening test such as [hepatitis B core] antibody will detect some patients early enough in the stage of their disease to remove the risk of transmission of infection by transfusion."

In relation to -- sorry, let me start again.

(Pause)

SIR BRIAN LANGSTAFF: Take your time.

MS FRASER BUTLIN: If I might just get a glass of water that will help. Thank you.

Apologies, Professor Barbara. This is a letter from Dr Craske to you in May 1983 and it is headed "Screening of Blood Donors to Remove the Risk of Transmission of the Acquired Immune Deficiency Syndrome AIDS", and Dr Craske writes, in the second paragraph:

"From a recent review of the literature I have made, I am doubtful whether the use of a screening test

such as [hepatitis B core] antibody will detect some patients early enough in the stage of their disease to remove the risk of transmission of infection by transfusion."

There is then reference to what has become known as the San Francisco baby case. Then he says:

"I think it will be worthwhile investigating the possible use of screening tests which might be found to be suitable at a later date, but I think the only effective way will be to use some sort of questionnaire to donors, and to rely on their altruism and honesty with regard to homosexual exposure."

You, as I understand it, Professor Barbara, didn't agree with Dr Craske's view of the utility of hepatitis B core antibody testing in relation to screening for AIDS; is that right?

A. Could we just go back to that bit of the para where he makes that comment?

Q. Indeed, it is the second paragraph.

A. Because the wording is important:

"... I am doubtful whether the use of a screening test such as [anti-HBc] will detect some patients early enough in the stage of their disease ..."

SIR BRIAN LANGSTAFF: I think he must mean, must he not,

some donors? Because this is talking about the risk of passing it on, isn't it? Have I got it wrong?

A. Sorry, Sir Brian, I missed the first part of your question.

SIR BRIAN LANGSTAFF: I'm sorry. I will just remove my mask. I was just wondering about the use of the word "patients". Because what he is talking about, I think, is avoiding the risk of a donor with AIDS or with HIV infection -- with an infection which may transmit AIDS, passing it on, and when the word "patients" is used here, it is talking about donors presumably, is it?

A. I think so, Sir Brian. I think John Craske is using the word "patient" as a sort of generic term, a potential donor who might actually be a patient.

SIR BRIAN LANGSTAFF: Yes.

A. But my -- what I didn't agree with is the aspect that we are not aiming to detect a donor who is undergoing hepatitis B infection, but we are looking at anti-HBc in the context of a phrase that virologists use about viruses running in packs, a common source of infection for various agents, like intravenous drug use. And my feeling was that there was some possible merit, certainly worth considering, of anti-HBc as an indication of past or present infection with

an agent that could, as it were, co-infect with HIV.

MS FRASER BUTLIN: We have already heard --

A. Sorry -- does that make sense?

Q. Thank you, Professor Barbara.

We have already heard that the North London centre introduced confidential questionnaires given to their donors and that those questionnaires included a box that a donor could tick to say "Please, don't use my blood for donation".

You then tested those donations, didn't you, that came from those donors who ticked that box?

A. Yes, and we would have looked at anti-HBc.

Q. What did you find in relation to the correlation between people ticking that box and the results of hepatitis B core antibody testing?

A. I don't remember the exact figures. Doubtless it is in our literature somewhere, either yours or mine or both, but there was an increased rate of anti-HBc in donors ticking the box.

Q. So it might suggest that hepatitis B core antibody testing was a useful marker to eliminate donors who shouldn't have donated?

A. In the context of HIV, it was a potential surrogate test that could be considered but you would have to also consider the numbers that didn't test positive

and the possibility that, if someone had anti-core, it wasn't caused by a factor that would put them at risk. They may have been born, you know, with an early infection of hepatitis B.

So it was an idea but it wasn't a clear cut idea.

Q. But your view at the time, Professor Barbara, as I understand it, was that that would have been a useful surrogate test?

A. It could have been a useful -- I don't think I ever formulated it in my own head as something that I would definitely want to press ahead with but it was an idea, it was a concept that might have some utility.

Q. And in centres where there was not the opportunity to tick a box on a confidential questionnaire to say "Please don't use my blood", might the hepatitis B core antibody testing have been of particular use?

A. Yes, in the absence of a self-exclusion questionnaire, it may have had some value.

Q. Because it would have picked up those people who you identified where there was a correlation between ticking the box and testing positive on the hepatitis B core antibody testing?

A. Yes, but unfortunately it would also have detected

people who had been infected at birth early in life or soon after, maybe because they were in areas of high hepatitis, came from countries of high hepatitis B incidence.

Q. Once HTLV-III screening tests were introduced, in the North London centre, it is right, isn't it, that when a sample tested positive initially then you would test it in duplicate to assess whether it was a repeat reactive?

A. Correct.

Q. And if it was, then the blood would be collected and held?

A. The blood would be withdrawn from inventory and kept in my laboratory in a designated secure fridge.

Q. And at that point is it right that it would then be tested using the Western blot test?

A. We would send the samples to our reference laboratory -- this was 1985 and before we had our own reference laboratory -- and they would do repeat tests. There would be a Western blot test. And also, in my laboratory, I had developed a modified haemagglutination assay, using re-agents from a Japanese company, Fujirebio, where you could dilute the cells, the red cells, used in the assay, and then put them into V-weld microplates, centrifuge, and you

would get a dot. You would put the plate at a slope and if the dot stayed as a dot, it was agglutination, and if it formed a teardrop streak as those red cells fell down the well, it was a negative. And that enabled us quite economically and rapidly and very sensitively to titrate the positivity. And if you got a titre by this assay of, say, over 1 in 32 or 1 in 64 -- and they could range out to 1 in 1,000 -- you could be -- gives you a lot of confidence that it is a real positive.

Q. And when did that set of testing in your laboratory become available?

A. It was soon after the ELISAs became available, and it was something that we found very useful as an adjunct to the ELISA testing, so it would have been sort of mid to late 1980s.

Q. Do you recall if that testing that you developed was available before or after the first generation of screening tests?

A. It was after.

Q. Can you help us in relation to the Western blot test. In the documents, and we will come to it in a bit more detail in a moment, there is a lot of discussion about confirmatory testing that I understand was done at the Middlesex Hospital?

A. Yes.
Q. Was that the Western blot test or was that some other form of confirmatory testing?
A. I'm pretty certain now that it was the Western blot. And as I have said elsewhere, a straightforward ELISA gives you a yes/no: there is antibody or there isn't antibody.

The Western blot, if I put it this way, allowed you to see the anatomy of the antibody response so you could see which antibodies to which components of the virus were present. And the more -- you would want at least two antibody lines, so the more components of the virus could be detected by the antibodies in the Western blot, the more confident you were of the genuineness of the reaction.
Q. Is it right the Western blot test had been available for some time prior to the ELISA testing?
A. No, I don't think so, because you would have needed to have the virus isolated and in the Western blot you would break up the virus, run it down a polyacrylamide gel, blot off that tube of gel, and get a series of viral antigen lines stuck onto the blot. Then you would add your sample and the antibodies would bind to that.

So you needed to have isolated the virus and

defined it.
Q. But in terms of the technique of the Western blot test, that technique had been developed, albeit in relation to other viruses?
A. Indeed, yes. Western blot was a recognised form of assay.
Q. The first generation screening tests were available from March 1985, but you had concerns about those first generation screening tests in relation, particularly, to false positive rates; is that right?
A. Absolutely.
Q. Given that the technique of the Western blot test was available prior to March 1985, why was that not a solution to the concerns about false positive rates?
A. That's a good question. I presume because laboratories or manufacturers hadn't formulated the Western blot around HIV. But that is my presumption at this time. I can't remember. I -- yes, I -- that is the best answer I can give to that question.
Q. In terms of your concerns about false positive rates, can you help us with what they were particularly?
A. Yes. With the ELISAs that Dr Robert Gallo licensed when he had discovered "HTLV-III", the manufacturers had to use his cell line, which was rich in HLA antigens. I have described elsewhere how the virus

buds through the infected cell and takes with it cell wall antigens, which will contain HLA antigens and when you do your test, anybody who has HLA antibodies would come up as reactive and the reactivity rate could be anything from 2 per cent to 10 per cent of tests and only about 1 in 10 of the reactives would be confirmable as real.
Q. At the time of the first generation screening tests, was your understanding and expectation that confirmatory tests were in the pipeline and likely to arrive pretty soon?
A. We knew they were working on them but, to be honest, I didn't have a timeframe for when they might become available.
Q. You have talked us through your concern about the false positive rates and the possible numbers that were involved. Would you accept that by waiting for confirmatory testing to be available it meant that blood that was infected with HTLV-III was entering the blood banks and out to the hospitals and on to recipients?
A. Yes, I would accept that but I would also make the comment that, initially, the feeling was -- and this was because there was such a long, if you like, incubation period between infection and the

development of immune suppression and the various diseases that that allowed into the patient -- there was a feeling that only a small proportion of infected people would go on to become ill and symptomatic.
Q. Was that a feature of your thinking when you were raising concerns about false positive rates?
A. Yes, that would have been in my mind, as well, yes.
Q. With the benefit of hindsight, might it not have been better to use the first generation tests to enable blood to have been put on hold and then to be re-looked at when confirmatory testing came on stream?
A. With the benefit of hindsight and knowing what we now know about the severity of HIV, I think we could maybe should have examined how we might do that without totally disrupting the management and the supply of donors and donations, because the same considerations apply to HCV, which doubtless you will come onto, and the problem was that, in the UK, certainly we wouldn't exclude a donor without telling them.

If we were to exclude a significant number -- remember just at North London it was 1,000 donors a day -- and if we were excluding 10 to 50 say, just as a figure that might have been, the practicalities of doing that safely and securely would have been very difficult. Then you have got the knock-on effect of

the impact on the supply of available blood for the hospitals and then you would have to work out what you told the donors.

Q. That issue of what you tell the donors was something that was Professor Contreras' role; is that right, rather than yours?

A. I was able to counsel donors -- as I told you, I had initiated the hepatitis B counselling in the service, it was about the first in the country -- with Dr Dane's advice and with any input and help that I needed from clinical colleagues, but mainly Dr Hewitt and a team of other medical doctors would have been doing the talking to the donors. And this would have presumably included talking to donors who may have been infected but we couldn't be sure, which would have been a very difficult discussion to have had.

Q. I want to move on then, Professor Barbara, to hepatitis C at surrogate testing, we picked up some of that earlier this morning and we discussed the centre's use of hepatitis B core antibody, ALT and AST testing in a case of post-transfusion hepatitis.

We move forwards now to 1987. If we could have PRSE0003767, please.

We have here a letter that you wrote with

Professor Contreras to The Lancet, dated 1 August 1987, discussing the testing of blood donations for non-A, non-B hepatitis. It is responding to a letter from Scottish colleagues that the Inquiry has seen previously. In the second paragraph, you and Professor Contreras says this:

"Has the time for a prospective study already passed [in relation to surrogate testing]? This seems to imply that the longer an unproven test is used, the greater becomes the pressure to use it. This is not an argument that should commend itself to those practising transfusion medicine. Why should we have to wait 3-4 years for an answer? If the problem is serious this will be revealed, in acute [non-A, non-B hepatitis], within a year of initiating the study. The need for controlled studies of the incidence of [non-A, non-B] post-transfusion hepatitis will not disappear with the introduction of routine screening of blood donations with tests of unproven value."

Then you carry on to say:

"How far can the argument stretch that 'all known methods' should be used to avoid the risk of [non-A, non-B hepatitis] after transfusion? The bulk of [non-A, non-B hepatitis] may still be transmitted even after surrogate screening. Are we certain that

patients would succeed in a legal action if they contract [non-A, non-B] hepatitis after the transfusion of blood untested for [hepatitis B core antibody]? Why should [non-A, non-B] post-transfusion hepatitis be such a special case that we have to make tremendous efforts to prevent occasional infections?"

Is it a fair reading of this letter, Professor Barbara, that the premise of your argument, yours and Professor Contreras's argument, is that non-A, non-B hepatitis infections were occasional?

A. Yes. I think our understanding at the time was that they were occasional. As you know, we did try and monitor any post-transfusion hepatitis infections that might relate to non-A, non-B. We also had the feeling that non-A, non-B was not as severe as hepatitis B.

Q. Then if we carry on down through the letter to the final three paragraphs --

A. Remind me of the date of this, please?

Q. August 1987.

A. Yes, thank you.

Q. It picks up:

"Transfusion services must not bow to irrational pressure for measures whose efficacy is unproven. In the UK, Transfusion Centre Directors resisted commercial pressure for premature introduction of unsatisfactory

screening tests for anti-HIV. They should show the same resolution with [non-A, non-B] hepatitis."

Before we pick up about the non-A, non-B hepatitis, can you help us with what you were referring to in relation to the commercial pressure for premature introduction of HIV testing?

A. Yes, companies would have been very keen -- the initially licensed companies would have been very keen for the Blood Service to introduce the anti-HIV testing because, of course, it was a huge market.

Q. Then the letter goes on:

"At our transfusion centre, 400,000 blood components are available for transfusion per annum. We have received an average of only four reports of [non-A, non-B] post-transfusion hepatitis annually for the past ten years, and we repeatedly remind clinicians of the need to report infective complications of blood transfusion.

Again, was your view of the value of surrogate testing based on your understanding of the limited number of post-transfusion hepatitis reports from clinicians?

A. Yes, the limited number of reports, our perception of the limited clinical benefit and our awareness of the diversion of resources that introduction of surrogates

would have entailed: cost, in other words.
Q. Given that the centre was using these tests to decide whether to exclude a donor from the panel when there had been a report of post-transfusion hepatitis, why was that not then good enough to apply to the screening of all donations?
A. If you consider the whole range of donations and then concentrate on a report of possible post-transfusion hepatitis, you are, if you like, narrowing down the focus where you can concentrate your efforts and get the most value out of the surrogate testing. So, it is what I said before: you got the smoking gun, so it merited that concentration of effort and that really didn't apply for the whole range of donations.
Q. Was that a resource question, Professor Barbara?
A. It was partly a resource question and that wasn't just in terms of the cost of the test but, of course, the staffing, but it would also involve a diversion -- sorry, a reduction in the amount of available blood for issue and, of course, it would also that mean donor management and donor counselling -- you would have to take that into account because there would have been considerable implications for that as well.
Q. Then if we move forwards to June 1989, NHBT0000076_037, please.

We have a meeting on 9 June 1989 of the "National Study on Surrogate [Non-A, Non-B] Markers in Blood Donors", and we can see just under the stamp on the right that you were present and you were acting as the secretary at that meeting.
Then if we go over the page, as part of the progress reports we see -- sorry, back one page. Should be page 2.
Sir, I'm afraid we have had a difficulty. We don't have pages 2 and 3. It is a very short part of page 2 that perhaps I can simply read into the transcript.
SIR BRIAN LANGSTAFF: I think read it out but take it slowly so that Professor Barbara can follow.
MS FRASER BUTLIN: Absolutely.
SIR BRIAN LANGSTAFF: And if he wants you to repeat it, please just ask, Professor.
A. Thank you.
MS FRASER BUTLIN: There is a heading "NLBTC". So the North London centre. And you are -- it says this:
"Dr Barbara, report F.
"1 in 150 anti-HBc negative donors were repeatedly anti-HCV positive. 2.2% of 64 NLBTC donors with elevated ALT were anti-HBc positive.
"Dr Barbara received additional data from the

centre during the meeting for anti-HCV rates in anti-HBc positive donors. The revised figures for this data following repeat testing is 4.4% of anti-HBc positive donors have given positive results for anti-HCV (1 in 23).
"To date, the anti-HCV test provided consistent results and was convenient to perform."
So as at June 1989 it appears to have been your view, Professor Barbara, that the anti-HCV tests were producing consistent results. Is that your recollection?
A. In terms of repeatability of reactivity. So whether the reaction was real or false positive, it was consistent in as much as it would come up again when you re-tested it.
Q. If we then turn to page 4 of the document, please. We have:
"3.7.4. Donors positive for anti-HCV
"Repeatable anti-HCV positive or 'grey-zone' donors should be flagged, without counselling or notification. Plasma to be stored frozen. Future donations to be treated similarly pending decision on the significance of the anti-HCV assay."
Next to the word "counselling" we can see a little asterisk. And the asterisk takes us to the

bottom of the page.
(Pause)
Keep going further down, please. There's a further heading under the name "Dr John Barbara". Yes, it's at the very bottom there, the heading "Anti-HCV reactions and donor counselling". Thank you.
It reads:
"On reflection and after discussion with Dr Hewitt, consultant in medical charge of Microbiology, and Dr Christine Moore we feel that the anti-HCV results should not be withheld from the donor at counselling, especially if they corroborate one or both surrogate marker findings. Notification would include emphasis that the test is still in the research phase, as they were informed at the beginning of the trial. Findings may not be 'absolute' but are extra evidence suggesting that the donation is unsuitable for transfusion. We think this will reduce rather than increase doubt and worry on the part of the donor. Provided Ortho Diagnostics allow us to do so, we think that the GP should also be informed of these results, as research findings."
A. Yes, I have read that, thank you.
Q. This view that you had taken, together with Dr Raafat,

Dr Hewitt and Dr Moore, appears to suggest that in the --

A. Sorry, could we have that back again so that I can keep -- be able to look at it?

Q. Of course. There we go.

A. Sorry to interrupt. Yes.

Q. It appears from this note in these meeting minutes that you and others had taken the view that, in the context of a research trial, donors could be told about results that were you weren't entirely sure of the significance of; is that fair?

A. Yes. In fact, in a research trial one might argue there was less ethical pressure to inform donors of any, if you like, positive findings, because this wasn't an extant part of our testing repertoire. But obviously on reflection here, even though it was a research trial -- and this emphasises the importance we attach to keeping our donors informed -- even though it was a research trial we obviously had recommended that the donors would be told, with all the emphases that you can see presented here.

Q. And so in the face of uncertainty, you and your colleagues had found a formulation that you felt was appropriate to use with donors?

A. In the limited context of a trial with numbers of

donors that would then be involved. And if you -- you can't really extrapolate that to routine donor screening because the numbers would have been far more enormous. And also, because it was a trial, one would be able to say to the donors, "As a clearer understanding evolves, we will inform you."

Q. In thinking about extrapolating it to all donors, was the difficulty a matter of resources for that counselling to happen?

A. Extrapolating to all donors, yes, the resources would have been an important factor. And as I have said before, the impact on the blood supply would have been significant as well.

Q. Can we turn now to NHBT0000017_006, please.

We can see at the bottom of this page the initials "DH/JAB" and "RME", "23.6.89". The JAB there is you, isn't it?

A. Yes, and the DH is the late Dr Howell.

^Name Check Yes.

Q. So this was a "Preliminary report No. 2" on the HCV assay?

A. Yes.

Q. And if we -- there's then a couple of pages of some tables of results, but if we carry on to page 4 ...

There is another problem with documents, sir.

Page 4 is missing. It is two bullet points that I wanted to take Professor Barbara to, so if I may I will simply read it into the record.

On page 4 there is a heading "General comments":

"1. Test seems reproducible, robust and meaningful.

"2. Confirmation of some sort is obviously required."

Does that accord with your recollection of your views in June 1989 of the hepatitis C testing?

A. That it was reproducible --

Q. Robust and meaningful.

A. As I said previously -- certainly with any test, if you tested and got a result and re-tested and got a different result, you would be very unhappy with it. And that wasn't the case with this, with the assay. So the results you got, whatever they meant, were reproducible, and therefore robust.

Sorry, what was the second point?

Q. It was reproducible, robust and meaningful. And then the second point was the need for confirmation testing?

A. Oh, yes. Yes, that was important.

Q. Can we then pick up NHBT0000188_017, please.

This is a letter to The Lancet from yourself and

Professor Contreras. It is dated 8 August 1989. If we can then carry on down to the body of the letter, please. You say this -- there has been a flurry of publications on hepatitis C in The Lancet:

"We agree that the new Ortho ELISA for anti-HCV clearly appears to be a specific assay for the major agent causing post-transfusion non-A, non-B hepatitis. It is obviously incomparable with any of the previous attempted assays for NANB virus and provides a welcome advance over surrogate markers for infection with this virus. However, in the context of donor screening, precipitate action should be avoided. As in other assay, the predictive value of a positive result hinges on the prevalence of the marker in a given population. While the test scores well in panels of well characterised NANBH sera and in samples from patients with a diagnosis of NANBH, we do not know the predictive value of the test in low prevalence populations such as UK blood donors. In this context, it is essential to have confirmatory assays to eliminate, for example, the possibility of cross reactivity with yeast antigens, before sensible policies for generalised screening of blood donations are implemented."

It carries on. You discuss an evaluation that

has been undertaken and you say:

"Between 0.5-1% of blood donations have been found to be repeatedly reactive (unpublished observations). Excluding this proportion of blood donors might appear to be a minimal problem. However, when related to the annual 2.5 million blood donations in the UK, the problem is certainly not trivial."

You also highlight the enormous and costly undertaking that would be required of contacting and counselling blood donors.

Then, over the page:

"Considerations of the cost-effectiveness of routine donor screening must await the advent of reliable confirmatory tests as well as faster screening tests."

Professor Barbara, this letter was written in August 1989. In relation to confirmatory testing, at that point, were you aware of confirmatory testing being developed?

A. I was aware that people were working on confirmatory testing. I didn't know what stage it was at.

Q. Was your sense that it would be available very soon?

A. In all honesty, I can't recall.

Q. If we just look again at that final sentence of the letter, the considerations of the cost effectiveness

of the donor screening. That appears to rather sum up your concerns about the hepatitis C tests. In relation to the cost, both in terms of the cost of doing the tests, the loss of blood donors and the difficulties in relation to counselling donors; is that fair?

A. Yes. It is worth remembering that roughly all previous screening tests would have cost -- for each test for the up to 2 million donations a year in Blood Service -- would have cost anything up to -- with HIV it became 50 pence a test. The economical assay I developed for hepatitis B was actually pennies per test and Ortho would be charging £2 a test. So, in terms of the overall impact on budget, it would have been enormous.

So I think cost effectiveness did figure very highly in our considerations. Could I also -- if we go back to the beginning of that letter, is that possible?

Q. Page 1.

A. This letter here. If we go right back to the first paragraphs --

Q. NHBT0000188_017.

A. -- second paragraph, if we could, please. Again, with hindsight, our wording would have been better, that

anti-HCV clearly appears to be an assay specific for the major agent causing post-transfusion non-A, non-B hepatitis. And certainly all -- myself and my colleagues were excited at the realisation that this assay would be doing very well in Harvey Alter's panel which was used to test candidate assays.

Previous to this assay, none of them showed any correlation with the status of the samples. So when this assay came it was -- it gave us considerable hope that there would be the ability to specifically detect. But I'm making this point because it wasn't an assay of high specificity, it was an assay specific for that agent.

Thank you for your patience.

Q. Following on from that, given that it was, as you understood it, specific to that agent, would it not have been better to introduce the testing at this stage, even if that had meant that you couldn't have any confirmatory testing?

A. The same arguments applied to what we discussed previously with HIV and, again, the question of the severity and the clinical impact of non-A, non-B was still something that hadn't been fully elucidated.

Q. If we move forwards --

SIR BRIAN LANGSTAFF: May I just ask a question about

this. Can we just highlight, please, the second paragraph again?

Prior to the cloning of what became known as hepatitis C by -- I think it was by the Chiron Corporation, there had been significant discussions about surrogate testing on non-A, non-B, had there not?

A. Yes, sir.

SIR BRIAN LANGSTAFF: There was a difference of view, as I understand it, between those who thought it was worth introducing and those who thought it might not be. Here, the second sentence:

"It is obviously incomparable [that is the Ortho ELISA test] with any of the previous attempted assays for NANB virus ..."

Then this:

"... and provides a welcome advance over surrogate markers for infection with this virus."

It might be thought to follow, that, if it was an advance over surrogate markers -- I mean, do you agree that it was an advance over surrogate markers?

A. Yes, Sir Brian.

SIR BRIAN LANGSTAFF: It would make the case for introducing some sort of test, which otherwise surrogate markers would have indicated, rather

stronger, one might have thought. Would that be a fair conclusion?
A. Yes, I think that is a fair comment.
Q. Perhaps you can help with this, one of the downsides of introducing a test with -- aimed at identifying non-A, non-B, rather than hoping to eliminate non-A, non-B by identifying other markers, is that you would wish to talk to the donor concerned because, plainly, they may have a problem and they have to know about it. Suppose you had had surrogate testing, would that have involved talking to donors whose donations were excluded? You did say earlier, I think, that if any donation was not accepted you would expect to tell the donor that that was the case and broadly why.
A. Yes, Sir Brian. If we were say to have introduced combined raised ALT and anti-HBc testing and then one of the options was to consider anyone who had raised ALT and who was anti-HBc positive as a higher risk for non-A, non-B, then the donations would have been excluded and we would have then told the donor that we were excluding future donations and we would try and explain why, which would have been a bit difficult because we would have to tell them that we were trying to err on the side of safety while we regretted having to lose their current and future donations.

It also would raise the question of whether we then needed to do look-back on the recipients of previous donations from that donor.
SIR BRIAN LANGSTAFF: So if indeed, as you think it was, this was a considerable advance on surrogate assays, is it likely that there would probably be less counselling of those who were not infected than would have been the case with surrogate testing, had it been introduced?
A. Do you mean there would be fewer donors as candidates for counselling because there would have been fewer of them detected by the assay?
SIR BRIAN LANGSTAFF: Yes, I do.
A. I can't work those figures out offhand.
SIR BRIAN LANGSTAFF: Don't try and do it now. If you have a moment after this to think, well, what would the comparative figures probably have been, as best you can tell, do please let us know. I think it is better to do that than try and say something now which may not be right.
A. Yes, so comparing the donor loss with first generation HCV and surrogate testing. It is a very interesting point and I can't remember if it ever came up, it may have done but, yes, thank you for that.
SIR BRIAN LANGSTAFF: Well, thank you.

MS FRASER BUTLIN: I want to move on to the second generation testing.
Could we have NHBT0000191_011, please. It is a letter from Ortho to you, dated 11 January 1991. It says:
"In order that I can ship you the Second Generation RIBA-HCV assay for clinical evaluation, I am required by the US Food and Drug Administration to have you sign and return the attached declaration."
Then, over the page, we have the signed forms that we don't need to go to.
Do you recall whether or not you were, in fact, sent the second generation RIBA assay?
A. I'm sorry, I can't recall. I presume I would have been if I signed it and sent it off and we would have been keen to see it but I can't specifically recall. I'm sorry.
Q. Do you recall doing any testing of the second generation RIBA?
A. I am sure we did but details, no, I cannot recall.
Q. Then if we turn to NHBT0000073_065, please.
We have a letter from Dr Gunson to all RTDs dated 3 April 1991.
Then if we go to the body of the letter please, it says this:

"You will recall that in my letter to you of 15th February I suggested that the 1st July 1991 might be an appropriate date to commence anti-HCV screening of blood donations.
"You may be aware that since the three-centre trial of anti-HCV tests was completed, Ortho and Abbott have produced second generation test kits which have additional antigens to the C-100 of the test we have evaluated. There may also be other companies supplying anti-HCV tests."
Then the final paragraph on this page:
"It is undoubtedly in our interest that this evaluation takes place. However, to complete this study and become operational by 1st July 1991 is too tight a schedule. It is difficult to state precisely a revised date, but I think we should aim to commence routine screening for anti-HCV by 1st September 1991."
As I understand it, Professor Barbara, you agreed with Dr Gunson's suggestion that evaluation was required?
A. Yes, I think evaluation would have been required. If we could go back to the beginning, so that I can get my head around the dates again --
Q. Of course, it is --
A. -- and which tests --

Q. It is dated 3 April 1991.
A. And it is in regard to which generation of ELISA?
Q. If you just go to the second paragraph of the letter --
SIR BRIAN LANGSTAFF: I think it is actually a RIBA test rather than an ELISA.
Q. It is a RIBA test.
A. Ah, sorry, forgive me, I'm getting fuddled.
SIR BRIAN LANGSTAFF: Am I right in thinking that the first generation test was an ELISA test --
A. Yes.
SIR BRIAN LANGSTAFF: -- and the second --
A. And the second generation too.
SIR BRIAN LANGSTAFF: The second generation was a RIBA test, RIBA assay?
A. Sorry, Sir Brian, the second generation test was also an ELISA but it had more antigens on the solid phase of the microplate of the ELISA. The RIBAs were always confirmatory tests.
SIR BRIAN LANGSTAFF: So when we saw the original letter -- if we just go back to it for a moment -- NHBT0000191_011, that we looked at a moment or two ago.
The second generation RIBA HCV assay, the RIBA is a confirmatory test that comes with it or what?

How do I understand that?
A. Yes, Sir Brian. The RIBA, the recombinant immunoblot, is the assay that gives you the anatomy of the stark antibody response that the ELISA will give you. So this was the second generation confirmatory assay from Ortho.
SIR BRIAN LANGSTAFF: Thank you.
MS FRASER BUTLIN: In relation to the second generation ELISA tests in April 1991, is it fair that you and others from a scientific perspective fully expected those second generation tests to show significantly improved sensitivity and specificity?
A. Yes, because the range of antigens on the solid phase was better, we would have expected not only increased sensitivity but increased detection range, so the ability to detect more types of antibody positive samples.
The specificity we wouldn't have known about. It was still on the same basic format, so the potential problems that the first generation assay had could still apply to the second. But if we had confirmatory tests, the supplementary tests really of RIBA -- because RIBA wasn't totally confirmatory, it was supplementary, it was giving you a clearer picture of what the antibodies were. But with that in place

we would have been much more comfortable.
Q. Was there any reason why the testing of the second generation tests couldn't have occurred in parallel with the introduction of testing for all donations?
A. In hindsight I believe not. I have to add, provided we got the RIBAs, yes.
Q. Can I turn now to NHBT0088770, please. That's the fifth page, I need the first page, please. Do we have any further pages?
Dr Barbara, I cannot show you the first page, but there is nothing I want to particularly address on it other than to introduce the document, which is a document published in Reviews in Medical Virology in 1991, the heading of which is "Blood Transfusion Services should have begun screening for Hepatitis C when an antibody assay first became available".
And it is described as a debate I think: "for" with Dr Brown and Professor Thomas --
A. I -- yes, I remember --
Q. -- and you wrote the "against" bit of the article.
A. Yes.
Q. What I'm going to do is pick up the journal article from page 3, please, which is where your writing starts.
A. Okay.

Q. We can see at the bottom left against the proposition and your name.
A. Yes.
Q. And if we go to the top of the second column we can read this -- what I will do, Professor Barbara, if I may, is read some of it and then I will ask you a question about it.
A. Okay.
Q. "One single crucial factor in any decision concerning the introduction of a new (and in this case, very expensive) pre-transfusion screening test for blood donations must be examined: in the absence of screening, can significant transfusion-transmitted disease be associated with the agent in question? This is often the factor that is most obviously prone to geographical variation. Although the data relating chronic liver disease (CLD) to transfusion history are very sparse, striking differences are apparent between different countries."
Then you address a study from Japan which showed what you described as very high figures suggesting an aetiological connection between transfusion and liver disease. Whereas you indicate there that wasn't present in UK studies.
Then if we go down to the next heading, please:

"The rates of PTH in Japan and the UK mirror the extent of the association between CLD and a history of transfusion; while PTH rates in Japan are high, they are very low in the UK."

Then if we go over the page. Under the heading "HCV seroprevalence" you note that:

"Surprisingly, the seroprevalence in, for example, Japan (1.5%) is not markedly different from countries such as the UK (0.3-0.7%) although the rates of PTH differ enormously."

Again, in this article you were making the same link, weren't you, that chronic liver disease is caused by post-transfusion hepatitis. So again in this article you were relying on the reports of post-transfusion hepatitis to get to those seroprevalence -- sorry, to get to the rates of post-transfusion hepatitis?

A. Yes, the incidence of post -- yes. Yes.

Q. Then if we see under the "Assays for anti-HCV" you say:

"The first-generation assays for anti-HCV employed solely the C-100-3 antigen derived from a non-structural region of the virus. Antibody responses to this protein may be more likely to reflect chronic rather than short-term acute infection with the agent. These assays

suffered from the following disadvantages:

"1. Low predictive value in the absence of supplementary tests, which were not available until some time after the screening tests were marketed.

"2. Short-lived antibody in a significant proportion of subjects; in some cases this may reflect false-positive, rather than genuine transient reactivity."

A. Did you want me to just talk to that briefly?

Q. If I just go through to point 4 so that we have it.

3 is the:

"Long delay until seroconversion, following infection ..."

A. Sorry.

Q. And 4 is the:

"Low titre of the anti-C-100-3 response."

What did you want to add to that, Professor Barbara?

A. Sorry, if we go back to the first two, points 1 and 2. Yes, the C-100 antigen, this point about it being from a non-structural region, this is the -- relates to the fact that when a virus replicates in its host cell, there will be proteins, antigens made as part of the virus production process that are not then incorporated into the actual structure of the virus.

And in terms of tests for detecting antibodies to a viral infection, you get better antibodies to the actual virus bits itself, the structure. You have -- you know, if you imagine the virus floating around in the bloodstream, it is going to elicit more of a vigorous antibody response than the little bits of non-incorporated, non-structural antigen that are left in the infected cell.

So, that was one point I wanted to make, that although the antigen was specific to hep C, it wasn't the best -- it wasn't the choicest antigen to have derived.

Because of what I've just said and -- the fact that you haven't got the vigour of antibody response, if you had an acute infection you are going to be less likely for an antibody to C-100 to be positive than if you had a chronic long-term infection where cells would have been continually producing the non-structural proteins. So, I just wanted to make that point clear there.

Q. Picking up on that issue of antibody responses maybe more likely to affect chronic infection, did that give you pause for thought that perhaps acute symptomatic post-transfusion hepatitis was not a good marker to use for future issues with chronic liver disease?

A. That's a good point. I'm not sure that I'd worked through that logic.

Q. Particularly in light of the disparity between the rates of post-transfusion hepatitis in Japan and the UK?

A. Yes, I suppose that could have been a factor that one would have to consider. I would really have to think about that.

Q. If we carry on then in this article to the conclusions, page 5, please, we can see that you have summarised the reasons for not initiating anti-HCV screening as soon as tests first became available as follows:

"1. No evidence for an association of transfusion and [chronic liver disease]."

Was that your view in 1991, that there was no evidence for an association of transfusion and chronic liver disease?

A. Yes. I think that it was. One didn't link chronic liver disease with transfusion in any significant degree.

Q. Then:

"2. Very low rate of [post-transfusion hepatitis] and transfusion-transmitted HCV infection."

I think we have already talked about the rates

of PTH a number of times:

"3. Defects of the first available assays (low predictive value in low-prevalence populations; possibility of false negatives).

"4. Absence of supplementary tests initially.

"5. Enormous workload and cost implications with the risk of diversion of resources from existing screening programmes and the resulting dilution of their efficiency."

Did these remain your views as to why tests were not introduced when they first became available?

A. Yes. As to why tests were not introduced when they first became available, yes.

Q. Can I then turn to NHBT0036250_025, please. This is an attendance note taken by solicitors in December 1999. We can see that you attended their offices from 12.25 until 4.30 in the afternoon and what we have got here is a six-and-a-half-page note, so it is a very condensed note of what I understand was a lengthy conversation; is that right?

A. I guess so. It is a way back, so these would have been condensed notes, yes.

Q. Can we put up the third, fourth, fifth paragraphs, please. "He said", I think that's you, Professor Barbara:

"He said that he felt that they were an open target [this is in the context of the litigation] because he thought they could make an issue of the decision not to use anti-HBC and ALT, again about the decision not to use the first generation tests. He thought that the gap between the introduction of the second generation tests and screening which had been introduced was indefensible. He did not know what he could say about that."

Just pausing there, is that still your view?

A. This is the summary that a legal individual would have made of our quite protracted discussions, yeah? This is what was summarised?

Q. It is an attendance note with the solicitors and, as I said at the beginning, we recognise it is a very condensed note of a lengthy conversation.

A. Okay.

SIR BRIAN LANGSTAFF: Just for my interest, the interview took about four hours but how long is the note, how many pages?

MS FRASER BUTLIN: Only six and a half pages, sir.

SIR BRIAN LANGSTAFF: I see.

A. It really is hard for me to remember at this stage but I think this would be a fair summary of what I would have said and both from a strictly legal point of

view, and also from a scientific point of view, my personal feeling at that time was that a gap between the introduction of second generation tests when they became available, and then when actually screening was started, would have been indefensible in the legalistic term but also in the scientific term, that if we had a more specific test then why weren't we using it?

Q. You go on to say they did -- or the note records a summary of your conversation as follows:

"They did not go for first generation tests because of the cost benefit, the lack of scientific evidence and the disruption to the blood supply.

"First there were the first generation tests and there was a lack of confirmatory tests and then the second generation tests. There would not have been any reason other than a timetable as to why the second generation tests were not used and he found that hard to justify."

A. Yes.

Q. Then:

"He would say that they never believed that Hepatitis C was that serious but he thought it would be difficult to hold to that view when the 'rarities' were in front of him."

Is that your recollection of what you explained at that time?

A. Yes. I mean, I think that's a fair recollection of what was -- what I was -- was there at that time, yes.

Of course, when you concentrate -- if you suddenly bought a Skoda car, you begin to notice that everyone else seems to be driving Skodas. And so if you home in on a specific issue like serious cases of hepatitis C, it then becomes a more significant question. I can understand that.

MS FRASER BUTLIN: Sir, I'm about to move on to a different topic. I have three short matters I need to address with Professor Barbara and then obviously we will need to invite any further questions from the Core Participants.

SIR BRIAN LANGSTAFF: When you say short matters?

MS FRASER BUTLIN: I think I will be more than ten minutes, sir, perhaps 15 or 20, but my timing, sir, unfortunately is, as with many counsel, never very reliable. I may be shorter, I may be longer.

SIR BRIAN LANGSTAFF: Yes. I think in the light of that, what we will do is we will take a break now for lunch, come back at 2.05 pm, if that's all right with you, Professor, and finish off those questions. Chances are we will have another break some time after 2.30 or

thereabouts, and that will probably be the last break, as such, in the day I would expect.

A. I hope I'm not adding to any delays by my somewhat confused responses.

SIR BRIAN LANGSTAFF: No, no, no! Good heavens me, don't worry about that at all! The important thing is to get your evidence as clearly as we can and as well as we can, and we have the day set aside to hear you, so you need not worry. We are making very good progress I think. So, thank you.

A. Thank you, sir.

SIR BRIAN LANGSTAFF: 2.05 pm.

MS FRASER BUTLIN: Thank you.

(1.08 pm)

(Luncheon adjournment)

(2.05 pm)

MS FRASER BUTLIN: Before we pick up, Professor Barbara, the three final topics I want to discuss with you, we were discussing earlier today various matters addressing sensitivity and specificity of the testing and I have just been asked to highlight that Professor Barbara's witness statement deals with the scientific details of that to quite a considerable extent. I'm not going to take him to it but just to highlight for Core Participants that it is there in

his statement.

SIR BRIAN LANGSTAFF: Yes, thank you.

MS FRASER BUTLIN: The first matter, Professor Barbara, I want to ask you about relates to hepatitis B core antibody testing not as a surrogate test for hepatitis C but in the context of a study that was being undertaken in relation to its use to reduce hepatitis B transmission.

Firstly, were you aware of that study taking place in the early 1990s?

A. Sorry, to reduce the risk of hepatitis B?

Q. That is right.

A. There were lots of studies in that regard and I was certainly very interested in the idea of anti-HBc to detect what I called tail end carriers who were at the end of carriage but might have some residual infectivity especially in a large volume of a donation.

Q. Can we turn then to DHSC0004709_153, please.

A. You will forgive me if I have this heated wheat bag on my neck. I have developed a very stiff neck.

Q. Of course.

We have a letter here from Harold Gunson dated 7 October 1993 sent to all RTDs/Chief Executives about anti-HBc testing of blood donations and it addresses

the decision of the MSBT not to pursue routine testing of all blood donations for anti-HBc.

The letter then says this:

"This decision was reached after consideration of the following:

"(i) All ELISA tests for anti-HBc gave false positive results; even the more specific tests the false positivity rate appeared to be in the order of 10-fold.

"(ii) There were no agreed, satisfactory confirmatory tests which means that there be an inability to provide definitive health information to a considerable number of donors."

A question arose in earlier Inquiry hearings about this point, if there were no confirmatory tests how could it be known that the ELISA tests gave false positive results? Is that something you can assist us with?

A. Yes, I have written an email to the solicitors helping me, Vicky Morris, and I believe they were trying to get this through to Sir Brian.

Q. Perhaps you can just explain for us now verbally, if you can, how it comes to be said that you have false positives without a confirmatory test?

A. Yes. There is no single confirmatory test for anti-HBc and laboratories working on this would have

had access to a battery of tests, they would have initially tried to detect any HBsAg with as sensitive a test as possible, which eventually of course became PCR. They would also titrate the anti-HBc to see how strong the reaction was and of course the stronger reactions were more likely to be real. They could then also test for anti-HBs because the presence of anti-HBc could either reflect continuing infectivity with undetectable HBsAg or it could reflect clearance of virus with undetectable anti-HBs. But if an anti-HBc positive donor had anti-HBs at a level of 20 milli-IU per ml that would be considered protective or immune and it would also confirm the anti-HBc result.

You could also test for this other marker, anti-HBe, which is -- e antigen is part of the different antigens in hep B and e antigen is associated with the icosahederal virus with the core of -- the icosahederal core and because it is associated with the virus, it is also associated with infectivity. And anti-HBe is what develops when the virus is cleared.

So with this range, this battery of tests, you could get a pretty good idea if an anti-core reaction was real. Now, of course, you couldn't do this in

a routine setting because it is not a single confirmatory test and indeed, for research purposes, you could even ask the anti-core positive individual if they'd agree to be vaccinated when hep B vaccine became available and if there was undetectable anti-HBs that would, of course, have been boosted. So it was this battery of laboratory and almost research tests that could give us the handle on what was real or not.

Q. The second topic I want to discuss with you is --

SIR BRIAN LANGSTAFF: Can I just summarise for the sake of perhaps the understanding of those who may not have had the advantage of reading your statement first, in which you set out quite a bit of this.

What I think you said, but tell me if I have got it wrong, is that there wasn't a single test that you could use, ie as a confirmatory test, but there were a number of different tests which shone a light upon different aspects of it, together, if you used those tests you could say with a fair degree of certainty you have got the virus, or the antibody?

A. That's absolutely right, Sir Brian, yes.

SIR BRIAN LANGSTAFF: Thank you.

MS FRASER BUTLIN: Moving on then, Professor Barbara, to my second topic. You have said in your statement that

you built and maintained relationships with the manufacturers and suppliers of test kits. Can you help us with what that involved, how you built and maintained those relationships?

A. Well, of course, for hepatitis C because we had -- I could identify some donors who were very likely to have transmitted non-A, non-B, and we detected that through surrogate testing, the fact that they came up in at least one, sometimes two post-transfusion non-A, non-Bs -- we saw that paper about a donor implicated twice -- we were able to provide plasma to Professor Richard Tedder and, working with Wellcome Laboratories, as they were then, they produced an independent British clone. So that was a close working relationship with a commercial company.

Then, for enhancement of tests, for tweaking of tests, for any feedback that we could give to manufacturers to make tests better, easier, quicker, we would certainly want to do that, and I also got companies to put in reagents to show us when a serum sample had been added to a diluent. For a surface antigen test you just added undiluted serum and you could clearly see the sample in the microplate well. But when you were adding, say, 10 microlitres of serum

sample to 100 microlitres of diluent, you couldn't be sure you had added that, and manufacturers developed -- they tweaked their assays so they could add chemical reagents that didn't affect the test but that changed colour when you added sample. And also they added colour to all the other reagents so that you could tell that you had performed each stage, the five stages or so of an ELISA assay.

So the relationship -- I never thought of "them" and "us", I always felt that we were working together and the more help we gave them, the better the test would be. You know, to enhance patient safety.

Q. Can I turn now to a letter.

SBTS0000067_087.

It is a letter written by John Cash to Harold Gunson dated 7 September 1992. The substance of the letter reads as follows:

"As a consequence of a number of consultations over the last months it is clear to me that throughout the UK BTS, and perhaps beyond, there is growing concern that there may have emerged a small cartel which is manipulating the development of microbiology donation screening test evaluation (and the to some extent confirmation testing).

"This manipulation is perceived to be directed

towards the personal research interests of Dr Barbara, Professor Tedder and Dr Mortimer and not necessarily the best interests of the UK BTS. More recently concern has been expressed that some or all of these individuals may be inappropriately associated with certain commercial manufacturers of microbiology donation test kits, such that their professional advice is being influenced by the interests of the commercial organisations with which they are associated.

"These are weighty matters and of a very serious nature and I'm bound to advise you that very recent events have led me to share many of these concerns."

Then over the page:

"It is my earnest hope that as we move forward to an NBA that the concerns expressed above can be appropriately dealt with."

First of all, were you aware of this letter at the time or shortly after?

A. No.

Q. And were the concerns --

A. Harold Gunson never showed it to me. Never brought the issue up.

Q. That was my next question. Did he raise any of the concerns that are written in this letter?

A. No. And with all due respect to the late

Professor Cash, I think Harold thought they were rubbish. I can honestly say they were totally unfounded. Myself and the colleagues mentioned there did indeed work with manufacturers for the reasons that I have already said. As a patent holder -- as a co-patent holder for hepatitis C, I never received any of the royalty monies. They went to the centre, actually into my laboratory, to purchase enhanced bacteriology screening test, which eventually became the basis of our bacteriology reference work.

It is also of interest, I can't remember when, but Professor Cash offered me the job of running the Scottish National Micro lab. Without interview. It was just a straight offer. So I don't think he could have thought too ill of me. But when I saw this first, it came as a total surprise. And totally unfounded as well. Sorry if I'm a bit passionate about that.

Q. I want to then move to my last topic, Professor Barbara, and that is in relation to organisational matters across the Blood Transfusion Service.

In your statement you have said that you feel that the North London centre was sometimes held back by slower centres. Can you help us with what you were

referring to there?

A. I think sometimes we had developed initiatives and ideas, like, for example, the AIDS questionnaire or the self-exclusion questionnaire, which we thought were very relevant and significant, and I think other centres felt we were being over the top and being too intrusive. And there were issues like this where we might have wanted to introduce concepts, set up national registers, which sometimes we got some resistance about.

Q. Was your perception that that slowness changed, either for better or worse, when the National Directorate was set up?

A. I think all I can say there is when the National Directorate was set up, we had at least got a unified service that, if you like, was all singing to the same hymn sheet. So I think that, provided that we had the platform to put our ideas forward, they could be considered nationally, and if it was felt there was something relevant, then, that could then be initiated. So I think it probably helped.

Q. You have also said in your statement that you think there was a gradual drift in attitude and direction towards a more precautionary approach, and you used the word "drift" rather than "shift".

A. Yes.

Q. Why was that? Why did you feel it was a drift rather than a shift?

A. Well, if you use those words in relation to the evolution of flu variants, year-by-year you get slight changes, and that's a drift. Every now and again you get a massive change, and that's a shift. So I don't think there was an immediate massive change. I think that as a growing awareness of the relevance of transfusion-transmitted infections that came about, for example, with the development of SHOT and the better reporting of hospital -- from hospitals of transfusion infections, I think people became aware that one needed to do as much as one reasonably could to prevent or reduce the risk from transfusion infections.

With the advent of variant CJD and the precautionary principle as espoused by Mr Frank Dobson, who was secretary for health, when he came to talk to us at a British Blood Transfusion Society annual meeting, he basically was saying that the precautionary principle ruled, and with the interventions for variant CJD you will be able to see that if anything was thought that it might help, it was introduced.

Q. Given the enormity of the AIDS crisis, why did a shift in attitude not take place at an earlier date, from your perspective?

A. In relation to HIV --

Q. Why did that --

A. -- did you say?

Q. Yes. In relation to HIV, why did that not mark a point when a shift in attitude and direction took place?

A. I think in large part because it took us quite a while to perceive just how uniformly devastating an HIV infection was. Because, as I said before, it could take years before immune deficiency developed and before that showed itself as opportunistic infections in often very horrible, very aggressive Kaposi's sarcoma, Cytomegalovirus, otherwise very mild, that would prove very fatal, toxoplasmosis.

I think it took quite a while to understand that HIV was not just horrible in certain cases but it was uniformly devastating.

MS FRASER BUTLIN: Sir, those are the questions I have for Professor Barbara. I have had some questions already from recognised legal representative but I recognise that we should take a break to allow them to send in any further questions.

SIR BRIAN LANGSTAFF: Do you have any sense of how long you might need?

MS FRASER BUTLIN: I think only ten minutes, sir.

SIR BRIAN LANGSTAFF: Let's take quarter of an hour and come back at 2.45.

Professor, what happens -- forgive me for talking with my mask -- now is that Core Participants, whose representatives have been listening to what you have said so far, may have questions which they want to put to you, and they put those through counsel. She must first of all, obviously, be told what those questions are, and that's what this next period will give a chance to happen.

So we will come back at 2.45 pm. I can't tell you quite how long we will be, it could be very short, it could be quite a long time, it all depends how many questions there are, of course. But 2.45 pm.

A. Could I just interrupt, Sir Brian?

SIR BRIAN LANGSTAFF: Yes, certainly.

A. You had asked -- you raised the interesting question which I've sort of paraphrased as: if we had introduced first generation anti-HCV ELISAs, would we have lost any more blood or donors and caused ourselves any more problem than if we had introduced surrogate testing?

Looking back at my very brief notes, depending on where you set cut-offs for raised ALT and anti-HBc, and if we were to use both those markers, I think we would have probably had less of a problem with first generation anti-HCV. But that wasn't a question that had arisen before. So it certainly is an interesting point.

And the other thing I remembered was that, actually, if you had taken appropriate cut-offs for ALT and anti-core and excluded donors who were both -- only excluded donors who were both anti-HCV pos and ALT raised, you were approaching the predictive value of real infectivity as you did with the first generation anti-HCV ELISAs.

So in retrospect, and with the benefits of hindsight, these are quite -- do prove indeed very interesting. So yes, thank you for bringing those up.

SIR BRIAN LANGSTAFF: Thank you for that answer. It confirms what I suspected may be the case but I'm not qualified to know, this is why I asked you. So thank you for that. That's fascinating. I shall have to work out what I make of the implications of that answer but thank you for it.

2.45 pm.

MS FRASER BUTLIN: Thank you.

(2.31 pm)

(A short break)

(2.45 pm)

MS FRASER BUTLIN: Thank you, sir.

Dr Barbara, I just have a few questions that others have raised for me to ask you about.

First of all, you suggested that Dr Cleghorn was a visionary because he recognised that rather than being a nuisance that you had to get round, he was aware that transfusion-transmitted infections would become an increasing problem. To what extent did other directors of Regional Transfusion Centres feel that dealing with transfusion-transmitted infections was a nuisance to get around?

A. Well, I think that the obvious expertise in blood transfusion was a haematological one. And I'm not sure that all the other directors, at least in the early days, recognised the potential significance and risk to patient safety that blood-borne agents might pose.

Again, I'm probably being somewhat flippant in my comments and I apologise for that, but I think that there wasn't the recognition of the significance and the impact that things might have and then one thinks about HIV and the concerns of variant CJD and, yes,

I would stick to my choice of words as visionary. I think that, over time, all transfusion directors did recognise the importance and I wouldn't want to dilute that but, for me, Tom Cleghorn could see it very clearly and of course that was, in part, because of his close association with Dr David Dane.

Q. Could we have CBLA0000043_040, please.

Professor Barbara, this isn't a document that was specifically flagged to you in advance but I'm not going to ask you about the detail in it. Given the significant contact you had with PHLS and CDSC were you aware of this letter, sent by Spence Galbraith, on 9 May 1983 to the Department of Health and Social Security?

Let me read to you just the core section of it. It indicates that Spence Galbraith had:

"... a case of the Acquired Immune Deficiency Syndrome in a haemophiliac in Cardiff who had received USA factor VIII concentrate was reported."

He then identifies that he's reviewed the literature, and this is the part I want to highlight to you:

"I have reviewed the literature and come to the conclusion that all blood products made from blood donated in the USA after 1978 should be withdrawn from

use until the risk of AIDS transmission by these products has been clarified."

I just want to ask you, Professor Barbara, whether you were aware of this letter at that time?

A. No, I wasn't aware of it.

Q. Moving on then to non-A, non-B hepatitis, I'm asked to explore with you whether you ever tested your views on the clinical severity of non-A, non-B against the views of hepatology colleagues who were dealing with the effects of it from a clinical perspective?

A. Yes, I understand the relevance of that.

I suppose we tended to live in slightly different worlds and we were aware, and became increasingly aware at symposia and seminars, of the concerns of clinical colleagues. I think to some extent myself and others in the Blood Service would have taken a view that if you spend your working day dealing with a particular condition, then you tend to think that that condition is prevalent everywhere. So I think I'd have to say that we didn't really test those views against our clinical colleagues. We did have in mind what they said and became increasingly aware of the actual relevance of a -- I believe small, but real proportion of cases of infection.

Q. Do you agree with Professor Contreras' evidence that

if there had been some sort of central advice from the Chief Medical Officer, this may have assisted you in having earlier understanding of the significance of the risk of non-A, non-B hepatitis?

A. Yes, I could see the benefit of that. Yes.

Q. Can we turn then to NHBT0007639, please.

These are minutes of the Working Group on Microbiology held on 15 January 1988. If we go over to the second page, please, we have paragraph 2.3. The minutes record:

"There is also a need for a central authority/body/institution to collect and coordinate information on, and give guidance on, quality control matters.

"Such a centre could record and provide information on data on supplies and quality of products in various RTCs; on reactions and diseases (eg malaria) developing in recipients of blood, blood components or plasma fraction products.

"Advice on special matters such as how to take blood from infected persons could be provided. It is not clear at present where the responsibility for checking the quality of blood packs, or for the quality of bulk plasma lies. In charge measure this must be at the Transfusion Centres where plasma is collected, but

the Plasma Fractionation Centres had also to check their bulk source material in whatever respects were possible."

In relation to the suggestion that there was a need for a central authority, et cetera, to collect and coordinate information, including on reactions and diseases that were developing, can you confirm that there was no body or group that carried out that function centrally at that time?

A. I believe there was not.

Q. What can you recall about North London's practice, if any, of informing the CDSC about post-transfusion hepatitis cases?

A. From when I started, again, because of the clear advice from Dr Dane, I would provide an annual report of our investigations, in conjunction with the Middlesex, on -- investigations into post-transfusion infection. And I would send a copy of this report to I believe then it was Dr Sheila Polakoff at CDSC. So this would be a regular and recognised exchange of information.

Q. What then would have been the benefits, do you think, of having a central body or group collecting and coordinating that information?

A. I suppose that procedure could have been rolled out on

a national basis, with a consistent approach across all blood centres.

Q. And with the benefit of hindsight, do you think such a national body, centralised body, would have led to improved data and understanding on a national basis of the incidence of post-transfusion hepatitis?

A. I think, with hindsight, it might well have done. I notice it covers quality control matters, and I must mention that NIBSC, the National Institute for Biological Standards and Control, also provided analyses of our routine QC test results on our tests for all agents, and we would get monthly reports on this, and again there would be seminars that were organised by NIBSC. Dr Ferguson -- Dr Morag Ferguson and myself used to arrange these jointly. So there was the developed and improved national quality control coordination.

Q. We looked at a letter earlier that you co-wrote with Professor Contreras to The Lancet in August 1989, in which you referred to the UK donor population as being a low prevalence population in relation to non-A, non-B hepatitis. It is a point we have discussed a few times, Professor Barbara, but I have been asked to clarify whether your reference to a low prevalence population was simply based on the low rate of

post-transfusion hepatitis reports that you received?
A. Yes, I suppose it must have been mainly due to that. And probably because of a feeling that there was a low incidence of reported cases of non-A, non-B in the general population.
Q. And finally, in relation to the hepatitis C assays, following your assessment in 1989 that the tests of the assays constituted a welcome advance over surrogate markers, was any thought given to using the first generation of assays in conjunction with surrogate testing?
A. I don't believe there was.
MS FRASER BUTLIN: Thank you.
Sir, there are no further questions that I have been asked to put to Professor Barbara. Is there anything, sir, that you want to raise?

Questions from SIR BRIAN LANGSTAFF

SIR BRIAN LANGSTAFF: Yes. Really two ends almost of the time spectrum, but the first in relation to hepatitis B and, given your interest in microbiology and virology and your knowledge of Dr Dane, you may be able to help with this.
When the screening test for hepatitis B was introduced in 1972, the first generation anyway of the tests had, as I understand it, a reputation of not

being particularly accurate, is that fair or not?
A. The immunodiffusion assay, Sir Brian, were accurate in as much as they provided a built-in confirmation by looking at the way the immunoprecipitant lines formed and whether you got lines of identity with new reactive samples. But it wasn't particularly sensitive. So I think I would say it was accurate but not sensitive.
SIR BRIAN LANGSTAFF: So it missed a number of cases?
A. Yes.
SIR BRIAN LANGSTAFF: That went on through the -- did it -- in 1975 the -- was it the RIA test or -- came in or maybe the -- (overspeaking) --
A. Or the haemagglutination test.
SIR BRIAN LANGSTAFF: Yes.
A. If I may say, the sequence was: initially it was immunodiffusion, and then an electric current was passed across the gel and you got what was called end osmo-immunophoresis, which was like immunodiffusion but made it faster, which of course helped in the very manual testing, the routine screening for release of blood within a half day. And I think it was slightly more sensitive. Then haemagglutination tests came in. Then radioimmunoassay came in. Then the ELISAs came in because of the concerns of using radioactivity,

iodine-125.
SIR BRIAN LANGSTAFF: At what stage did it manage to reach the level of universal or pretty universal detectability, the improved sensitivity of the test?
A. Again, from memory, the haemagglutination test -- actually, especially the enhanced haemagglutination test that I developed and that most centres in the country took up, would have detected between 90 and, say, 93 per cent of positives. Then the radioimmunoassay would have detected from 93 to 97, 98 per cent. So it was a very sensitive test and the ELISAs similarly, but then, of course, with PCR you were able to detect practically everything unless it was very early in the window period and with any viral infection there is a phase called the eclipse, where no test is going to be positive but given a donation given by the pint, as it were, such a large volume of inoculant, there may have been some residual infectivity.
It was a gradual and steadily improving process and it was lovely to see how the sensitivity could increase and of course when we had the availability of seroconversion panels, we could measure, very accurately, the sensitivity of the different tests and the tests from different manufacturers and, again,

that was so gratifying because it just made you sleep easier when you knew that your day's testing was going to be really much more reliable.
SIR BRIAN LANGSTAFF: The second end of the spectrum, as it were, which I want to ask you about is in respect of hepatitis C. My understanding is that the cloning of the -- the sequencing of the genome of the virus was first told to the world in a press release in 1988, 10 May 1988.
Now you won't remember the date, I'm quite sure, but you might remember the event. It was some time after that before the details, I think, were published, at the same time as it was suggested there was an assay which the Chiron corporation had available for use to detect the virus.
What was the reaction -- your reaction first and secondly the reaction of those around you -- to the announcement that there was a claim, at any rate, that whatever it was that was causing the large proportion of non-A, non-B hepatitis had been found and identified?
A. So virologists' equivalent of joy, Sir Brian, and a relief as well that, at last, this elusive and nebulous agent -- I don't normally like American phrases because they are clumsy but non-A, non-B

absolutely described what it was. It wasn't A, it wasn't B, we didn't know what it was. And here was clear evidence from Harvey Alter's very testing panel of samples, that when the Chiron assay, or the partners also who produced the ELISA, when they ran this there was such a predictive value in the assay for those samples, which were going to be -- a high proportion were going to be really, HCV positive.

And it was great relief that from this sort of side-field approach of cloning there had come an assay that was going to really get a handle on this condition, non-A, non-B hepatitis.

SIR BRIAN LANGSTAFF: What do you recollect happening in the period between the press announcement that it had been discovered -- and it was very nearly a year later I think that the actual details were published. What was happening in that period of time? What was the community doing?

A. I think that Chiron Corporation would have been confirming those findings. Of course, companies like to do a press release to generate interest. But for scientists, they would have wanted to be rock solidly sure that that was real. Then they would have been trying to formulate assays that were going to be robust, reliable, as sensitive as possible, as

specific as possible. And of course there would have been trials of any reagents produced. So this would have been what was going on in that time. And, you know, the scientific and medical community would have just said: well, we will have to wait because we know these things do take time.

SIR BRIAN LANGSTAFF: You would have known it was coming at some point?

A. We would have known that a test -- once you clone an antigen, we would have known that that would eventually form the basis of some sort of assay.

SIR BRIAN LANGSTAFF: You would think it was probably a credible press release because it had -- amongst other things, I think it mentioned the name of Harvey Alter?

A. Yes. Yes, yes. Harvey, of course, his panel, was instrumental in validating the validity of that. So that was a press release I could have believed in, yes.

SIR BRIAN LANGSTAFF: To what extent do you think part of the delay may have been the patenting of Chiron Corporation of an assay that could then be sold around the world?

A. I think that could have been a considerable part, because the patent was absolutely rock solid, and it

in effect blocked anyone from doing any research that wasn't based on their clone.

So one of the concerns we had was that there could be other -- other variants -- and we know about viral variants these days -- but the whole assay was based on this one particular variant, if you like, and we were concerned that the patent was so solid that it would block further progress.

If I may just add, when myself, Peter Glazebrook, Richard Tedder patented the, if you like, British HCV assay that Wellcome Diagnostics produced, we had hoped that there would be leeway for that to be used because it -- that clone contained a structural antigen and that assay, although it was the first generation of that assay, was actually more specific than the first generation Ortho assay, which didn't have any structural antigens. But in the end the patent court found against us. Which, I suppose, with the nature of the patent, was almost inevitable.

SIR BRIAN LANGSTAFF: Yes. So the market, in effect, was cornered, was it?

A. Absolutely, sir. Absolutely topped up.

SIR BRIAN LANGSTAFF: And having been cornered, I suppose that Ortho, whose test by then it was, would have expected to sell it around the world, I suppose?

A. Yes. And they only -- if I may digress, they only licensed other companies to produce HCV antibody assay based on the Chiron clone -- if those other manufacturers had some hold on Ortho, so if they had licensed something to Ortho, they would expect to have the HCV licence cross-licensed to them. So you had UBI and you had Sanofi Pasteur, but Wellcome didn't have any such thing, so there was no bargaining power in that aspect.

It also meant that they could charge a price that was at least four times greater than any of the other routine screening assays that we were able to negotiate in the UK.

Sorry, you have triggered emotions, Sir Brian.

SIR BRIAN LANGSTAFF: Yes, I can see that. But I suppose it leads to the next question, which was: if that was the case, and if I think it was April 1989 that the test and the assay was announced in -- or published in science, and therefore known to be available, and the methodology was understood, there was a test which could have been used?

A. Yes.

SIR BRIAN LANGSTAFF: It wasn't a question of someone saying, "Look, we have just found" -- as it was with HIV -- "We have found the virus, now we are going to

find a test for it"? The test was, as it were, readymade.

A. When that was announced, yes, '89 -- and I know a lot of countries started it in 1990, but as I have laid out and my colleagues have laid out, our concerns were the prediction which was borne out of the poor specificity because of the cell line used and the anti-globulin format which lent itself to false positives and the lack of the confirmatory test. But yes, there was a test.

SIR BRIAN LANGSTAFF: Thank you. That is all that I have to ask.

It may be some questions arise out of that, Ms Fraser Butlin, I don't know?

MS FRASER BUTLIN: I don't think we have had anything. No, sir.

Professor Barbara, is there anything else you would like to add before we finish?

A. Oh, in relation to the whole session?

Q. Indeed.

SIR BRIAN LANGSTAFF: Or anything that you want to say. We always give -- every witness has an opportunity to say whatever they may feel moved to say at the end of the evidence which they have given.

A. If I may then, if you will forgive me from reading out

a statement.

SIR BRIAN LANGSTAFF: Go ahead.

A. So I would like to finish by saying that I have tried to the best of my ability to assist the Inquiry on behalf of infected blood recipients and those who have been adversely affected. The only goal of myself and my colleagues was to help patients who might require blood as best we could. As a life-long blood donor, accredited with more than 250 donations of blood and platelets, this too reflects my goal (unclear).

Naturally I completely sympathise with any recipients of blood who have been harmed by transfusion of blood components or products. I deeply regret the suffering or harm caused to patients and their families by any inadequacies in the provision of what was intended to be life saving or life enhancing transfusions. I would have wished that this Inquiry could have happened sooner. This would have enabled the inadvertently but tragically harmed patients to have some redress and justice for what happened to them. During the course of the Inquiry, the injury they have suffered has been made so movingly clear.

I wish it could have been possible for me to attend the Inquiry in person and I very much hope that my evidence will have helped the Inquiry along the

path to completing its crucially important work.

SIR BRIAN LANGSTAFF: It has most certainly helped. It helps us enormously to have different perspectives because there are different perspectives on what took place and yours is, if it isn't unique it is very close to being unique as being the only microbiologist in a Regional Transfusion Centre, employed as such for many years. And the insight which you have given from that particular point of view, it is absolutely plain that you are devoted to your subject and to the science of it and it is very good of you to be prepared to give us the benefit of that and I'm very grateful. Thank you.

A. Pleasure, sir.

SIR BRIAN LANGSTAFF: And I hope that it hasn't been too painful an experience and that your neck is, as it were, warming up a bit.

A. Thank you for that.

MS FRASER BUTLIN: Tomorrow, sir, we hear from Dr McClelland.

SIR BRIAN LANGSTAFF: Yes, and that is which Dr McClelland?

MS FRASER BUTLIN: Brian McClelland, the Scottish Dr Brian McClelland.

SIR BRIAN LANGSTAFF: So Dr Brian McClelland tomorrow,

10 o'clock. Thank you.

(3.18 pm)

(Adjourned until 10.00 am on Thursday, 27 January 2022)

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