**Thursday, 13 October 2022**

**(10.00 am)**

**(Proceedings delayed)**

**(10.45 am)**

**SIR BRIAN LANGSTAFF:** Good morning, Professor Tedder.

**THE WITNESS:** Good morning, sir.

**SIR BRIAN LANGSTAFF:** Welcome to the Inquiry. May I apologise to those who have been waiting here and online for the slight delay there has been this morning. There are two reasons for it. One is traffic. The second is that some further documents needed to be shown to Professor Tedder because obviously there will be some questions about them and they have recently been disclosed to Core Participants.

We're now in a position to proceed. Let me explain to you the setup. You're talking obviously to the room in front of you and those in front of you are largely Core Participants and participants in the Inquiry. At the back of the room there may be representatives of the press from time to time.

To your left, there are lawyers who represent various of the interests in the Inquiry and, of course, Ms Richards who will be asking you the questions. Behind me over my right shoulder there is Mary who will in a moment or two ask you to take the affirmation. But



let me also tell you that your bigger audience, in terms of numbers, is beyond this room. It will be both in this building but mainly online, either on YouTube or live stream, and that will probably number into three figures. So those are the people to whom you are talking when you give evidence.

Mary.

**PROFESSOR THE HONOURABLE RICHARD SETON TEDDER (affirmed)**

**Questions from MS RICHARDS**

**SIR BRIAN LANGSTAFF:** I should have added that if you wish a break at any time, even at short notice, just please indicate.

**A.** I have a symbol in my hand agreed if I need to have a break, sir. At my age, I have a --

**SIR BRIAN LANGSTAFF:** You don't need to explain.

**A.** An urgency.

**SIR BRIAN LANGSTAFF:** But if you need a break -- you may need a break, and if you do, you will just have to ask for one.

**MS RICHARDS:** Professor Tedder, before we start, I understand there was something you wanted to say.

**A.** Yes. I may appear as a clinical virologist and an academic interested only in viruses. I am interested in viruses but I am even more interested, and almost at a point of terror sometimes, accepting the damage which



these tiny little agents can do in the human population and if I may be seen to be eloquent about viruses, it's not because I don't recognise the damage, and my sympathy goes to all of those who have seen at first hand family damage and loved people damaged by this virus.

**Q.** Thank you.

I'm going to start just with a brief overview of your career. You took a degree in zoology and then medicine; is that right?

**A.** Yes. I started working in zoology before I went up to Cambridge. I was tasked by the late Richard Harrison at London Hospital for dissecting dolphins and porpoises and I decided that, as much as I liked medicine, natural sciences and the advantage of being able to go outside the human species took me into zoology at university rather than medicine, which is what I had been anticipated to do.

**Q.** You then subsequently qualified as a doctor?

**A.** Well, ma'am, what happened I was offered the opportunity for a PhD in zoology and I did the first year straining mouse urine and looking for olfactory signals in mouse urine, coming back in the evening smelling of mouse urine, and decided, sort of, two-thirds of the way through that that it would be more sensible and



extending my involvement with animals to move back into humanity and resume my medical training at the end of my first year of a PhD.

Sadly, it's one of the few things which I've ever started and never finished. Maybe, who knows -- I came back into medicine, trained at the Middlesex Hospital, and became a virologist more by accident than anything.

**Q.** So you're what would be described as a medical or clinical virologist and that to distinguish that from the position of Professor Weiss who was a non-medical virologist?

**A.** Robin was not medically qualified but he was as concerned as me about the clinical impact of viruses on people. But, yes, I'm a medical virologist and a physician and a member of the Royal College of Physicians.

**Q.** You tell us in your statement you worked in 1973 as a house physician at the Middlesex Hospital Medical School Department of Virology and you worked under Dr David Dane, and I'm going to come back to Dr David Dane in a few minutes. You then, I think, did a period as a house surgeon in Kettering, Kettering General Hospital; is that right?

**A.** KGH, yes.

**Q.** Then you returned in 1975 to the Middlesex Hospital

Medical School. You were an Assistant Lecturer, Honorary SHO, and then Honorary Registrar in the School of Pathology at the Middlesex Hospital Medical School, 1975-76; is that right?
**A.** Yes.
**Q.** And then 1977 to '79 you were the Wellcome Research Fellow in the virology section in The Department of Medical Microbiology still at the Middlesex?
**A.** Yes.
**Q.** You then, 1980 to '81, became a lecturer in that department; is that right?
**A.** Yes.
**Q.** And then a senior lecturer and then, over the years, I think the Middlesex Hospital Medical School then became effectively the UCL Medical School?
**A.** Yes. I mean, those were sort of progression of seniority in any sense. It made very little difference to one's day-to-day work. You were still a full-time member of the Department of Virology.
**Q.** And 1982 to 2003, you were head of the Virology Department there?
**A.** Yes, at UCL.
**Q.** You've had a number of other professorships and roles. I'm not going to go through them all but they're listed in your statement. You're a professor of medical



virology and you were also head of the Blood-borne Virus Unit at Public Health England from 2007 to 2018?
**A.** Yes, I was loaned out by UCL initially on a sort of so many hours per week and eventually was asked to take a role in heading up the Blood-borne Virus Unit at CPHL, PHLS Colindale.
**Q.** Then you've been on a range of committees and working groups over the decades. Again, I'm not going to list them all but, for our purposes, that includes: EAGA (the Expert Advisory Group on AIDS); the ACBSB (the Advisory Committee on the Virological Safety of Blood); the Advisory Group on Hepatitis; the Advisory Committee on Dangerous Pathogens are some of the mini-committees that you've sat on over the years?
**A.** Yes, ma'am.
**Q.** Then you provided evidence to the Penrose Inquiry, the Archer Inquiry, and the Lindsay Tribunal in Ireland?
**A.** Yes.
**Q.** Your statement mentions that you have files of a deposition given in American litigation. I'm not going to ask you about the content or detail of that deposition, but is it right to understand you were asked to give evidence for the plaintiff or plaintiffs in a set of American litigation against a pharmaceutical company?



**A.** Yes, I think it was a general request but I was on the plaintiffs' side rather than on the commercial side.
**Q.** And you gave oral testimony as a deposition over, I think, three separate days?
**A.** Two or three days certainly. It's a long time ago, ma'am, but --
**Q.** Do you have any recollection now of the detail of any of those proceedings or the nature of the evidence in broad terms?
**A.** Well, I think in broad terms it was a question how you get to know your donors, how you select your donors, how you process the material that you have harvested from the donors, how you treat that, how you use it, and how you keep a weather eye on adverse events.
**Q.** We'll touch on all those issues then as we look at your statement and your evidence.

It's clear from the documents and your statement that you had a close working relationship with the North London Regional Transfusion Centre. How did that come about?
**A.** Partly because they were close, closer to us -- and I was working at UCL -- but also because I had knowledge of the transfusion service as a whole and I was particularly a good friend of now Professor John Barbara, who was a contemporary of mine, and then



working also in collaboration with Dr Mortimer at the Colindale kept me in the sort of Northwest London precinct, as it were.
**Q.** We'll see from time to time references to meetings and discussions with Dr Barbara (Professor Barbara now), Professor Contreras and others. Did you have any equivalent working relationships or any particular detailed knowledge of what was going on in other regional transfusion centres?
**A.** Well, I think at the various committee meetings and discussion groups there would have been an exchange of ideas/concepts because we tended to work together as a collaborative group rather than the formation of distinct bits within the framework. So there would have been discussion certainly.
**Q.** I want to start now by asking you a little about hepatitis B. If we just put your witness statement up on screen -- Laurence, it's WITN3436003 -- and if we could go to page 8. I just want to ask you a little about what you say in paragraph 20. So you say this:

"David Dane was Head of the Department of Virology, having returned from Ireland, where he was working with Professor Dick on the early polio vaccines and their antibody responses. He pioneered the use of the electron-microscope as a diagnostic tool and

demonstrated its usefulness for diagnosing herpes virus related skin eruptions. His role was research and diagnostics. He would be looking for things like diarrhoeal viruses and keeping a watching eye for electron-microscope diagnostics for blood-borne viruses. It was early horizon scanning, looking out for anything new."

Then you go on to talk specifically about hepatitis B but, before we do that, could you just explain to us this idea of early horizon scanning and what it was that Dr Dane was doing in that regard?

**A.** Well, David was, I think without question, one of the pre-eminent medical virologists, or virologists generally working within the medical field, not only in this country but globally. He was an amazingly perceptive, intelligent virologist, looking for -- and I think I did provide you here with an electron photomicrograph of hepatitis B to demonstrate how you can actually look for a virus.

I think he would have -- sadly, he passed away before we really got into the depth of the HIV epidemic, or pandemic, and that is to me always a great sadness because he would have brought tremendous wisdom to people's thinking. I use the term "horizon scanning" because he had an open mind and if he saw a disease



process happening in a population, he would want to know about it, learn about it, and was there a virus involved with it? Let's not predicate just viruses; was there an infectious agent involved in a disease process? And he would have wanted to determine more about it. He would have attempted to rescue the agent, propagate the agent and indeed, as I said in a rather vernacular sense, he would look for it with the electron microscope and ... yes.

**Q.** In relation to viruses and the spread of viral disease, what in particular could be the role of electron-microscope diagnostics in the 1970s?

**A.** It is interesting, it has come back into its own with this problem of the virus infection in young men. The skin eruption. Easily diagnosed with an electron microscope. It is a technique that not many people have access to, because you have to be trained how to use an electron microscope, and it is not as easy as a conventional optical microscope, where you have something on the slide, you stain it, and you look at it down the microscope. You have to use an electron microscope and you don't know, when you are looking for something under an electron microscope, what you are going to find. Because a virus has a discrete structure. I can tell a poxvirus -- even I could tell



a poxvirus from a herpesvirus from a filovirus. But you've got to demonstrate that in the sample that you've taken from something, whether it's a lesion on somebody or it is a stool sample from a human or an animal, that you can actually see the virus that you are looking for. And I use the word "see" in the sense of visualise it by electron microscopy.

**Q.** You go on to talk in the next paragraph about Dr Dane's work in relation to hepatitis B. You explain he was working on the epidemiology transmission routes of hepatitis B. He developed assays for the detection of hepatitis B surface antigen.

Then if we go to the top of the next page:

"He confirmed both sexual transmission and parenteral transmission."

Do you have any recollection as to when he was in a position to confirm the sexual and parenteral transmission route?

**A.** For hepatitis B?

**Q.** Of hepatitis B, yes.

**A.** Well, I remember we recognised in a particular group of people that there was sexual transmission. This must have been the very early 1980s, because he retired in '82/'83, so it would have been the beginning of the 1980s. And it was just recognising that there was



transmission of hepatitis across various relationships.

It was looking, in the sense, epidemiologically rather than using an electron microscope to look physically, but obviously he demonstrated with the electron microscope that he could actually see the hepatitis B virus.

**Q.** In your statement -- it is at paragraph 27, bottom of page 10, please -- you then set out your understanding, in broad terms, in relation to hepatitis B when you joined the Middlesex Hospital Medical School, and you explain, picking it up in the third line:

"... a viral infection that has a different outcome in different people. Infection may cause an acute hepatitis illness in a person; alternatively, the infection may not cause any acute symptoms yet may sometimes be persistent; some persistently infected persons may get the severe illness of liver failure many years in the future. We thus knew that it caused chronic liver disease, acute illness ... and that was transmitted by ..."

The routes you then describe.

So would it be right to understand that in the '70s, at the time you were working at the Middlesex Hospital Medical School, it was understood that hepatitis B was a serious illness?

**A.** Ma'am, I'm trying to put my mind back to my years as a medical student, because that's when I would have had my first exposure to David Dane, and only because I needed -- he was the only laboratory that was prepared to let me, as a medical student, use a radioactivity gamma counter because of an experiment I'd set up when I was in America and I wanted to continue that.

And I began to work there and realised that the virologists were really very interesting people. So this would have been -- I'm not sure when I actually would have had that complete portfolio of views about hepatitis B. But I do remember the early recognition of transmission of acute hepatitis B through non-injecting -- non-sharp transmission, which implied sexual transmission between people.

**Q.** Do you have any recollection, again asking you to cast your mind back to the 1970s --

**A.** Oh, god.

**Q.** -- of how big a public health problem hepatitis B was thought to be?

**A.** Ma'am, that's 50 years ago.

**Q.** I know.

**A.** Give me the decade again you are asking -- this was --

**Q.** The 1970s. So whilst you are working with Dr Dane essentially.



**A.** Well, I would have been a very young -- I would have been a very junior pair of hands in his laboratory and I would have been interested to learn from him that this the -- this was about the time that the Willowbrook's School experiments were conducted, whereby children were inoculated or infected with a short incubation hepatitis, which turned out to be hepatitis A, and a long incubation hepatitis, which was hepatitis B, and the Willowbrook School showed that the two didn't interact, there was no immunity, one transferred to the other.

So beginning to recognise this was a transmissible infection, yes, I would have acquired that knowledge from David, and I would have seen that. And the recognition that it was parenterally transmitted through inoculation accidents would have exteriorised, gone into the field of, well, if you are using a large quantity of blood, you are certainly going to get an infection.

**Q.** Was it do you think understood by you and your colleagues, at least at Middlesex, in the 1970s that hepatitis B was -- there was a particular issue of prevalence amongst gay men?

**A.** Well, I am sure if David had been there and -- he would have -- yes, we must have recognised it was transmissible like that, but I was not aware of this



being particularly a disease amongst men who had sex with men, at that time.

**Q.** Just a couple of further questions in relation to hepatitis B. You've referred to the assay being developed in relation to hepatitis B surface antigen. Why was hepatitis B surface antigen the main antigen associated with the detection of the virus?

**A.** Well, I don't know, ma'am, if you have that photomicrograph still, if you can put that on the screen --

**Q.** I don't think we do have it, I'm afraid.

**A.** That is a great pity --

**Q.** We might be able to get it later.

**A.** -- because that would show you the density of the hepatitis B 22 nm form, which is a small particle, which is surface antigen by any other -- Australia antigen if you like. That is the marker, the most common marker in the bloodstream of people who are infected with hepatitis B. And that is, in short, hepatitis B surface antigen. It is abbreviated to HBsAg. HBsAg, we virologists refer to it. Other people call it Australia antigen, but that is historical.

That is a marker evident in people's bloodstream the moment they are -- well, within a week or so of infection, within five to ten days, you will have first



the viral DNA coming into the plasma and then the viral antigen. So within ten days of infection you will be Australia antigen positive or hepatitis B antigen positive in the plasma.

**Q.** Then in terms of the core antigen, HBc, and leaving aside any role that might have in surrogate testing for other conditions, that we will come on to at a rather later stage, what part would that core antigen -- what part did it or could it play in the detection of hepatitis B in the '70s or early '80s?

**A.** Can I just extend that into a description of what happens to the markers in acute hepatitis B because this is relevant. If I was to infect myself now, within 5 days to 10 days I would have viral DNA and surface antigen coming into my bloodstream. Within 4 to 6 weeks I would have a liver which is full of hepatitis B virus, which in itself is doing some liver damage, but then my immune system wakes up and says: hey chaps, we've got a virus in these hepatocytes, we're going to take them out. Not realising that if you take out all the virus-infected cells, you're actually putting the person into -- you are putting that liver into incipient hepatic failure. This is when you go yellow.

And if you have fulminant hepatitis B, which means that the immune system is over aggressive, it will

probably kill you. It will destroy your liver. And the only way you can save somebody is by transplanting them, and you really don't do that in the acute phase, you haven't -- in those days you don't have time. So you die of fulminant hepatitis.

What is more likely, in a significant proportion, the virus actually colonises the liver, doesn't stimulate the immune system other than producing antibody to core antigen, which is anti-HBc, so you then become surface antigen positive in the peripheral blood and you have antibody to the core component, anti-core, in the peripheral blood.

Does that answer your question?

**Q.** Yes?

**A.** It gives you a way of defining recency of infection because you can look at the type of antibody that is the anti-core, and in the acute infection you will have a lot of IgM. It's diagnostically useful.

**Q.** Then you have two passages in your statement talking about aspects of HBV that I have been asked to ask you to explain.

If we can go back to the statement, Lawrence, and go to page 50. Bottom of the page.

It is just picking it up in the last line and then over to the next page, you say:



"As an aside, I should note that we tend not to use the term 'carrier' anymore because it implies the benign 'carriage' of the virus which is passively in the host when in fact it has the potential to cause long-term and sometimes severe liver disease in the host."

I have been asked to ask you to explain a little more about that.

**A.** "Carriage" generally implies a benign relationship between the carrier and the carried. It doesn't actually give a measure of the fact that the virus is turning over in somebody. It is a persistent infection. You could carry something and be unaffected by it, have it not replicating and it just waiting to replicate and cause disease.

That is not how hepatitis B works. Hepatitis B, when it becomes established in the liver, it is a persistent infection. And that is why I tend not -- for years, decades, I used the term "carrier" because it meant somebody who is carrying the virus, but it actually demeans the importance of virus persistence rather than virus carriage.

**Q.** Then, paragraph 155, so the next paragraph, you say this:

"Over the years working on this virus ..."

So we are still talking about hepatitis B here:



"... has taught me a lot about the biology of persistent infection, the level of virus replication (inferring the level of infectivity), its variability and the evolution of the virus during its persistence in the host (which is quite a complicated interrelationship)."

Again, I have been asked to ask you to explain how the virus evolves during its persistence in the host and what implications that might have for the health of the patient?

**A.** Okay. It is a difficult concept and it is one which I ran into a difficulty of trying to make it clear to people. If I can take the analogy of the Red Queen in Alice in the Looking-Glass. You remember the Red Queen used to run around in circles going nowhere? And this is a concept which I try to apply to hepatitis B: it is continuing evolving but it doesn't actually lead it anywhere. It is just mutational change. Five years' time you will find other mutational changes. And these mutational changes vary in the type of virus persistence infection with hepatitis B.

I don't want to lecture but shall I put that in context?

**Q.** Yes.

**A.** When you become persistently infected with hepatitis B,



the virus is turning over very quickly in your liver and it is producing a component of it called the e antigen, which becomes a marker of high level virus replication. And the e antigen is almost a surrogate for -- it leads the immune system a bit astray.

What happens is the virus then evolves and no longer expresses e antigen in the way you would expect. There is a mutational change. But this allows the virus to sit in the liver and continue to replicate and it does so by not allowing bits of itself to be thrown out into the plasma, recognised by the immune system and got at. So it goes into a sort of covert stage but still turns over in the liver, still comes out in the liver, and still is infectious in the blood.

During that evolutionary period it is relatively silent until you get into the stage of the immune system suppressing the persistence. And this is -- sorry, this sounds a little bit of a eulogy -- just makes me ...

In the early part of the infection, which may be 20 or 30 years, you produce e antigen as well as surface antigen. The virus is then suppressed because the immune system sees bits of that. And then, further on, the virus will then change its expression of the e antigen to allow it to continue to replicate without being seen by the host. So the virus comes back and you

progress into chronic liver disease.

This evolution, to me, is a very subtle way of the virus being suppressed, escaping, continuing to grow. And you see that there are a range of mutations which are required in the virus in this escape period.

**Q.** Thank you.

I'm just going to turn then to non-A, non-B hepatitis, and sticking with the 1970s, to the extent we are able to divorce that from your later knowledge.

If we put the statement back on screen, please, Lawrence, and go to page 11, paragraph 28.

You say this in paragraph 28:

"Non-A, non-B hepatitis ... had a very similar pattern of transmissibility by blood -- components and products -- although one of the bizarre phenomena which impacted our view of the severe and chronic nature of NANB was that acute infection was often very mild clinically such that persistence was not associated at that time with an understanding of its ability to cause severe end-stage liver disease. Post transfusion transaminitis was for some time (wrongly) considered to be 'trivial'."

Then you refer to intravenous immunoglobulin preparations.

When you talk about this bizarre phenomena impacting



"our view" of the severe and chronic nature of non-A, non-B hepatitis in the '70s, which is what this question is asking you, who is the "our" you are referring to there? Is that the Middlesex Hospital Medical School Virology Department or are you speaking more widely?

**A.** I think it was a wide view of medical virologists, transfusion people, physicians. Looking back on it, the description of mild, transient -- often -- transaminitis, that is inflammation of liver, a little bit of liver enzymes coming into the peripheral blood, little bursts of activity, and then settling down and perhaps going along like this, close to the cut off, below the cut off, above the cut off, you know, a little bit of turnover, and thinking that that is trivial.

Well, it wasn't trivial. And certainly in retrospect now, if you follow people 20 years through this period, you meet the crunch time when the inflammation which has been going on here has damaged the liver such that the liver is no longer able to sustain the individual. So it is not trivial but I think it was -- the fact that, unlike acute hepatitis B, which could be devastating, it could be fatal, the acute hepatitis B can kill, acute non-A, non-B gives you a little bit of transaminitis. And if you compare those two, you can see how you could be --



you can see how it would be easy to say, "Well, it has been such a trivial infection now, it is not going to matter, look at hepatitis B doing this and non-A, non-B or hepatitis C doing this, this doesn't matter, this does matter". Which was understandable in some ways but was actually not the case. Because the further out you go, what has been a little ripple on the surface becomes quite a devastating end-stage liver disease.

**Q.** Why was it, do you think, looking back now, that virologists, those in the medical community, as you describe, took the severity of the acute symptoms, if I can put it that way, as the indication of the long-term -- the potential long-term effect of the disease?

**A.** I'm not sure, ma'am, that we did that. I think what -- a virologist would always be nervous about anything that is a persistent infection. I think my colleagues in clinical medicine and perhaps gastroenterology would have been less concerned about something which comes in and gives you a tiny little tickle of abnormal LFTs, elevation of liver function tests or below -- above or below the cut off, would consider that to be less damaging than something comes in and says, "Hooray, I'm here, bang". But, of course, with hepatitis B, this means that you are going to get rid of the virus. This



coming in with hepatitis B means you are going to get the long-term persistent infection, which is as damaging as the long-term persistent infection of hepatitis C.

**Q.** Then do you have any recollection as to when your understanding of the non-A, non-B hepatitis could indeed be severe and chronic, when that was something you appreciated or began to appreciate?

**A.** I think one was always nervous about it as a clinical virologist, finding somebody with a persistent infection, especially if we haven't seen them before. I think it was when one realised that there were people who were anti-HCV seropositive presenting with end stage liver disease, or severe liver disease, that one was very quickly forced into realising this was not a trivial infection but this was the beginning of something which would then escalate towards severe chronic liver disease.

When that happened for me, I don't know. But it would have been a realisation from the gastroenterologists that this virus infection actually mattered a great deal.

**Q.** Now, you talk in the next paragraph -- so if we can have paragraph 29 visible -- about Dr Dane's major concern about the American commercial sector and producers of blood components and fractions recruiting prisoners and

paid donors. You refer elsewhere in your evidence to that being I think his "red flag", the use of prison blood and paid donations his red flag, and:

"... his overriding need to 'know your donors' ..."

Can you just -- and this was obviously during the 1970s because that was when you were working with Dr Dane before his retirement in the early '80s. Can you just tell us a little more about that and about what it was that Dr Dane was particularly troubled about in that regard.

**A.** I think it was in the scenario of not -- now, I'm not criticising anybody and I don't want anybody to perceive that I'm criticising the transfusion service or people who were dealing with this because, at the end of the day, you needed to have enough plasma to come through to sustain the clinical requirements.

And the American approach was that you could harvest -- I use that term advisedly -- you could harvest blood more easily from your prison population, which would tend to be male rather than female, and you could give an incentive to harvest the blood but you knew very little about their -- what drove those people, what took them into prison, why they were in prison, let alone what they did in prison. And David's view, in as much as I recall it, and I see it written, and I know he



was always concerned that you must know your donor, you can't know anything about the -- you can't know anything about the excipients (and that's things which shouldn't be in the blood) are in the blood unless you know something about the donor: who they are, what they do, where they do it, how often they do it, who they do whatever it is with whoever they're doing it, in the very broad vernacular sense.

But unless you know your donor you won't know what transmission of agents they are at risk from, and this may be something you don't know when they were on holiday in Guatemala or when they were on holiday in West Africa. Unless you know your donor, how are you going to assess malarial risk, how can you assess the microbiological risk, and indeed sexual transmission or injecting drug transmission risk. If you don't know your donor, you're relying entirely on testing.

**Q.** And do you have any sense of how widely Dr Dane's concern in the 1970s about this (the use of prison blood, the red flag, the need to know your donor), how widely that concern was shared?

**A.** He would have certainly been propagating that view in the late 1970s/early 1980s without a shadow of a doubt because it was always "know your donor" and know your donor so that you know what is the blood component or



blood product you're going to give somebody, and I think in fact many of my colleagues in the transfusion service in the late '70s and early 1980s would have been exposed to this question.

**Q.** Now, you've referred in your evidence, in your written statement, to in particular the concern about the American commercial sector recruiting prisoners as donors. Do you recall any discussions, whether with Dr Dane or others again in the '70s or early '80s, about the extent to which blood was still being collected from prisoners in the United Kingdom?

**A.** I don't recall, but I would be very surprised if we had not -- given everything that I've written, that we had not discussed it. I'm sure we had discussed it but I can't give you a time and date, I'm afraid.

**Q.** Then I just want to ask you about something that is in your evidence to the Archer Inquiry. So if we could have on screen please, Lawrence, ARCH0000011 and if we go to ... let's find the beginning of your evidence. I can't find the first page now but, in any event, I assure you this is your evidence.

If we go to page 171, there is just one passage here. There's a question from a member of the Archer panel, Ms Willets at lines 3 to 4, a reference to people suffering from a mild form of haemophilia and it was



clearly preferential that they were receiving cryoprecipitate, and then I just read out your answer and just you about that. So you say this:

"Can I turn that round: it was obligatory that they did not receive concentrate. It was the other way round; people had small requirements for Factor 8 replacement. Certainly in our hospital, under the late Jimmy Stewart, who was the consultant haematologist, he maintained, with the agreement and support of David Dane, on the one [hand] and John Crasse ..."

Is that a reference to Dr Craske?

**A.** That -- I'm sorry, ma'am -- is a misspelling. I hadn't seen that.

**Q.** "... [Dr Craske], on the other, both of whom you will have heard of, that the policy should be that unless you had to give a concentrate -- this was in the late 70s and early '80s -- don't give a concentrate unless you absolutely had to, maintain people on cryoprecipitate."

So is it right to understand that was your understanding of the policy for the treatment of haemophiliacs at the Middlesex Hospital in the late '70s/early '80s?

**A.** It was how you supported the haemophiliac population and I think the change came when you were giving it on routine demand, routine exposure, rather than waiting

for a crisis and this evolved into problems that we had with a busy haematology department where people would come in in what I call a "bleeding crisis" and, in that situation, we always tried to treat with the cryoprecipitates rather than using a concentrate. But obviously if you were faced with a critical situation and you did not get the blood clotting under control, the patient would die, then you have to use concentrate.

But I think Jimmy Stewart and David Dane between them tried to make sure that if there was any opportunity of controlling the illness of a patient, it was done on cryoprecipitate, rather than concentrate. But, you know, one had to use concentrate to save a life if it was necessary.

**Q.** And would it be right to understand that what underpinned that approach was the risk of transmission of hepatitis at that point in time? Or that would have been a main, a central part of it?

**A.** That would have been the major concern, until such time as the concentrates became heat-inactivated and there was microbiological control on the concentrates for heat inactivation and then that altered the balance of risk.

**Q.** We can take that down, thank you.

Did you have any particular knowledge in the '70s and '80s of the notification mechanisms, such as the



yellow card system, for incidences of viral disease such as hepatitis?

**A.** Well, I was aware of the yellow card system. I was also aware of the desire that there should be feedback to the transfusion service of every adverse outcome because, unless you have that, how do you know to analyse risk and benefit and have a protocol in place to introduce a change to reduce risk, to maintain benefit? It was really important that my colleagues in the transfusion service had a way of getting that information.

**Q.** And was there any sense at the time of the extent to which the yellow card system, or indeed any other systems, were effective?

**A.** I would not have been aware of that, ma'am.

**Q.** Can I then just turn to ask you next a little about HTLV-I and HTLV-II --

**A.** Yes, ma'am.

**Q.** -- before we come on to HTLV-III, as it was initially named, and HIV.

In the early 1980s, you were doing work in relation to HTLV-I and II, and I'll ask you about the development of tests a little later. But what's your recollection of what was understood about those two viruses?

**A.** Well, ma'am, it started off with HTLV-I, which we knew then was transmitted from mother to infant, was



associated with only one disease which we knew of at the time, which was an adult T cell lymphoma leukaemia later in life which was very difficult to treat. And therefore, as it was a potentially oncogenic human retrovirus, the principle was to stop it being transmitted from mother to infant and that meant, even though we don't do it nowadays in the United Kingdom, you should have screening in the antenatal clinic to identify the infected mum and give her support, counselling, social support, family support, and access to breast milk substitute. It's the same as it should be nowadays.

**Q.** So that was HTLV-I --

**A.** Yes.

**Q.** -- and an understanding of the link with a form of leukaemia.

Then how did an understanding of HTLV-II come about? Again, I'm looking at the early 1980s.

**A.** Okay. It's interesting. Am I allowed to use my hands to demonstrate?

**Q.** Yes, of course.

**A.** If you have a population of reactivity in an immunoassay, which if you look at it is like that *(indicated)*, and then as you look at it from there, your positives are out here *(indicated)* -- so you've gone



from a high signal to a low signal because you're using a competitive test -- you will have your negative population and your positives there. And if you are a curious virologist, you will see things in the middle, neither positive nor negative, but they're always there and you take them and you repeat them and they're always there. So you say, well, okay, let's get another bleed and you repeat them and they're still there. Then you've got the intellectual exercise of there are your negatives, there are your positives, here are your not negatives, but they're not positive either.

But the important way is to think of those as they're not negative. So what are they? And that turned out to be the sister virus of HTLV-I, it turned out to be HTLV-II, and we still don't really know what the long-term sequel to that is -- or I don't. I know HTLV-I has leukaemia, it also has a neurological disease which gives you a tropical spastic paraparesis (which is where your spinal cord gets damaged and your limbs don't work), and other sort of inflammatory association diseases with HTLV-I.

HTLV-II is a similar retrovirus and, because I found we had the negative population, the positive population and this population, I wanted to have a test for this. So I replicated what we did for the HTLV-I test with

HTLV-II sera and then I had a test which gave you positives, negatives, and things in the middle. There was the HTLV-I, here was the HTLV-II, and there was the negative population and it taught me a great deal about how to build immunoassays, and these were competitive assays where you looked for the test serum blocking a labelled antibody, which would bind to the solid phase because there was an antigen there, and your patient's sera comes in and if it goes in and stops this coming on, you get a loss of colour. And that's a competitive signal and that tells you your patient has an antibody which blocks that.

**Q.** We will come back to the assays that you then worked on.

I want to turn next to ask you about your awareness of AIDS and how you came to have some knowledge in relation to that. Again, if we start by picking it up from your witness statement, if we can have that back on screen, Lawrence, and go to paragraph -- page 12.

You say this in paragraph 31, picking it up at the end of the first line:

"... it is hard for me to define the individual points at which I became aware of AIDS, its association with blood and/or blood products, and its nature as the outcome of an infection which was most likely to be a virus."



Then you refer to the HTLV virus as being the focus of much of your attention in the early 1980s.

Then you have set out in the following paragraphs of your statement references to documents that the Inquiry is familiar with, the MMWR report, the Morbidity and Mortality Weekly Reports. If we just go to the bottom of the page, you say this:

"Having reviewed the MMWR reports, I have a distinct recollection of noting the association with the bathhouse arena, which had a similarity to transmission of hepatitis B. While I cannot be sure, I think this means my recollection of first noting AIDS in a significant way was the MMWR report for 18 June 1982."

We will look at that in a moment. You refer to it and then you say:

"It specifically relates to the homosexual male demographic on the West Coast and has references to bathhouses. I should stress that, while the date of the report is 18 June 1982, I cannot actually be sure at what point I became aware of what was reported."

Just in terms of receiving the MMWRs, in 1982 how would you have actually received copies of that report?

**A.** Well, at that time, I would have either been in or -- I had a very close relationship still with colleagues at CPHL, the Central Laboratory for Public Health, Central



Public Health Laboratory at Colindale, and I would have been involved in discussing things like such as this. I seem to remember that the striking thing in one of these MMWR reports was the fact that they had also seen this in injecting recreational use of drugs which then, by the time you've got a sexual transmission and an injecting recreational drug transmission, if that's not hepatitis B, it's got to be something very similar to hepatitis B. It's got to be a transmissible, parenterally transmissible, virus infection.

**Q.** Just for the sake of completeness, I'll put this MMWR up on screen because it's not one we've looked at as frequently in Inquiry hearings as some of the ones from July onwards. It's RLIT0001690:

"A cluster of Kaposi's Sarcoma and Pneumocystis carinii Pneumonia among Homosexual Male Residents of Los Angeles and range Counties, California ..."

Then there's a reference to reported cases in the first paragraph, data on sexual partners, and then the third paragraph talks about sexual contact in particular areas, in Los Angeles and Orange County, the development of symptoms. Then the fifth paragraph talks about the patients from Los Angeles and Orange Counties being directly linked to other patients and being part of an interconnected series of cases, and then there is



a reference to locations.

Then we have the editorial note which refers to sexual contact and the possibility of -- sorry, and the inter-related sexual contacts between the various cases.

Is it right to understand, doing the best you can, you think this is an MMWR which -- I think the way you put it in your statement -- might have been your first noting of the disease in a significant way?

**A.** Yes, ma'am. I think there is also a contemporary for this where they actually show cases arising in those who are using injectable drugs and that, I think -- this was one which makes you think of a transmissible agent but then the moment you've got injecting drug use involved as well, you then have to say, well, it's parenteral as well as sexual.

**Q.** Certainly one of the documents that you've referred to in your statement which refers to drug use is BAYP0000028_011. It's the second page. This is, if we can pick it up at the heading halfway down the page:

"(AIDS) in Prison inmates -- New York, New Jersey."

We can see this is January 1983. Then there's a reference, I think about five paragraphs down, to ten patients reporting they were heterosexual before imprisonment and then the nine patients with PCP were regular users of intravenous IV drugs.

Then if we go to the next page and just look at the editorial note, which is not quite halfway down, under the italicised bit:

"Editorial note: Since male homosexuals and IV drug abusers are known to be at increased risk for AIDS, the occurrence of AIDS among imprisoned members of these groups might have been anticipated. Increasingly, epidemiologic observations suggest that AIDS is caused by an infectious agent transmitted sexually or through exposure to blood or blood products. Because of the difficulties inherent in interviewing prisoners, data elicited in such interviews must be viewed cautiously. Given this caution, the histories obtained from the inmates indicate that all or most of their drug use, and, by inference, their exposure to a blood-borne agent, occurred before confinement."

That's one of the other pieces of information you refer to in your statement as giving rise to or helping you form your views about this.

**SIR BRIAN LANGSTAFF:** Just before we take this off the screen, if I may, there's a reference to the assertion in the first sentence, first line:

"Since male homosexuals and IV drug abusers are known to be at increased risk for AIDS ..."

The reference is to the CDC, so that would be the



MMWR, update on acquired immuno deficiency syndrome and that's a reference in 1982. It was published in 1982. We know from this, although we haven't put up the particular publication, that IV drug users were known to have AIDS. Can we go back to the RLIT0001690 which we saw a moment or two ago, which was the June 1982 MMWR because I noticed something when it was on screen. RLIT0001690.

If we go down to the bottom of the page and the editorial note, the editor there is looking at possible readings of what this might show the world about the origins of AIDS or what caused AIDS and it talks in the last paragraph:

"Exposure to some substance (rather than an infectious agent) may eventually lead to immunodeficiency among a subset of the homosexual male population that shares a particular style of life."

It refers then to using amyl nitrite as a stimulant.

So far as you are aware, that used as a stimulant for sex was unlikely to be a stimulant involved in IV drug use presumably?

**A.** That behaviour I can't comment on, sir, but, I mean, I think what this led me to perceive was that there was a commonality of men having sex with men and people who were sharing drug usage, injecting drug usage, which put



it into the parenteral agent concept which of course, for my mind, having been brought up with hepatitis B, was yet another footstep: it has to be a transmissible agent.

**SIR BRIAN LANGSTAFF:** So it was the same sort of pattern of observation of the disease as you would see with hepatitis B?

**A.** Yes.

**SIR BRIAN LANGSTAFF:** And that would lead you, would it, to think that you would have transference by needle -- that's obviously the IV drug user -- and therefore from transfusions and those who suffer from haemophilia?

**A.** I think, sir, it would have led me to look at the causal agent in exactly the same frame, conceptual frame, of looking at hepatitis B and that would lead, as you have suggested, sir, to the progression of persistence disease, transmission, et cetera, but, in this case, an agent that we don't know what it is but we know its behaviour and we have a feel for how it transmits amongst people based on everything that we know about hepatitis B and its transmissions through non-parenteral and parenteral routes.

**SIR BRIAN LANGSTAFF:** So your conclusion, from what you're saying and given your background, is that this was something which may be a virus, may not be a, virus but



it was an infectious agent which could be transmitted by blood.

**A.** Yes. Parenterally transmissible; so, by definition, yes.

**MS RICHARDS:** The way you put it in your statement, I don't think we need to go back to it, but you describe hepatitis B as the best analogue illness for what we saw in the 18 June MMWR report for AIDS. You say it's that linked to the nature of hepatitis B that made you first think that AIDS could be a virus and associated with exposure to blood. We can take that down. Thank you.

It's apparent from your statement that you recall that this in the course of 1982 was something that would be the subject or was being the subject of active discussions with colleagues such as Dr Barbara, Dr Mortimer; is that right?

**A.** Yes, it would -- I mean, anybody who had exposure to hepatitis B and working with it in populations and epidemiology would have said this is something -- this has to be something very similar.

**Q.** You attended a meeting in Washington. I think you can't date it precisely but -- the evidence you have given both to Penrose and in your statement -- is probably late 1982, early 1983. I think you describe it as the first retrovirus meeting.

First of all, can you just help, when you call something a "retrovirus", what does that mean?

**A.** It's a term -- when a virus grows and replicates, that means when it grows in the cell, how does it grow? Most viruses you put in, they unwrap, their genome becomes available and it replicates and makes more, throws more of the virus out, and goes on. That is the conventional virus.

A retrovirus is handicapped in a way because its genome -- sorry, this is going to be a wee bit of an explanation, sorry. I don't want to confuse.

There are two types of nucleic acid, there is DNA, which is normally what is in our nuclei and is what is passed from population to population, and there is RNA, which is taken -- the message on the DNA is transcribed -- is changed into a message in the RNA, and that is a signal to the cell: make this.

So a conventional virus will either be an RNA, "Make this, make more of me", or if it is a DNA virus, in, comes out, "Make more of me", and it makes more.

Retroviruses are a little more complex than that, because the nucleic acid is RNA but it comes into the cell and rather than say, "Make me more", it says, "I'm going to take my signal and I'm going to bypass you, I'm going to make it into a DNA signal". And it goes into



the cell. And then the DNA signal is then used to make more RNA to make more virus.

So it has to go this reverse step, from RNA putting it into the normal mechanism, which is DNA, and that is called reverse transcription. Because normally, if you read a book, it is: DNA makes RNA, makes protein. What we are saying here is: this is a RNA virus, which comes in, makes RNA, makes DNA, makes RNA, makes protein. The reverse transcription step is characteristic of retrovirus and is obviously susceptible to certain drugs.

**Q.** What can you recall about the meeting in Washington?

**A.** It was interesting. It was a lively discussion. And I probably raised a big black by being fairly rude to one of the eminent speakers.

**Q.** Who was Dr Gallo?

**A.** Bob Gallo, yes.

**Q.** What was it that gave rise to what you have termed a brush with Dr Gallo?

**A.** Well, he became a drinking companion in later life. I hadn't seen him for decades and I would always -- I'll always fondly recall this.

He had conceptualised -- he had interpreted the data to say that we have got to have a human retrovirus and why not let's have HTLV-I or HTLV-II. Because we know



about these viruses. Why not have these as a cause of a transmissible retrovirus infection in men who have sex with men and people who share drug injection? This would do. And it would be something like HTLV-I or II, one of those two viruses would be responsible.

Working with Robin Weiss at the same time, I had developed serological tests for HTLV-I and HTLV-II, the antibody to the viruses. I can explain how I did that, but it was very simple, and it's what taught me how to do it for HTLV-III when that came along.

We had a serological test for HTLV-I, we had a serological test for HTLV-II. Remember the two populations I talked about, where we had a test that put them both into the positive area?

And we tested young men and other people who had AIDS and we didn't find a significant level of the virus infection in those who were ill and those that weren't ill. And when he said, "Well, HTLV-I or II is the cause of AIDS because, look, it is a retrovirus and people are dying from a retrovirus infection", I put my hand up in the air and said, "Well, we've looked in" -- I can't remember -- "20 or 30 cases of AIDS, and we only found 1% or 2% of these have HTLV-I or II. How are you going to say that something which is down at the fraction of 1% or a couple of per cent of the population



of everybody who gets AIDS -- that's not causal. You can't say that something which affects 1 in 50 -- what about the other 49 people who have this dreadful infection? So you need to think again."

**Q.** But it was Gallo's thinking, was it -- that is the reason why, at that time and for a further couple of years, it was referred to as HTLV-III?

**A.** Ah, well, Bob liked to have viruses in his cluster. So he had HTLV-I and HTLV-II, so having HIV and calling it HTLV-III, it became within his portfolio, bless him. There was nothing -- I mean, there are other HTLVs out there nowadays, in countries in Africa. So the number is going to increase and it is better to have it.

**Q.** Sir, I note the time. We started late but we have now been going for an hour and a quarter, which would be when we would normally take a break. I'm in your hands as to when this we break.

**SIR BRIAN LANGSTAFF:** Yes, I think for two reasons we will have -- well, three reasons we ought to have a break. The first is we have had an hour and a quarter. The second is, I am sure Professor Tedder could probably do with a break. And thirdly, so could the stenographer.

**MS RICHARDS:** Exactly.

**SIR BRIAN LANGSTAFF:** So we will take a break and we will come back.

Now if we come back at 12.30 pm we will go on then until 1.30 pm and then we will have a late lunch at 1.30 pm, if that's all right? I see that those around me nodding and saying that will allow the appropriate arrangements to be made. So 12.30 pm.

**MS RICHARDS:** And the normal explanation for Professor Tedder.

**SIR BRIAN LANGSTAFF:** Yes, I will give that just now.

Professor, you are under oath. The rule is that anyone who is under oath may not discuss any evidence they have given or any evidence for that matter they think they might yet be asked to give, with anyone, whoever that anyone is, but they can talk about anything else they like.

**A.** I understand.

**SIR BRIAN LANGSTAFF:** 12.30 pm.

**MS RICHARDS:** Thank you, sir.

**(12.00 pm)**

**(A short break)**

**(12.30 pm)**

**MS RICHARDS:** Professor Tedder, I want to turn to interactions you had or an interaction you had with the Department of Health in 1983. I'm going to pick it up, first of all, with your evidence to the Penrose Inquiry.

So if we could have, Lawrence, PRSE0006049, and if



towers and we were told this was really not any of our business and it was not going to be a problem and go away and stop rocking the boat."

Over the page. You say:

"Both Philip and I -- well, I can't speak for Philip. You would need to ask him. But I was somewhat taken aback and pretty irritated."

That was your recollection to the Penrose Inquiry. As I understand your witness statement to this Inquiry, Professor Tedder, you don't have now particularly distinct recollections of that meeting?

**A.** I think that's -- I have to say, I think that's right. You know, a lot of water under the bridge, I'm afraid. I do remember feeling disappointed that there was not a willingness to go forward and grab opportunities. But, you know, that's a sort of colouring of what I think went on. But if you are asking me who said what, I can't comment.

**Q.** After that meeting you wrote to Dr Walford. I will come to that letter in a couple of minutes, but I want to ask you first about some observations you make in your statement.

So if we could have WITN3436003 back on screen. If we could go, please, to page 18.

You say this in paragraph 54:



we go to page 96, please.

If we go to the bottom of the page. You were asked at line 19 about whether you had met Dr Diana Walford in connection with the whole AIDS problem. And you explained you had. You referred to the meeting in Washington which I was asking you about before the break. Then you say:

"After that and after the meeting, the NIH meeting, discussing this, it must have been early 1983, Philip and I ..."

That is a reference to Dr Philip Mortimer?

**A.** Correct.

**Q.** "... went to DHSS to ask what the plans were for -- what was ready -- what was going to be the plans for readiness to deal with what, I think I alluded to earlier this morning, was a disease or infection which sounded awfully like Hepatitis B in terms of affecting the same group, having the same sort of transmissions."

Then you refer to having heard in early '83 or end of '82 about haemophiliacs also being involved.

Then if we go down to line 19, you say this:

"So we felt empowered to go and ask the DHSS what could we do to explore this, and it was as cold a meeting inside the room as it was outside. It was a sort of cold spring morning up in one of the DHSS



"I was not aware of plans to deal with a new and emerging infection and cannot comment on what if any processes were then in place. In the early 1980s, hepatitis B was a disease which caused a clearly apparent acute illness presenting as jaundice and hepatitis in gay men. Had there been appropriate public health surveillance for this, it would have opened up the immediate possibility of using the same approach in the same population looking for the disease of 'gay-related immuno deficiency syndrome', 'GRIDS'. This would have given the absolute substrate for investigating the emergence of HIV disease in the same population."

Two questions arising out of that paragraph, Professor Tedder. Firstly, what kind of public health surveillance did you have in mind that could have been appropriate for hepatitis B?

**A.** I think notification to an organisation who would be able to look for further transmission of infection from the infected individual; provide protection and -- to those who might have been exposed -- if the time was right, there are options for passive antibody given to people who have had an exposure to hepatitis B. But also the opportunity to immunise the at risk population. I use the "at risk" very specifically in this case, the

sexual partners of. And hopefully to encourage a degree of behavioural change to prevent onwards transmission of hepatitis B.

All of those could be offered partly from a laboratory who's had the central role in knowing where these acute infections were, relating it to the GUM clinics or the health clinics, and the health clinics and the GUM clinics themselves would have had a role in passing that information back to their patients and people seeing them and trying to suggest that maybe they modify their behaviour.

**Q.** And there was no such system in place that you were aware of at the time?

**A.** Well, I think there were but I wasn't aware of it being truly effective. But that's just -- that's me as a clinical virologist.

**Q.** Then can you assist in understanding the reference in that last sentence to the "absolute substrate". What did you mean by that?

**A.** I think what I'm suggesting, if you put that in place, in having a mechanism for surveillance, interaction with the risk populations, this would have given one an opportunity to say: Hey folks -- because we don't want to put a gender on it -- we've have talked to you about hepatitis B, just be careful because there's



something out there which we think has a similar role of transmission, and if you can protect yourself by your behaviour from getting hepatitis B, you may also benefit from getting infected by whatever the cause of this new and emerging disease is.

**Q.** If we can look at the next three paragraphs please. You say this:

"55. Even today with the advent of Covid, when I was able to produce within the first few weeks of the emerging coronavirus epidemic, serological tests which could identify both acute and past infections with Sars CoV 2, and offered these to the Government, they were entirely disregarded.

"56. ... My reflection suggests that the pressure to develop antigen-based and PCR-based diagnostics for detecting the infection in the acute phase distracted people from the value of having serological tests which could be used on a population basis to map the transmission of this virus within the population as a whole.

"57. Interestingly we seem to be running through a similar scenario 36 years later. It was very similar to the response I received when I told DoH in 2020 that what we needed for Covid was an *antibody* test. I think people focus on finding a test to the virus and forget



the value of having a good serological test for antibody against an emerging infection which facilitates determining when, where and how a virus may be passing in the host population."

If we go back to the previous page. I'm not going to ask you to talk on any level of detail about your interactions with the Government into Covid -- we are not the Covid Inquiry and there is a Covid Inquiry which has recently begun -- but it is more the reflections that you are able to draw based upon interactions back in the early '80s with the Government in relation to AIDS and then interactions more recently, as you describe here, with the Government in relation to Covid.

Do you have any thoughts or reflections on why there seems to be, as you describe it, a short sightedness about the value of certain kinds of tests and certain kinds of response?

**A.** I think there is two components in an answer to that. If I can get the trivial component away first, which is: there is a great reliance by DHSC and people in that area of authority to distance themselves from an academic who comes and says, "I have got a damn good test and this will work", because it is not commercial.

And to get a test of the sort of ability that we have, you would have to get it commercialised and -- and



length of time, you have to find a partner and so on. But it means that your technology is not absorbed in the response.

Now, partly an antibody test doesn't help you really to make an acute diagnosis unless you have an antibody test that can measure the process of the antibody and the acute infection, which is IgM antibody. And that is what we have for Covid. We have an antibody test which can identify past and acute infections and be delivered by a dried blood spot, all of which enable you to say: Mr Smith, over in Little Whopping on the Lyme or somewhere, I'm going to send you a card, drop two or three drops of blood on there and we will tell you whether you have had Covid", or whatever infection. That, to me, as a virologist and somebody who is an old-fashioned animal who likes to measure antibody, is an alternative to the modern approach, where you have to take a throat swap or nose swab, you have to send it to a laboratory for a PCR.

I am not decrying PCRs because I love PCRs, and we have developed them over the years, but people will forget an antibody will map your transmission of an agent in the community rather than having to get acute infections from everybody. For every acute one infection you have, you may have 15 or 20 people who

have been infected. If you are going to do a population of 500 people, it is much easier to do 500 dry blood spots than 500 PCRs where you each have to go up and take the appropriate sample at the appropriate time.

People forget serology. It is a good way of determining the evolution of an epidemic into a population, the distillation of the virus going through the population.

**Q.** If we turn back into 1983. Following the meeting that you and Dr Mortimer had with the Department, if we go to the letter that you then wrote to Dr Walford, DHSC0003824_164, it's a letter of 20 May 1983 to Dr Walford and you refer in the first sentence to the meeting:

"Thank you for seeing Dr Mortimer and myself last week. I gather Dr Catterall has already written to Dr Graveney, I expect you like I have seen a copy of his letter. It is, I believe a reasonable statement of intent. However as a virologist I should like to make a number of comments on the whole problem of AIDS not merely related to a GUM framework.

"This condition is likely to be caused by an infectious agent or agents. Its epidemiology bears a striking similarity to hepatitis B. Since hepatitis B became a growth industry we have gained a great deal of



knowledge about the infection and about the illness by study of a small, epidemiologically easily identified patient group, the male homosexual. In the UK where hepatitis B is not a major health problem, it has still proved possible to examine the problem of this disease by examining this group of patients.

"AIDS is essentially limited at present to the same, easily identified group. So although AIDS has many wider implications covering for example the fields of blood transfusion and risks to medical staff, it is only by detailed study of the homosexual patient that we will be likely to make [a] significant contribution to an understanding of its aetiology.

"The work on AIDS covers three main aspects; to be successful I am certain that a multidisciplinary approach is vital. Also since the evidence is that the disease is becoming established in the UK I think that it is necessary to set up this project now."

Now, just pausing there, can you recall -- difficult question, I suspect, so many years after the event -- what evidence it was that you had in mind that the disease was becoming established in the UK? What kind of information you might have had which led you to make that observation?

**A.** It was 39 years ago. It is a bit difficult to recall.



Ma'am, I should think it is again coming back to the similarity of disease patterns in the population of hepatitis B and what you would -- how you would control hepatitis B is identifying where it is, transmission events, counselling people who have been infected, counselling people who are infected. Hepatitis B, of course, we had a vaccine. For this, even now we don't have a vaccine; so it comes back to counselling people and giving people good advice. That's really ultimately, when you have something new in the community, that's what you have to do.

**Q.** Would it be right to understand, because you'd gone to this meeting with Dr Mortimer, and we haven't said who Dr Mortimer was, he was based at PHLS so he would have had access to whatever data PHLS had by this stage about the extent of infection with AIDS?

**A.** He would have but don't forget PHLS, like the Department of Health, is split into bits and there may not necessarily be routine transmission of information from one group to another. I mean Philip -- who's still a good friend of mine -- is a medical virologist and we, as virologists, may or may not be privy to what epidemiologists have or want or what clinicians dealing with the patient groups have or want.

If one could break it down and get people to talk to



each other without feeling of, you know, there's a sectarian limit to where I am and somebody else over there will say, "Well, it's all our problem; let's try and solve it for the betterment of people", you need to get talking and you need to break down barriers -- and that's barriers both up to government, down from government, and at the level of people who are dealing with individuals and populations.

**Q.** Then turning to the letter, you outlined three areas of work that you thought was important to undertake. I'm not going to read those out but we can see the heading "Patients, immunology, virology."

If we go over the page and just pick it up in the third line, you say this:

"If there is a single novel aetiological agent causing AIDS and if this agent is present as a viraemia (likely since Factor VIII is implicated) it may well be amenable to the sort of approach [which] identified hepatitis B and the human parvovirus."

Pausing there, can you help us understand the statement that it was likely to be viraemia because Factor VIII was implicated?

**A.** Well, it feels like -- if you gave the pattern of this -- not the clinical features, but if you gave the pattern of this disease and transmission events and who

it affected and who it didn't, you'd say, well, that's a transmissible agent, sexually transmitted, probably causing persistent infection and persistent infection is leading to disease, and there may be an illness of acute infection which we don't know about. That is how I think a clinical virologist would have thought about that and then, having thought about that, would say, "Well, we need to have a way of testing. We need to develop diagnostic assays" and that includes virus assays and includes looking for virus -- literally looking for virus -- and also antibody tests.

**Q.** Then we can see in the next paragraph -- I don't need to read it out -- but you set out the kind of support in terms of funding that you were looking to the Department of Health to provide (inviting the Department of Health to consider providing) to enable the work that you describe there to be undertaken.

As I understand it from your statement, although the documents suggest that some funding was received from the Medical -- or may have been received from the Medical Research Council, there was no funding that was provided in response to this by the Department of Health itself?

**A.** That's a difficult question for me to reflect on, as I say, nearly 40 years ago.



I'm not sure. I think we did. We were put forwards with support to the MRC to seek funding with Professor Adler, who was the GUM physician or GUM physician lead in our local clinic.

If you're asking me exactly what returns we got, I'm afraid I really can't recall. All I know is we were busy the whole time providing testing for anybody who wanted it. That's all we could do at that time.

**Q.** The letter you wrote to Dr Walford was, I think, the same date as the publication by Montagnier in Paris of the LAV 1 data. In relation to that, did you have any knowledge in advance that that was going to be published or any knowledge in advance of what had been found?

**A.** No. No, I didn't.

**Q.** Sorry, we can take that down.

Do you have any recollection of what your response was and reaction or the reaction of colleagues to the Paris publication?

**A.** No because I was a serological -- my activity was a serological component of a response to this new agent. We did not have the facility in the first instance for propagating the virus in tissue culture. We developed that but we certainly never had the expertise to make primary isolates and that was not our remit. That fell to Robin Weiss and his colleagues, and indeed gifted



LAV-I from Montagnier.

**Q.** We will come on to that. Just then sticking with the interactions that you had had with the Department of Health, if we go back to your statement, WITN3436003, page 22, you say this in paragraph 69 and 70:

"While the wording that was used in the meeting with Dr Walford may not have specifically been 'stop rocking the boat', the sense that I got was that the DHSS did not regard testing as a problem. She indicated that they would not provide the necessary funding.

"The issue was that, by early 1983, we knew that we needed to do something quickly. I think this is why I found the response from the DHSS particularly difficult. I felt that we were ready to go and do something to help, but the DHSS was saying that it was not really their business to fund it. It is worth remembering that by this stage, because of my work on HTLV-I and HTLV-II, the modelling indicated that we could easily make an assay for HTLV-III B."

Is it right to understand from your evidence, both to Penrose and here, and the fact of the letter that you wrote, that your sense was that the Department was not responding with a sufficient sense of urgency, a sufficient sense of the importance of addressing it?

**A.** It's difficult to reflect on that, what I actually felt



at the time. I think it's when you have a sense of I've got something which could really be helpful and I'm able to produce this, "Look, I've done this" and you get a feeling of, "Well, that's all right but it's ..." you know. So not exactly "so what" but didn't get a feeling of, "Gosh, that really could be important. Let's take that forward".

Now, all I can say Philip and I would have felt disappointed.

**Q.** Then, if we go to paragraph 81 of your statement, page 27. You say this:

"In respect of funding, I do have a recollection of a meeting with someone from the DHSS sometime later -- I think around 1984 when we had serological tests. It might have been at an MRC meeting although I am unsure. I remember there being someone from the Department of Health who said words to the effect of 'haven't we done well'. I recall seeing Professor Weiss bristle at the suggestion. He responded that the Department had done absolutely nothing. I remember the man backing away from Professor Weiss. It was completely incorrect for him to say 'we' as we had essentially been forced to fund the entire programme by other means -- from internal funding, funds to me in my dual post at the time, through the [Health Authority], or possibly

through the MRC."

Do you have any further recollection of that later meeting or does that paragraph essentially describe what you can recall?

**A.** I do think you should ask Robin Weiss what his recollection of that is. I have never seen him be so irritated -- in a perfectly gentlemanly manner but just saying, "This is not on, what you're saying. You haven't done anything. How dare you say haven't 'we' done well. You should be saying haven't 'you' done well".

**Q.** Can I just ask you, on the issue of public health funding more generally, to look at something you said to the Archer Inquiry. So ARCH0000011, please, page 160. Now, just to put it in context, this part of your evidence takes place as part of a discussion about self-sufficiency and the upgrading of BPL and the Chair of the Inquiry says this at lines 4 to 9:

"I think we are very conscious of the dangerous of hindsight ,but what has been suggested is that it was a little late, by 1974, to think about upgrading the facilities for processing this. Elstree, for example, was a long way behind what its capacity could have been if there had been a timely investment.

**"Answer:** I think that's probably fair. I think



the -- if one blames anybody, it is the financial structures in this country which sometimes don't put money into health service emergencies, and certainly one didn't recognise this as an emergency until much, much later, or it became an emergency because of a failure to invest."

Then the Chair's comment:

"Of course the time to provide for something is before it becomes an emergency."

And you say:

"Indeed, yes."

Now, I'm not asking you about the issue if funding for the redevelopment of BPL. It's the general observation you make there about the financial structures in this country which sometimes don't put money into health service emergencies or perhaps don't put them in sufficiently far in advance. Again, are there any observations drawing on the many decades in which you've been involved in research and scientific development, any further observations you would have about that?

**A.** No, it feels very reminiscent of what I've currently about the serological diagnostics for SARS-CoV-2. I can understand the reluctance of people in the position of authority to be seen to be favouring an independent



sector approach from an academic or group of academics who say they can do something well.

I suppose I can understand it but, speaking as somebody who could have done something for SARS-CoV-2, we still have the best tests out there which predicts neutralising antibody and can we find anybody in this country to manufacture it? No. It's sad. I don't think it will make a great deal of difference because the commercial assays are out there now but, you know, we were ready to run with it last year.

**Q.** So would it be right to understand from your evidence that, both then and now, it might be thought that there's an infrastructure problem in terms of what one does and how one makes the best of the developments that scientists can make and how that is then put into practical implementation?

**A.** I mean, that's a difficult question to answer because there is a competitive air in academia. You know, academic centres will always want to see their centre benefiting as opposed to others benefiting. So there is -- it's not exactly a neutral environment in which you come forward and say, "I've got something which actually matters".

In generality, it would be nice to feel that if you came up with something which was really good, people



would listen to you. I mean, look what Mitchell had to do with the Spitfire. He had to take it out and fly it and demonstrate it and then its worth was recognised and so one went forwards. That's perhaps a very personal analogy because I'm an aviator.

But it would be nice to feel that there is a way of engaging with government, engaging with DHSC, which doesn't fall into "I'm breaching a zone because I want my academia to be recognised above everything else" which is, I think, how many people perceive academics. It's not actually that. Sometimes it's the academic group will have something which really has a benefit for society and it's finding a mechanism to enable that. That sounds awfully pretentious and I'm not trying to lecture people up there how to deal with this, but it just would be nice to have a mechanism where you could have a non-confrontational discussion with DHSC or somebody and say, "Look, actually what about this? This matters".

You know, we almost got it right. We have had it right sometimes with people, but it would be nice to have it out in the open and a mechanism for doing that.

**Q.** Can I then ask you to look at a document from October '83. This is moving on or moving away from the interactions with Dr Walford or others. It's

CBLA0001749. These are the minutes of a meeting of the MRC Working Party on AIDS. The date is 10 October 1983 and we can see there the list of members, departmental observers and then "by invitation" and you are there identified as being present by invitation.

If we could go to page 3, paragraph "(c) Aetiology" says:

"The aetiological background to AIDS was considered with passing allusion to the antigen overload hypothesis. An increased microbiological load with multiple infections associated with active virus replication in the host was thought a possible mechanism for immunodeficiency. The more widely held view that AIDS was due to a novel 'AIDS agent' was also discussed."

Then there is a further discussion which I'm not going to read out. We see there again the reference to the analogy with hepatitis B, however.

Then the next paragraph says:

"Retroviruses were considered ... it was noted that HTLV was a possible candidate on the basis of its known tropism for T helper cells. However a critical evaluation of the data led to the view that it was more probably an opportunist was unlikely to be the aetiological agent."



And then this:

"The assumption that the agent was necessarily a virus was challenged and the need to keep an open mind on organisms such as protozoa was stressed. Systematic antimicrobial therapy might provide leads on such agents. It was noted that blood product associated cases could enable some of these alternative hypotheses to be tested."

Given it's October 1983 by this time, it may be suggested that it's surprising that there is challenge to the assumption that the agent was necessarily a virus. Do you have any recollection now of from whom that challenge came, the list of members if we go back to the first page?

**A.** No, I don't, but looking at it, there is always an interplay between epidemiologists and microbiologists, and there's also an interplay between microbiologists of the larger organisms, the parasitologist, the medium size which are the bacteria, and those who deal with the smaller things which are virologists, and there will always be a discussion with something new.

If the pattern of the disease fits with hepatitis B, as I think it did, those of us who are virologists will say, "Well, that feels awfully like something we've seen before in the forms of transmission of hepatitis B".



The parasitologists and the bacteriologists will say, "Well, hang on a tick. Okay, we hear what you say but are you sure that it's not a bacterium? Are you sure it's not a parasite or a protozoan?" The answer is no, but one's overall feeling as a virologist is that this fits very well.

Then you would need to ask a parasitologist whether this fits well with the parasitilogical aetiology or bacteriological aetiology. Does that answer your question?

**Q.** Well, I think probably it's best you're able to, given that I was asking whether you had any recollection of from whom the challenge came and 40-odd years later, 39 years later that might be a --

**A.** The simple answer, ma'am, is no, I don't recall but I think I can see the reasoning behind it.

**Q.** I'm going to move now to the topic of donor selection. At this point in 1983 there is as yet no test in relation to HTLV-III/HIV. We'll come on shortly to your work in developing a test.

So donor selection was a means of reducing the risk of transmission through blood and so I want to go back to what you said to the Archer Inquiry on this topic, ARCH0000011, and if we go to page 154. It picks up on the evidence you've already given us earlier, professor,



about Dr Dane in a little more detail. If we pick it up at line 15, and again the context of the discussion had been about self-sufficiency, but you say this at line 15:

"Well, I think one of the -- this probably sets me apart from a number of people, but I think the question of blood products and blood safety, one of the most important mantras I still believe, even in this day and age is 'know your donor'. Know your donor and know the infection risks in your donor.

"If I can just step sideways from your question for a moment and give you a current example, we have good British donors who go abroad and they go to some area of the world where there is something else out there that we don't know of, for example, malaria, dengue, which is a virus infection, not terribly nice, rabies, we have had transmissions of rabies in Europe and other virus infections."

Line 6:

"These sorts of issues show that you need to know your donor and you need to know the environment in which your donor is, where they come from, where they are travelling. Obviously it is not xenophobic, we restrict people who have been in areas of the world where these microbial infections are common. That becomes

an extension of the concept to be self sufficient:

"Self-sufficiency was driven not so much -- in my experience, was not driven by the financial requirements or ease of manufacture or trying to protect a home market, it was just merely a principle that it is much better to take your blood and tissues and organs from donors whom you know where they have been, they are in your country and they will not harbour something which is not enzootic, endemic, whatever, whether it is in animals or humans, in this country, and they will not bring something in. That is the principle of self-sufficiency."

Then you tell us in your statement, and you told us before, this was a view you had effectively inherited from Dr Dane.

Now, in the UK then, even in the UK, so leave aside the question of importation of concentrates from America, to "know your donor", would it be right to understand that there has to be some attempt to assess and understand the behavioural and travel history of the donor and questions have to be asked, therefore, of the donor?

**A.** You've put two things together, travel history and knowing about -- (overspeaking) --

**Q.** I have.



**A.** Both. You need to know what behaviour, what travel, where and why a donor has been in order to have a reasonable aspiration of what the risks are.

**Q.** Sometimes those might be questions that are easy to ask in the environment of a donor centre but sometimes they may be questions that are difficult to ask, in particular if it brings into play issues about lifestyle, homosexuality, drug use and so on?

**A.** Yes. I mean, this is one of the difficult issues. And the most graphic example would be a husband and wife being bullied by friends to go and give blood. So then the husband is asked personal questions about sexual preferences or sexual exposure and it is very difficult -- you can imagine the situation where somebody who doesn't disclose a risk to his spouse has to disclose it or should disclose it to whoever is asking the question. So therefore you need to have the question to the donor being given in a controlled environment without other people listening, including spousal contact, and that can be difficult.

**Q.** We heard evidence in the Inquiry that within the North London Regional Transfusion Centre they were able to develop certain initiatives, in particular a questionnaire, which would mean someone would still give their blood but they would be able to indicate



privately on a document that their blood should not then be used for transfusion but could be used for research?

**A.** Well, we know, don't we, that not everybody actually got the questionnaire. So it actually has to be more proactive than that. It has to be a controlled environment in which you have a question and answer session between the examiner and the donor.

**Q.** If we go back to your statement then, please.

WITN3436003. Page 36.

So bottom of the page 36, paragraph 108, you make the point that we have just been exploring about being able to assess behavioural history and travel history:

"... central component of blood safety ... all comes down to - 'do you know your donor; do you trust them, are they telling you the truth?'"

Then if we go over the page, you say this in paragraph 109:

"I did hold this view in the 1970s and 1980s and I believe that reasonable and sensible efforts were made to ensure this in the UK ..."

And then you say this:

"... although my own view, when AIDS emerged, would have been to seek to exclude from the outset all men who had sex with men. I do understand why this was not the initial response based on the information as it was



being received from the US where the target for exclusion was the homosexual man with multiple/many sexual partners/contacts."

Now, I'm not going to take you to the AIDS leaflets. You have seen them for the purpose of your statement, and the Inquiry has looked at them on a number of occasions, but we know that that first AIDS leaflet sought to discourage people who have, present tense, many sexual partners. Men who have many sexual partners with other men.

And as I understand it from your statement, your view was that the better course would have been to ensure that all those who had or had had sex with a man at any point in time would be excluded?

**A.** Yes.

**Q.** So although, as I understand it, you are not critical of the efforts that were made, the initial donor leaflet, at least viewed in retrospect through your statement, in your view didn't go far enough?

**A.** I think we had discussions, and if you are asking me when and where, I can't recall, but we, as a virological fraternity, talking to the transfusion service, would have preferred to have any man who has had sex with another man. Not asking whether it was receptive or insertive but just if you had had sex with another man,

full stop, we would prefer you to be absent yourself from being a donor. However you do that and however you deliver that policy is open to a lot of discussion, but that is ultimately the end stage. You want that person to not present themselves as a donor. And preferably not present themselves for a donor to be turned away or to have to step down, because that puts a lot of potential strain on the family relationship.

**Q.** You referred in your statement to a meeting that you attended with Professor Contreras and Professor Barbara, as they now are, with representatives of the gay community in London, and there was initial concern expressed at what was seen to be potentially discriminatory towards those who were gay. But is it right to understand that, as the matter was talked through, those concerns were able to be addressed and you describe in your statement there being, at least in relation to the North London Regional Transfusion Centre, a constructive relationship with the gay community?

**A.** Yes. I wish I could remember the name of the young man who became quite a friend of us at the transfusion service. And it was actually the inception of the Terrence Higgins Trust which became very much a mechanism for interaction between young men who have



sex with young men and the community, whichever part of the community, outside. So that was a meaningful and useful interrelationship exchange of views and advice -- bilateral advice, how to find the best way forwards.

**Q.** Then, if we just look at minutes of a meeting of the working group on AIDS.

It is at DHSC0002251_011.

These aren't the minutes of a meeting, this is a minute dated 27 November 1984 -- the date is on the second page, we don't need to go to it -- from Emmy Abrams in the Department of Health to Dr Harris, who we know was a deputy CMO. But it refers to a meeting of the Advisory Committee on the National Blood Transfusion Service Working Group on AIDS.

In terms of test kits we will come back to that, but I want to pick up what's said at paragraph 1(iv):

"They were not in favour of closer questioning of donors to see if they were homosexual etc. They were in favour of a local session leaflet (such as is used now) which gets people to answer a list of questions amongst which are the AIDS questions. There was concern that too close a questioning might be counterproductive."

I don't know whether you can help us in understanding the thinking there. Why would too close a questioning be counterproductive?



**A.** Well, we are in a similar scenario today, where there is not enough blood to supply the needs of the NHS in the UK, and I think there was a concern -- you could interpret counterproductive in two ways, and you are asking me now and I'm not quite sure what you meant by that, I -- there would be the counterproduction of making people not disclose because their family were present and didn't want to disclose that they weren't fit to be donors. So that was one counterproduction of not having the right mechanism for asking.

The other is that, as we are at the moment, we have a shortage of blood to supply the requirements of the Health Service in the UK this week and that close questioning might lead people to say, "Oh, you know, I went up to give my blood and I was put in a room and I was questioned for 15 minutes as to whether I was a man who had sex with men, et cetera, or whether I was doing recreational drugs, and I am not going to go back and give blood again". That sort of approach -- that sort of -- I'm not sure it's right to call it "aggressive questioning", but that in-depth questioning in front of other members of the family might mean that your donor panel becomes reduced from -- and you lose people who are really perfectly valid, good donors.

It is a question of how you deliver what you think



is safely in a way that is not going to have counter production, not become unsafe in terms of losing too many donors. And you need to explore it with people. We also need to explore it with donors and ask them how would they prefer to be questioned about this. You can give people a leaflet but, as we know, some of the leaflets weren't seen, they weren't interpreted properly, and, at the end of the day, it's got to be safe for the recipients of the blood and blood components. There is no question of that.

**Q.** Do you know whether there was ever any systematic attempt to assess or model what might be the effect on the blood supply, or was it really at a level of there just being a general fear that there might be adverse consequences for the blood supply?

**A.** If you are asking were there any objective attempts to use different approaches and measure the outcome, I'm not aware of them. I think it was a general concern that, as we see at the moment, it is sometimes difficult to have sufficient donors prepared to come forwards to support the needs of the NHS.

You know, loss of 5% of the donors because of taking -- if your approach to donor questioning was unfriendly and aggressive, you can see that donors could take umbrage, you lose 5% of your donor problems, we are

already 17% short of what we need. You know, it is a risk either way, and you have got to find a way to protect people. That's what we have to do, in the broader sense.

**Q.** Just going -- in terms of questioning people in a way that's aggressive or confrontational, of course it ought to be perfectly feasible to know your donor, to ask your donor questions in a way that is friendly, non-adversarial, and which explains the importance of why these questions are asked and the importance of answering them truthfully.

**A.** Whether it is where your recent travel has been or whatever. And this would be central to David Dane's mantra: know your donor -- get to know your donor.

**Q.** Just one final document on this topic, which is one of the documents you saw this morning, Professor Tedder. It's DHSC0002249_026.

And it is a transcript from The World This Weekend on BBC Radio 4, 18 November 1984. We can see the trigger for you, and indeed Dr Gunson, then being interviewed, appears to be the reports from Australia that there had been the deaths of three babies who had received transfusions of blood donated by a male homosexual suspected of suffering from AIDS.

If we go over the page, we pick it up halfway down



the page, there is reference to the work that you were undertaking to establish a test, and then you are asked whether you felt:

"... following events in Australia that the risk were such that homosexuals in this country should be banned from becoming blood donors."

I want to read what you said, because obviously this was relatively contemporaneous, this is what you were saying in November of 1984:

"No not a ban so much as a self-inflicted ban, perhaps, rather than a state-controlled ban. One would hope that the transfusion centres would educate their donor panel sufficiently to make it very apparent, and easily apparent to the donors why they should not give, and how socially irresponsible it would be of them to continue giving if they were male homosexuals."

Then the question:

"So you are saying, then, that no practising homosexuals in this country should now give blood?"

Your answer:

"Yes, and this has been the Department of Health's policy in this country for a number of months now."

Question:

"Do you suspect, though, that they are still giving blood?



Your answer:

"Yes, I know without doubt that male homosexuals continue to give blood. It is particularly important when you have a blood team going to a place of work, like for example a factory, and you have half a dozen men coming up to give blood, and they all read the AIDS leaflet, and if somebody then turns round and walks out from the public area, all his mates will say, oh, didn't know about Fred being gay. Now, this is a, this is a very real problem, and the way the American clinics have got round this is by having nurse attendants, or physician attendants, who interview on a one-to-one basis the donor, and they can ask them in private. And indeed, in New York, where infected donors are probably of the greatest prevalence anywhere in the world, they have enabled the system to work whereby the donor comes up, gives his blood, and then at the same time as giving the blood, says whether it is to be used for research purposes, or whether it can be used for human therapeutic purposes."

Just pausing there. The evidence we have heard, Professor Tedder, is that that was the system that was then -- deliberately modeled on the New York system, that was introduced specifically into the North London Regional Transfusion Centre.



Then your answer continues:

"This allows the homosexual to have his blood taken to make some contribution, if you like, towards research, but marks the serum and the blood product as not being fit for human consumption."

Then you are asked about the leaflet and whether that is enough and you say this:

"Well, at the risk of making myself unpopular with my colleagues in the blood transfusion service, I think it is very clear that the exposure of donors to the AIDS leaflet is insufficient, because I myself have two male friends, who are not gay, incidentally, but have acted as blood donors, and in both instances, neither of them were asked, nor did they see the AIDS leaflet. Now, that is worrying if that can happen to two friends out of the small number of blood donors who are my personal friends."

Then, over the page, you were asked your view on the position in Australia, and you say this:

"In the United Kingdom, the proportion of blood donors who may be infected with the AIDS virus is vanishingly small. That is true of 1984, whether it will still be true in 1985 or '86 I think is questionable. It must be seen only as a matter of time before British blood products and British donors become

contaminated with the virus."

Then the interview goes on to interview Dr Harold Gunson.

18 November 1984, you would have in fact known by this time, because of your testing of Dr Ludlam's patients, the Edinburgh cohort, that British blood products had been contaminated with HIV?

**A.** Yes, I mean, I think Professor Ludlam's problem up north was not -- I don't think any of us anticipated that. He certainly didn't. His aspiration was to show that his recipient panel was clean -- clear of HIV infection, and this did not take into account the high and expanding frequency of use of -- injectable drug use for recreational purposes on the east coast of Scotland, to the east of Edinburgh, where there was quite a body of recreational drug usage. Which, of course, had been -- I use it in the non-inflammatory sense -- exposed by and used by HIV to become established in that population. Exploited.

**Q.** We will come back later no doubt this afternoon to further issues relating to Professor Ludlam and the testing.

Sir, I'm going to move now to the development of the HTLV-III test. Rather than start that with 4 minutes to go before lunch, shall we break now for lunch and I'll



start it after lunch?

**SIR BRIAN LANGSTAFF:** Well, it is quite a big issue, so let's take a break now shall we until 2.25 pm.

**MS RICHARDS:** Thank you, sir.

**SIR BRIAN LANGSTAFF:** 2.25 pm.

**(1.26 pm)**

**(Luncheon adjournment)**

**(2.25 pm)**

**MS RICHARDS:** Professor Tedder, this afternoon I'm going to be asking you about the development of the test for HIV and then its introduction into the transfusion service.

Before I do that, can I ask you to just tell us what the difference is between an RIA and an ELISA in terms of the two assays?

**A.** Right. The only difference -- there is actually no difference between an RIA and an EIA *per se* because the components of the tests are the same, but the only difference is that the read-out in a radioimmunoassay is radioactivity and the read-out in the EIA is colorimetric. In fact, some EIAs are very much more like RIAs because the generation system actually generates photons; so you measure a signal, as you would with radioactivity which is measuring radiation, and in EIAs you measure photon emission. The simple EIA is a system where it changes colour but the more modern



ones actually generate photons. So it's is just the signalling is different.

**SIR BRIAN LANGSTAFF:** May I ask if the difference is essentially in the label which is attached?

**A.** It is. The label is radioactive in one and you measure gamma radiation. In the other, it's colorimetric and you measure the colour -- essentially that. Although those are the simple two extremes, there are some forms of EIA which actually measure photon emission, which is very similar to making gamma emission, so they can meet in the middle.

**SIR BRIAN LANGSTAFF:** Yes.

**MS RICHARDS:** Now you in the early 1980s were working on the development of a HTLV-I screening test and that was how you first came into contact with Professor Weiss; is that right?

**A.** Yes, it would be because I needed to -- I wanted to have a serological test, therefore I needed to have an antigen (that's a protein from the virus) and I didn't have the facilities to grow that virus because we needed strict containment -- and I got into serious trouble once because something wasn't contained as it should have been -- and I needed the virological proteins, therefore I liaised with Robin Weiss.

**Q.** And you describe in your statement the nature of the



assay that you developed, and you used these terms: it was a one step simultaneous competitive assay. Can you just help us understand what that is?

**A.** Right. This is where you really need a board. Okay, here we have a piece of plastic which is on this side it's solid and there is a hole in here and this would be known as a microtitre well, and it's round so you can put liquid in here, and it's constrained by the plastic.

On the plastic, we have a protein. This is the protein stuck on the plastic. If I'm making what I call a competitive EIA, here's the protein, here's the label that wants to get on there. So if I ask somebody to come up and block this, they will put their hand on there. Do you want to do this?

**Q.** No, I think if you just talk us through it it's easier, otherwise the microphone doesn't pick me up.

**A.** Okay. There is the solid phase with the protein on it. Here's a label which is going on to there. If somebody wants to get on there first or is stronger than getting on this, they block this from going on there. If this is an antibody that's going on there and it's stronger than this, this can't get on there so you lose the signal. So you get a direct competition. You go from generating signal to completely losing the signal and that's because of competition.

So does that -- is that -- do people follow that principle? So it's a competition binding onto there and it is blocked by an antibody. That is what I call a competitive immunoassay.

**Q.** You've explained in your statement and, I think, produced a slide that there are a range of different assay formats, antibody assay formats. We'll just put it briefly on screen.

**A.** Yes, please.

**Q.** It's WITN3436004.

**A.** I hope this is going to be the slide -- yes. Okay.

**Q.** So the competitive assay which you were just describing is the type 3?

**A.** Yes. I mean, I would say, ma'am, that I'm probably one of the few people who uses type 1, type 2, type 3, type 4. I use it because, as a serologist, it's easier to say, but basically you can see in the type 3 you've got one incubation where you incubate the label, antibody with the red star on it, with an antibody with no star on it. And if there's a lot of no antibody with no star, the antigen -- the label doesn't bind and you get no signal. So you go from having a strong signal, with the absence of antibody, to no signal with the presence of antibody.

**Q.** You again explain in your statement that your view was



that the competitive assay was the most effective?

**A.** I'm not sure that I would use the term "effective". Can I just explore that for 30 seconds?

**Q.** Yes, absolutely.

**A.** The advantage of the competitive assay is twofold. One is that you have to have, as you look at that slide -- go across the bottom, the lower antibodies, the competing antibody, it has to be an effective competition otherwise you don't get a signal. And if you have sticky sera, which would give you non-specific signals in the type 1 assay, which is what manufacturers make, you would get a signal in the type 1 assay but you got no signal in the competitive type 3 assay. And when you are dealing with something which is really a damaging infection, if you're telling somebody that you've got antibody, you're infected, you need to be certain or you need to be as certain as you can of the specificity because if you get a reputation for having a bad test, nobody will come forward to be tested and it and that's damaging to the environment, that's damaging to people.

So I personally like the type 3 competitive assay, and I was working on HTLV-I and HTLV-II with a type 3 assay and it was natural to do exactly the same for HIV and come up with what eventually became the Wellcozyme



assay.

**SIR BRIAN LANGSTAFF:** May I ask, the black Ys, horizontal Ys that we see there, they are the antibodies?

**A.** They are, sir.

**SIR BRIAN LANGSTAFF:** The protein of interest is, what, the antigen that you suspect might be there, is it?

**A.** Yes. The target protein in the key is whatever virus you want is the protein that is specific to that virus or from that virus.

**SIR BRIAN LANGSTAFF:** So when you've got the fluid which you're testing, the sera, which contains the -- may contain the protein of interest and may not, when it goes into the wells on the plate, and the mixture of sera is passed over it, the antibodies will bind to any antigen, any protein of interest in the wells. Is that the position? But if there isn't any antigen, they won't?

**A.** Broadly speaking, that is correct. The difficulty in the type 1 assay is if you take that target protein away -- that's the top left-hand corner where it says "first incubation" -- you've got the plastic which is the sheared part, then you've got a face and then you've got the viral protein.

If you take that viral protein away in type 1 assays, you quite often find there is a significant



antibody binding because the antibody sticks to the solid phase. This is particularly true in areas where you have other infections causing an agitation of the immune system. Malaria is the classic one where type 1 assays are an absolute nightmare in malarial countries, and they found that currently with the assays for SARS-CoV-2, that there is so much stickiness, the assays are non-specific.

That is the great advantage of the competitive test. You have to have an antibody of sufficient quantity and sufficient magnitude, which you frequently find in an infection, to block the specific binding of the label onto the antigen. And it gives you -- it's a one-step assay. You put the two components into the well, you leave it for an hour, maybe an hour and a half, two hours, then you wash it and develop it. So you don't have a second incubation. So technically in the laboratory it's quite easy to do.

**SIR BRIAN LANGSTAFF:** Thank you.

**MS RICHARDS:** So you had used this type of assay to develop the test for HTLV-I. You then used the same principles essentially to develop a test for HTLV-II?

**A.** Can I take you back about 30 years?

**Q.** Of course.

**A.** I developed the same protocol for hepatitis B for core

antibody in hepatitis B and it was very easy, having done that. So when a new virus came up, or any virus came up, we could make a competitive assay. I actually played with making one for cytomegalovirus at one stage and then when HTLV-I and II came round, I made competitive assays for those. So when HIV or HTLV-III came round, it was a natural pathway to go down.

**Q.** So if we then go to your statement, WITN3436003, page 56, you say this in the paragraph at the bottom of the page, and this is in relation then to HTLV-III:

"The matter then became one of waiting for a suitable sample that could be used to develop a working HTLV-III test."

So is it right to understand that once you had become aware of HTLV-III, you understood or believed it was a virus. You knew you had this methodology that you describe through the HTLV-I and HTLV-II in your earlier work. Was it the case that you realised fairly quickly that you might well be able to test for this using the competitive assay, and what you needed was then the sample in order to complete that process?

**A.** We needed -- how can I -- putting it in the terms that we had the antibody on the solid phase. We had antibody which was labelled in the fluid phase and we needed a supply and when I say "we needed", it was my



laboratory for my research and my diagnostic work locally, I needed a supply of antigen that could go on to the solid phase and then act as a component for an assay.

In fact, the easiest way was to have an antibody on the solid phase which would pull the antigen down, stabilise that, and then use that as a solid phase for the competitive assay and it was -- from my point of view, I did not have the facilities in the very early stage to grow up a category 3 component in a safe laboratory without breaching health and safety regulations.

**Q.** Now, in terms then of the wait for what you've described there as a suitable sample that could be used, I just want to try and clarify what happened or what may have happened in relation to the offer of a sample from Montagnier's team in Paris.

You put it in a handful of different ways in your statement. I do not mean that in a pejorative sense, professor, I just want to look at what you say and try and work out what happened in relation to that and when.

If we go, first of all, to paragraph 53, which is page 17, you talk about being aware of the work of Dr Brun Vezinet and Luc Montagnier:

"... wished to derive a UK isolate. ... we asked



for and were provided with a sample, although some difficulty was experienced with the initial delivery. I [don't] recall precisely when I ... became of their work, although I was aware of it when it was published."

You refer to meetings, including with Francoise Brun Vezinet. That's the first way in which you put it in your statement.

Then paragraph 179, which is page 56 -- so the paragraph we were just looking at -- you refer at the bottom of the page there to a sample from Montagnier going into transit but ending up being lost due to delays and then, finally in this statement, paragraph 459, which is page 138, you say in the third line:

"As far as I recall the original sample of the viruses from Paris was safely packaged and sent in the mail but stayed in the post office over the weekend and the cells died."

So just in relation to that first sample sent from Paris to the Chester BT laboratory, or Chester BT Institute, what is your best understanding of what happened and when?

**A.** It was despatched by Luc Montagnier and his colleagues for Robin and, for some reason, it got caught in the post office over the weekend and was not kept -- I do



not know whether it was live and wet at the time it was sent or whether it was frozen and cryo-preserved ready to be revived when it came into Robin's laboratory. Either way, by the time it came into the laboratory, the cells were no longer viable.

**Q.** And do you know roughly when that was? Autumn '83 or -- I think I've seen a reference somewhere, not necessarily from you --

**A.** Well, it would -- without going into the paperwork or asking Robin, I don't know but it was obviously very early on because that was the cell culture from which he was going to deliver us a secure supply of what would have been LAV 1 or LAV-Un.

**SIR BRIAN LANGSTAFF:** I think Professor Weiss may say in his statement -- and he ascribes a date of October, I think.

**MS RICHARDS:** It's either September or October. It's the autumn of 1982; that's right, sir. Then just if we put up on screen a letter that was sent by Professor Weiss to the MRC in May 1984, JEBA0000148, and we can see the date 11 May and then, if we look at paragraphs 2 and 3 in this letter, paragraph 2 refers to collaboration with medical virologists, in particular with you, Professor Tedder, and the development of radioimmunoassays, described there as specific and sensitive to HTLV. Then Professor Weiss says:

"We already have Montagnier's isolate in our laboratory and I hope to have collected Gallo's isolate by the time you receive this letter."

Now Professor Weiss's statement indicates that what happened was a second sample was sent over from Montagnier in February 1984, which may explain then the reference to having Montagnier's isolate in the laboratory. Do you have any further knowledge of how Professor Weiss was able to confirm that he had the isolate from Montagnier from this date?

**A.** No, I don't because, although I worked closely with Robin and he was a friend and support on this, that was his area of activity, not mine, because, as I say, we did not have at that stage the facilities for growing a category 3 pathogen.

**Q.** And just for the benefit of the transcript, Professor Weiss's statement -- we don't need to put it on screen at this stage but it's WITN6868001 -- I don't have the paragraph number to hand but he explains that a second sample was sent in February '84.

Now, there's then reference there to a waiting for an isolate from Gallo or expecting one at around this time. What's your recollection of how it was that you eventually --

**SIR BRIAN LANGSTAFF:** I'm sorry to stop you there. We've



got a problem, a technical hitch, with the stenographers so we just need to take a quick break to sort that out.

**MS RICHARDS:** Okay.

**SIR BRIAN LANGSTAFF:** The reason I'm stopping you is because it's on the transcript, the transcript therefore has to record everything and it's not doing at the moment. I'm sorry about that, professor. We'll take a five-minute break.

**MS RICHARDS:** Thank you.

**(A short break)**

**(3.04 pm)**

**SIR BRIAN LANGSTAFF:** Thank you for your patience. I understand that normal service is resumed.

**MS RICHARDS:** It appears to be sir, yes.

**SIR BRIAN LANGSTAFF:** Let's try it and see.

**MS RICHARDS:** Just to give the reference that I had previously given, I referred to Professor Weiss' statement, and I found the paragraph number in the break. It is paragraph 320 where he says that the second sample from Montagnier was sent at the end of February 1984.

If we just go back to your statement in any event Professor Tedder -- so WITN3436003, page 57 -- you explain in the paragraph at the top of the page your recollection that the sample was "derived from



a cell-line provided by Gallo", and then you refer to something called CBL1.

We have got a statement from Professor Weiss but can you just tell us what your recollection is of how it was you ended up, ultimately, with something you could use, and tell us what CBL1 is?

**A.** So from the time of receipt of a cell line through to having an antigen I could use for a culture; the first thing I had to do was to improve the security in the small laboratory which I had at Middlesex Hospital Medical School because we had already had a rather unfortunate episode where, from a ventilator, an exhaust should have gone -- went into a safety bottle and then out of the safety bottle and the safety bottle had been left outside the category -- the containment laboratory extraction platform, and that caused a significant amount of grief with the Health and Safety Executive.

But we worked round that and eventually agreed to have a -- culture flasks in the laboratory, and we experimented how to get the production of the antigen.

And you are going to ask what antigen are and I will try and explain that in a minute, because you had to ...

We found out that if you had the cells growing uninfected in a big flask and you inoculated them with the virus, that means so you put the virus in to infect



that culture and you get the culture running for a few days, the turnover of the cells would produce into the fluid, but not into the cells, an antigen which we could absorb on a solid phase and make a competitive antibody assay work very well.

The road to this was to have a big flask, with cells at a good density, and ready to infect them, and this is what we asked our colleagues in Porton to do and they actually elected to do it in a different way. They elected to take infected cells, put them in and grow them up in a large quantity. And that did not give us the antigen we needed. There was something peculiar -- or something unique by having a lot of uninfected cells pulsing the virus in, waiting a short time and taking the supernatant, as opposed to having a small number of infected cells and letting them grew up and take the supernatant. The first method worked, the second method didn't. And it is something to do with you encouraging rapid growth of cells to release virus envelope, which didn't happen if you expanded the cells from the small inoculant ready infected. That's why there was a difficulty in making the antigen. But we eventually got round that.

**Q.** Are you able to say whether if you had had access to a suitable sample, from whatever source, Paris, US,

developed by Professor Weiss, whatever source, if you had that in, say, summer or autumn of 1983, would you have -- would that have meant you would have been able to develop your test earlier than you did? Or were there other constraints?

**A.** Can I just -- be careful with the word "develop" the test. Because we had the test running on a small amount of virus, grown up in small vessels, to cover the amount of work that we wanted to do both in our GUM clinics and in the Local Health Authority. So we were not constrained for doing work that we wanted to do, the research and development work that we wanted to do.

What we could -- what we were not in a position to influence were those who wanted to grow this up to be able to supply a manufacturer with an antigen which they could use to make a manufactured version of our assay. And that was one of the difficulties, that our colleagues in Porton had to rethink how they were going to grow the antigen. And when they elected to follow our protocol as having the cells grown up, infect the cells as a -- at a point in time and then harvest, then they could come up with an antigen which would work.

**Q.** If we go back a little earlier to the point in time -- you tell us in your statement you had a working RIA that you could use for your purposes, and we will look at



The Lancet paper which shows how it was being used in a moment, but you had that working RIA by July 1984 or thereabouts, as I understand it.

Would you have been able to get to that stage significantly earlier if you had had the sample significantly earlier? Or is it impossible to say?

**A.** I am sure if it had been easier and quicker we might have saved a few weeks. But at the end of the day we needed to develop an understanding of how the assay worked, what its advantages were, what its disadvantages were, what its sensitivity was.

And that's sensitivity in two senses. Sensitivity, if you have a population like this room and some people infected, how many of them could we detect. So that is, in clinical use, the sensitivity of the assay. And then, speaking as an old-fashioned virologist, I've got five positive samples and I'm diluting them in negative plasma, how far can I detect them, that is the analytical sensitivity as opposed to the diagnostic sensitivity. And we had to work out both those parameters for the development of this assay.

**Q.** Can we then look at the September 1984 Lancet publication, just to see where matters had got to by that stage.

It is NHBT0000068_015.



We can see there the date, 1 September 1984. We can see the range of authors. And then if we look down towards the bottom of the page, if we just look at the summary:

"2000 persons in the UK were examined serologically for antibodies to ... HTLV-III. Sera reacting in a membrane immunofluorescence assay (IFA) to HTLV-III were also positive when tested against cells infected with lymphadenopathy virus (LAV 1), and cross-adsorption tests indicated that these retroviruses are probably identical. A competitive radioimmunoassay (RIA), which was wholly concordant with IFA, was used to screen the sera. 30/31 patients with the acquired immunodeficiency syndrome (AIDS) were seropositive, as were 89% patients with persistent generalised lymphadenopathy (PGL), 17% symptomless homosexual men, 34% haemophiliacs receiving pooled clotting factors, and 1.5% intravenous drug abusers. None of more than 1000 unselected blood donors was seropositive. These data confirm the close association between HTLV-III and AIDS and PGL and show that infection with HTLV-III is also prevalent in the populations in whom these syndromes are most likely to develop."

So that's the summary.

Then if we just go over the page, please. Under the



heading "Materials and Methods", we have got the first subheading, "Subjects". So we can see the groups who were tested. And so we've got: patients with AIDS, patients with PGL, symptomatic homosexual patients, sexual contact of AIDS patients, homosexuals at risk, heterosexual subjects recruited from GUM clinics, intravenous drug abusers being screened for hepatitis B. And then:

"(8) Haemophiliacs undergoing regular clotting factor replacement therapy, sometimes with American commercial factor VIII concentrate."

And then:

"(9) 1000 unselected blood donors.

"The patients were drawn from the Middlesex, St Mary's, and St Stephen's Hospitals. Blood from groups (1)-(6) was collected between June, 1983, and July, 1984, and the sera were stored ... Sera from haemophiliacs had been collected since 1982."

In relation to the sera from haemophiliacs, do you have any knowledge as to where that came from, in the sense of which centres?

**A.** No, I don't. But these would have almost certainly come through collaboration with the haematology department at the Middlesex and their contacts. Not necessarily samples would have come from the Middlesex but would

have been generated by Professor Machin and other colleagues.

**Q.** I think we also see Dr Craske as one of those named as those involved and it may have been samples through him as well?

**A.** Yes.

**Q.** Then we have the heading "Virus and Cells". Then we are told:

"HTLV-I, HTLV-II, and HTLV-III were kindly supplied by ..."

Then we can see the reference to Gallo and colleague.

Then LAV-I from Montagnier, Paris.

And then:

"HTLV-III was provided as a persistently infected cell-line HT ..."

I think the first part of that is self explanatory. Can you just tell us what that means, "HTLV-III provided as a persistently infected cell-line HT, clone H9"?

**A.** Well, it is a particular type of cell line. The HT cell line would be a human T cell line. And clone 9 means somebody has selected derivatives from the master culture and each derivative is called a clone. So it will be clone 8, 9 of human T cells.

**Q.** Then it continues that the:



"LAV-I was provided as a cell-free culture supernatant ..."

And so on.

Then we have the heading "IFA". So the immunofluorescence assay.

I won't read it but can you tell us essentially what that refers to?

**A.** Okay. This comes back to a question from the chairman about methods of detecting antibody. EIA, IA, immunofluorescence or radioimmunoassay. This is actually using an immunofluorescent antibody directed against human IgG.

So what you -- in this particular case, you've got the cell line growing on plastic. You've infected the cell line with, in this case, HTLV-III. So -- so start again. They won't have been ready on the cell line. So you've got infected cells growing in. You've spread them onto a surface. You've let them dry on the surface and you have stabilised them.

So there is the plastic. Here are the cells with the virus on it. So now you turn it down like this. So you've got the plastic surface or surfaces, and you have got the cells on there, each of those cells is expressing HTLV-III as it is growing in those cells.

You've fixed the cells. You basically killed them



and you dried them. And that is then a dried layer of cells which contain HTLV-III antigen.

You then take your serum from somebody. Known positive, known negative. You dilute it a little bit in buffered saline. And then you put it on there and you leave it for a period of time.

And antibody, if there is antibody in there, will stick onto the antigen. You then wash it and you say: okay, is there any IgG bound on that cell by putting in a fluorescence labelled anti-antibody? So if there is antibody there, the fluorescent label comes on and sits on there, so when you hit it with ultraviolet light it says, "Hey, I have got a bit of conjugate stuck on here". And when you actually do this, you can see the pattern of the conjugate sticking onto the infected cells.

**Q.** Then if we go to the top of the next column. So same page, but the right-hand side. We can see the heading, "Competitive Radioimmunoassay (RIA) for Antibodies to HTLV-III". That is the assay you have been describing to us that you developed?

**A.** Well, the HTLV-I assay was a competitive, pull it down, come in, then block it. That's just saying, okay, we have done it with HTLV-I, HTLV-II, here we do it with HTLV-III.



**Q.** Again, I'm not going to read it out but this article describes the process that was used in the assay. Then, if we go to the bottom of the page we have got the heading "Results":

"Comparison of Tests"

If we pick it up in the second paragraph:

"Sera which were reactive by IFA were all positive with RIA."

Then further detail given.

Can you just explain the significance of that sentence?

**A.** It means that there was absolute concordance between calling samples positive or negative in the two different assays, by immunofluorescence or by competitive radioimmunoassay.

**Q.** Then if we go to the next page, please, and we look first of all at table -- that is the second table down on the left-hand side. So it is table 2. So we have got the results there:

"AIDS patients ... 30/31 ..."

So that gives us the proportion with HTLV-III antibodies using these two tests. We can see, amongst others, the:

"Haemophiliacs who have received pooled clotting factors ... 63/184 ..."

So 34% had HTLV-III antibodies.

Then if we just go to --

**A.** Just before you leave that, people may be noticing that only 30/31 AIDS patients were antibody positive. The reason for that is you can get Kaposi sarcoma in people without having HIV. I think the one seronegative patient was a young man with Kaposi sarcoma in the absence of HIV infection.

**Q.** Then if we go to the bottom of this column and we have the heading "Discussion". It says:

"Two simple, reliable and specific assays for the detection of antibodies to HTLV-III have been described. Findings obtained by competitive RIA shows complete concordance with those obtained by the membrane IFA, indicating that results obtained by the two assays are comparable and that the RIA detects antibody and not antigen. In addition, the assays indicate that HTLV-III and LAV-I are indistinguishable."

Then there is a discussion which I am not going to read out about the results by reference to each of the different cohorts of serum donor.

Would it be right to understand the significance of what's described in this paper as being confirmation, first of all, that the assay that you had developed with Professor Weiss and colleagues worked in terms of



detecting the virus that caused AIDS?

**A.** I think that's right. It is comforting that a competitive immunoassay worked as well as an indirect antibody assay.

**Q.** And secondly it indicated that HTLV-III and LAV-I were essentially the same thing?

**A.** In terms of acting as a solid phase for an immunoassay, yes, they were.

**Q.** There is a second paper in The Lancet on that same date. I don't think we need to go to it --

**SIR BRIAN LANGSTAFF:** Before we go there. May I just ask because we started this discussion with a question about the timing of the test. This was a publication in The Lancet on 1 September. So the presumably it must have been in press, submitted to The Lancet a little while before that? Am I right?

**A.** I wouldn't disagree, sir.

**SIR BRIAN LANGSTAFF:** So, either sometime in July or August it would have been submitted to The Lancet, and the tests which gave rise to it must therefore have been in use before that time. Does that help at all with roughly knowing when the testing was done of these 2,000 samples, by the method you have described, in order for this article to be authored?

**A.** I mean, The Lancet is -- sometimes takes a little bit of



time to make its mind up, so I would suspect this was submitted to The Lancet in July, August, September, sometime -- July probably. And it's -- certainly July '84, early part of July '84 is when we were quite certain that we had an assay which was sensitive and specific and stable and reproducible in the form of a competitive RIA. Because we were -- interestingly, 4 July is the time we had a meeting, coincidentally, to say that we've a good test.

**SIR BRIAN LANGSTAFF:** So if any point arises out of the dating of the first test thought to be reliable in the UK, it would be early to mid-July '84?

**A.** There would have been other tests available. I mean, people had the immunofluorescence assay. I'm sure my colleagues in Chester Beatty would have a virus neutralisation assay. And we were just showing we had a competitive immunoassay which fitted with the routine -- if you can call it "standard" -- indirect immunofluorescence assay.

**SIR BRIAN LANGSTAFF:** So it is right then to say that various tests, probably, were available by mid-July 1984?

**A.** Yes.

**SIR BRIAN LANGSTAFF:** Thank you.

**MS RICHARDS:** And the second article in The Lancet, we don't



need to go to, but just for the benefit of the transcript, PRSE002140.

I just want to ask you about an observation Professor Weiss makes in his statement. So if we put Professor Weiss' statement on screen.

Lawrence, it is WITN6868001, and it should be page 25.

It is the bottom of the page. So this is in the context of a section of his statement in which he is discussing The Lancet article we have just been looking at, Professor Tedder. And then he says this:

"In terms of the overall significance of these findings, our article included a larger data set on blood donors and on recipient of blood products than previously published reports from any country. Perhaps more important for this Inquiry, the study revealed the actual rates of HIV infection in North London for donors and for different 'risk groups' for AIDS and more broadly in the UK for patients with haemophilia."

Then it is this sentence, really:

"The findings indicated the urgent need for screening to detect HIV infection even though the infection rates in healthy donors was currently low."

Would you agree with Professor Weiss' observation in that paragraph, but particularly in that last sentence?

**A.** Yes, ma'am. I don't think that necessarily it's completely linked with the previous data that we were going over, which was a small subset, but yes, I mean, the data here would have been in association with our colleagues in the transfusion service, and ...

**Q.** There had been, as I understand it from The Lancet article, the study that's written up in The Lancet article or the results written up include the thousand donors from the North London Transfusion Service. But would you agree that it indicated the urgent need for screening to detect HIV infection?

**A.** Well, yes, because what it's showing is whether one -- however one addressed this, the virus was present in the UK population and therefore, even though the prevalence in healthy donors was currently very low, you could not say how long that was going to retain low because, you know, once you have introduced an infectious agent into a population, if that agent is able -- if the reproductive rate of that agent is greater than 1, one person will beget two, two will beget four, and you are off onto an expanding population. So the sooner you can control it, the better.

**Q.** An issue I've been asked to invite your observation on is this: could the test that you've just been describing, and it's described in The Lancet article,



could it have been used to test plasma pool samples (for example, from PFC or BPL) or batch samples of Factor VIII concentrate?

**A.** Sorry? My hearing is not absolutely good. Could you say that last --

**Q.** Of course. So could the test, the competitive radioimmunoassay, have been in principle used to test plasma pool samples (for example, from BPL) or batch samples of Factor VIII concentrate?

**A.** Well, let's take it from the bottom first. Testing Factor VIII concentrate would not necessarily have an adequate level of antibody in it to be valid in any assay. I mean, it will have some but I don't know what level of dilution and sensitivity you would lose.

In terms of testing individuals going into that batch -- was that the first question?

**Q.** No. The first part of it was testing plasma pool samples, so the actual -- you got several thousand donations in every pool.

**A.** Okay. Then the same question applies. It could have been but I don't know the sensitivity. Let's say you have a 1 in 500 donors who are seropositive. You would have to say: would the test be adequately sensitive to always detect a 1 in 500 dilution negative plasma of the positive? And, frankly, I don't know at this time. The



assay was good but competitive assays probably are less sensitive in end-point dilution from an anti-globulin assay, but I really don't know.

It would have been interesting to do and whether it would have -- whether a negative would have been valid, which is what you need to prevent the onward transmission into risk populations receiving that plasma, I don't know.

**Q.** Now, if we go back to your statement, WITN3436003, page 31, we can see from paragraph 92, so the bottom paragraph on the page, you say around this period, and you're answering by reference to a question about 1984/85:

"I was one of a group of people providing testing as requested by haemophilia centres or directors. This was particularly so because the competitive assay was such an accurate test and therefore was in great demand."

We'll look at some examples of documents and, in particular, I've got some questions to ask you about the testing of Professor Ludlam's patients.

In the autumn of 1984, going into the beginning of 1985, can you recall who else within the United Kingdom was providing testing?

**A.** Who else?

**Q.** Who else was providing testing?



**A.** CPHL, the virus reference division at Colindale, would certainly have been.

**Q.** Do you know what tests they would be using? Yours or something different?

**A.** I think we collaborated with Philip Mortimer and he would have used at some stage the competitive RIA and we helped him -- I'm not sure if we helped him manufacture that or we sent him materials to build it. There would have been pressure to use the commercial assays which I think would have -- there would have been an Abbott anti-globulin assay and I suspect there would have been an Immuno anti-globulin assay as well.

**Q.** Other than Professor or Dr Mortimer, do you recall who else, I'm particularly looking here at the autumn of '84 and beginning of '85, can you recall who else, if anyone, was providing testing facilities?

**A.** Frankly, with the best will in the world, no. I'm sure there were but I've no idea.

**Q.** Then if we go to HCDO0000270_007 -- this may indicate that --

**A.** Could I just make an observation on 92 before it disappears off the screen?

**Q.** Yes.

**A.** We were approached by the haemophilia centre directors as a group to provide serological testing for them. So

that doesn't necessarily mean nobody else was doing it but it does mean that we had a very accurate and very specific assay.

**Q.** I think you'll find the document on screen confirms that. So the document on screen is the Haemophilia Centre Directors Organisation AIDS Advisory Document drawn up after a meeting at Elstree on 10 December 1984, and we can see it says in point 2:

"Tests for HTLV-III antibody are available for haemophiliacs via ..."

Then it's Dr Mortimer in Colindale and then you. So that would be consistent with your recollection.

Can we then just look at BART0000821. This now moves us to the beginning of January 1985. It's a meeting at the Middlesex Hospital Medical School on 3 January between you, Dr Mortimer and Dr Craske. Just on the issue of testing, if we go to the second page, there is a heading "recommendations". It says this:

"It was therefore decided to propose to the UK Haemophilia Centre Directors the following strategy for HTLV-III serology."

Then paragraph (a):

"All patients treated with Factor VIII and Factor IX concentrate in UK haemophilia centres would be offered an antibody test for HTLV-III antibody within the period



February to April 1985."

Then (b) is:

"Family contacts of patients. It was also hoped to offer HTLV-III antibody tests to relatives of patients who were found to be HTLV-III positive."

Then there's a reference to shortage of reagents and that would be better done by carrying out limited family studies to determine risk of spreadable infection before offering a test to all relatives. Then:

"(c) follow up of seropositive patients.

"(d) future HTLV-III antibody prevalence surveys."

Then if we just go to the top of the next page, paragraph 2 says:

"Haemophilia Centre Directors would be offered the chance of testing as many of their patients as they wished within the next three months. Haemophilia Centres would be asked to send sera to either Dr Tedder or Dr Mortimer for antibody testing."

Then there's a reference to copies of the report then being sent to Dr Craske at PHL Manchester.

It would appear from this that you and Dr Mortimer were effectively proposing offering to the Haemophilia Centre Directors testing of all patients with the expectation that would be between February and April 1985 and also some form of testing for family members.



The Inquiry has seen or heard evidence, in relation to some haematology centres, of patients not being tested until later than that -- so rather later than April 1985. We've seen evidence of patients being tested in the second half of '85 and indeed even into '86. Do you know why that was the case? Did you have -- was it difficulty in keeping up with the demand or was it simply that Haemophilia Centre Directors didn't always send the samples to you to ask you to test them?

**A.** No, I don't. I mean, I think it was an awfully unpleasant opening of an envelope and finding this virus where you hoped it wouldn't be and, you know, all I can say, it was, and it still is, devastating to look back on now and think of the harm which was going to arise from this.

All I know is that we tried to comply with all the Haemophilia Centre Directors' requirements and, at one stage, we were doing an awful lot of testing and I would say to you that the results of this are still -- should still be available. I left them behind at UCL when I left and the files should be available to the Inquiry through approach to virology at UCL.

**MS RICHARDS:** Sir, I note the time. Obviously we had the --

**SIR BRIAN LANGSTAFF:** Shall we go on until 4 o'clock because



normally we would have a half-hour break in the afternoon. We were scheduled to go on to 4.30 or thereabouts. If we go on now until 4 o'clock or thereabouts, we can have out -- in one sense, we've already had our break but --

**MS RICHARDS:** And then conclude for the afternoon at around 4.00.

**SIR BRIAN LANGSTAFF:** At a convenient time immediately or shortly after 4 o'clock.

**MS RICHARDS:** Certainly.

So I just want to ask you then next a little about the testing of Dr Ludlam's patients which was a little earlier than this. It was in the latter part of 1984. Before I do that, you referred earlier in your evidence today to an understanding about an IV drug use in Edinburgh.

In 1983/1984, do you recall there ever being any discussions about whether there were particular cities or locations within the UK, such as Edinburgh, which might, whether through international travel or IV drug use, become AIDS hot spots?

**A.** I don't, and I think that's evidenced by the surprise and the awful feeling of devastation with the results from the study we did with Chris Ludlam.

**Q.** Do you recall again from this time, '83/'84, any contact

with Dr Ray Brettle in Edinburgh?
**A.** Well, the name rings a bell but if you're asking me what the substance of the contact was, no, I'm afraid I can't at this distance.
**Q.** Then can you recollect how it was that you came to test Dr Ludlam's patients? How, by whom and on what basis was an approach made to you?
**A.** My belief -- and I say this as a belief. This is not fact, this is just I feel that it was recognised that the antibody assay that I had and which Philip was using at Colindale was of sufficient sensitivity and specificity to be able to be used to answer a really unpleasant situation of what is the prevalence of this infectious agent in a population, and I think Professor Ludlam realised that we had an assay and that I was amenable, obviously, to help anybody who required or wanted or would benefit from antibody testing.
We made that proposal and he was one of the first people to say, "Can you come and help" and we did.
**Q.** You had obviously tested a number of patients as we saw in The Lancet study, so whatever number it was, 180 or so haemophilia patients for the purposes of that study. In terms of post-The Lancet publication, was Dr Ludlam, as far as you can recall, the first Haemophilia Centre Director to ask you to test his patients or do you not



know?
**A.** At this distance, ma'am, I'm not sure. Certainly his was one of the more poignant requests for help.
**Q.** And do you have any knowledge of how those patients were selected? So you essentially received a number of samples and performed the tests on --
**A.** They would have -- we would have been told here is a selection of samples from haemophiliacs. Would you please tell us what the prevalent -- tell us the serological status of each individual.
**Q.** Do you have any recollection of your understanding at the time of why Dr Ludlam was approaching you in the autumn of '84?
**A.** I've thought about this considerably and the trouble is, when you start thinking about things from that distance, you're not quite sure whether you're following a pathway of novel thought or actually recording a memory. My feeling was that he would have wished to show how clean his population were from infection because it was all local Factor VIII. It wasn't imported from the USA. I think it was an aspiration to demonstrate cleanliness and in fact, as we know, the opposite was found.
**Q.** If we go to -- there's an extract from your evidence to the Lindsay Tribunal. It's at PRSE0001668. We can see it is entitled:



"Excerpt from the transcript of the evidence of Professor Tedder to the Lindsay Tribunal on 9 July 2001."
Then you were asked about the testing of the Edinburgh patients and your answer, seven or eight lines down from the top, is this -- I'm going to pick it up three lines into the paragraph that begins, "I think it was earlier than that." You say:
"I think it must have been in late autumn '84 when we did the first testing for him because it was -- it was certainly -- I will never forget. It was sitting in what used to be David Dane's room at the end of the corridor, looking out on an autumn sun which was a very hot sort of Indian evening, Indian summer evening, which should have been a lovely evening. It was about half past 7, 8 going through this litany of positive, positive, positive and Christopher Ludlam obviously getting more and more pensive and me feeling less and less kind as this evolution of damage done to a cohort evolved. That was the very early testing when he'd sent us cohorts of samples ..."
And then you say this:
"... which he already had a clinical suspicion that something had occurred and that was the beginning of the evolution of knowledge on the Edinburgh cohort."



What you said to the Lindsay Tribunal would appear to be slightly different from what you've just said to us in terms of your understanding of Dr Ludlam's thinking. Here it was a sense that he had some form of suspicion that something had gone wrong?
**A.** I think I was probably wrong. I think the balance of people's views is that it was a desire to demonstrate that something hadn't happened, rather than what has happened, and that that made it all the more devastating that it was an evolution of disaster unfolding rather than no disaster. And I will never forget that evening just talking with him and it was frightful, truly frightful.
**Q.** The Inquiry's heard evidence about a meeting that was then held in December 1984 at which information -- in Edinburgh in which information was provided to patients, including those who had been tested.
Did you have any involvement in that at all?
**A.** Not that I recall. I might have been asked as a virologist to give an opinion, I'm sure, but I do not recall specifically.
**Q.** Just, then, going to go back to some issues relating to the development of the test and, before we break for the end of the day, just look at a handful of documents from late 1984.

So, if we start with PRSE0003109.

Now, this is not a document that you would have seen at the time, Professor Tedder, because it is an internal Department of Health document. It is from Dr Alison Smithies, the date is 13 August 1984, and it is to Dr Harris at the Department of Health. We can see it is about setting up a working group to consider the introduction of the screening tests for HTLV-III antibodies.

I just wanted to pick up what Dr Smithies sets out in paragraph 2:

"You will be aware of the recent development by Dr Weiss and Tedder of a radioimmunoassay for HTLV-III antibody and the findings that the limited use of this test has revealed. It is proposed to extend the test to all blood donors at the North London Regional Transfusion Centre for a period of at least 3 months."

Just pausing there. Is that correct? Was that the intention, having done the testing on a 1000 there, that the plan was to then test all donors at North London?

**A.** I'm not sure what happened in the long run. Can you just rephrase your question again, because I was thinking about --

**Q.** Yes, of course. It is whether what Dr Smithies -- whether Dr Smithies' understanding as set out here was



correct at that time. Leave aside whether it happened or not, was it the case that the plan was to use the test, yours and Dr Weiss' test, for all blood donors at the North London centre for three months plus?

**A.** I'm not sure how much I would have supported that, because of the danger of drawing people in on the basis of one centre having testing therefore all centres having testing, that "We'll go get our AIDS tests at -- somewhere -- "at Chelmsford", and it wasn't North London.

I know there was an intention to run it at North London, and it would have had to have been run covertly for the danger of not wishing to draw people into the transfusion service to a centre where it wasn't being done. I'm not quite sure how to answer your question.

**SIR BRIAN LANGSTAFF:** I think it may help if you read on the next sentence.

**MS RICHARDS:** "As the donor population for North London RTC is drawn from an area where the incidence of AIDS patients and possibly contacts is currently the highest in the UK, it is hoped to extend the screening tests to at least two other RTCs. This, of course, depends on our ability to scale up production of reagents for the test, using either the virus isolate from Dr Gallo's



laboratory or a UK isolate yet to be achieved."

Then the suggestion is that that information will be used to base a policy about using the test more widely.

**SIR BRIAN LANGSTAFF:** It describes it as a screening test in three centres.

**MS RICHARDS:** Yes. Do you have any recollection of those plans?

**A.** Well, I know there was an intention to run at North London a period of testing there, and I think we could have -- they were conversant with using radioimmunoassay at the time because the hepatitis B surface antigen test was an RIA, so an RIA for a competitive RIA would have been doable. I think the issue is whether this would have been safe to do, testing in one centre without -- without making sure that the other centres were aware -- or unaware that this was being done, because of the danger of drawing people in who you -- with the best will in the world, you didn't want to attract people from the risk groups, on the basis of having a test, when they may not have a test in one centre but in another centre.

**Q.** Then the reference there to this "depending on the ability to scale up production of reagents for the test", in terms of scaling up, your statement tells us, and this is a general question now rather than this



specific point in time, that scaling up would require access to a sufficient volume of serum and a secure and steady supply of HIV culture supernatant?

**A.** Yes.

**Q.** And it is right to understand that it was the latter that was more problematic than the former?

**A.** We had a great deal of voluntary support from young men in the risk groups to provide their plasma for this purpose. It was quite a remarkable option to have this support from -- as I say, it would have been the beginning of the Terence Higgins Trust, before it was called such.

It would have been interesting to have to do this. It would have been requiring quite a lot of radioactivity handling, and that would have been containable. It was -- looking back on it now, I'm not sure what the reason for any delay in this would have been. It would have been the supply of the antigen. It would have been the radioimmunoassay, it could have been concern about not bringing people into the transfusion centre for testing until one had got coherent testing elsewhere. You know, this -- I have to apologise but, at this distance in time, I'm not sure which of those were the reasons for delaying.

**Q.** If we perhaps just look at one further document.

So if we move matters on to October 1984, to DHSC0002323_009.

This is another internal Department of Health document. It is from Dr Smithies again. It is dated 19 October and it is to Dr Alderslade, and we can see it is in response to a request from the Chief Medical Officer. Not a document you would have seen contemporaneously.

The answer to question A from Dr Smithies is this:

"Only pilot studies have so far been carried out on blood donors at the North London Transfusion Centre which have shown no evidence to antibody to HTLV-III in the 1,004 donors tested."

That is presumably a reference to the donors tested as part of the study reported in The Lancet, because the numbers match? Would that be right?

**A.** Yes.

**Q.** Then it says:

"The test is based on HTLV-III antigen acquired in the course of exchanges common to scientists between Dr Gallo and Professor Weiss. Professor Weiss and Dr Tedder, who together developed the test, did not feel that it was appropriate to greatly increase the amount of test reagent available without agreement from the US authorities to the increased use of the antigen.



The antigen has been licensed to five US pharmaceutical companies for development of tests and a vaccine. Dr Abrams wrote on 10 August to the Assistant Secretary for Health (in the US) requesting permission for the use of the US antigen in order to enable us to use the test more widely. We have had no reply."

Before we look at the next paragraph, I think you say in your statement you don't think this is quite right?

**A.** What I'm questioning is what the pressure was on to have more antigen for which testing and where. The difficulty -- and I think that the response from the American side was that it was given for research purposes only, not for quasi commercial use.

Now, whether you would consider expanding this seroprevalence study as quasi commercial use, I think the Americans would and we would not consider that a commercial exercise, it is just that we needed to have the data.

Does that answer your question?

**Q.** Yes. You have dealt with this in more detail in your statement, we don't need to put it up on screen, but you comment on this in paragraph 186 of your statement.

Then the second paragraph says this:

"In the meantime the production of test reagent has



been held up by the requirements of HSE [that's Health and Safety Executive] and the activity of trade unionists. The laboratory at the Middlesex Hospital is expected to be finally commissioned by the end of this week. It is hoped that sufficient reagent will then be mad available by the beginning of November to test all the blood donors at the North London Transfusion Centre."

Then we have in brackets the reference to not wanting to make that too public, for the reason you have already outlined.

Do you have any recollection now of what was being said here about the HSE and tradition unionists?

**A.** Yes, I do. It was an entirely avoidable phenomenon. What had happened was the exhaust from the testing and from virus washing goes through dry-out through pump and put into a discard pot, and the discard pot has a vent, and the vent was meant to be connected to a pipe which sat inside a class 1 cabinet. For reasons beyond my knowledge now, the safety pot was not put in the class 1 cabinet but was left outside it, and the Health and Safety Executive, understandably, took extreme umbrage to this and declared it as a major exercise, a major contamination, "Everybody has been infected".



We eventually tested everybody and nobody had been infected, but it was a salutary exercise of just how careful you have to be to maintain people's aspirations of safety. No harm was done because the safety pot was there, but nevertheless, because it wasn't in the right place, we had a -- I don't know what you would call it -- a category 3 style investigation and slapped wrists all round. It was an unpleasant exercise.

**MS RICHARDS:** Sir, I think that's probably the right time to pause because I have got a number of further documents to explore with Professor Tedder, just chronologically taking the story through to '85, but we can do that in the morning.

**SIR BRIAN LANGSTAFF:** Yes, it is.

The one question which arises out of this, there may be an unanswered question in some people's minds as to the activity of trade unionists. Is that the trade unions being concerned about the safety risk posed by the safety bottle, so associated with HSE, or is it something different?

**A.** It was the health and safety representatives in the department, who were also trade unionists, but I think that was coincidental, that they were people who were particularly worried about working conditions and colleagues and the safe, and therefore that incorporated

safety. So it is no criticism of trade unionists, it was actually, I think, an overreaction, but understandable, by the Health and Safety Executives, who happened to be trade unionists. But that was coincidental.

**SIR BRIAN LANGSTAFF:** And what excited this activity was -- is it the same incident you have just described?

**A.** Sorry?

**SIR BRIAN LANGSTAFF:** What gave rise to this activity, this concern, was the same incident that --

**A.** It was recognising the safety bottle was not in the cabinet, it was outside the cabinet. The fact that it was collecting anything, and there wasn't anything to collect anyway, was nevertheless slapped wrist and justifiable anxiety. And I learnt if you are going to put a bottle in the safety cabinet, you put it in the safety cabinet.

You know, I don't criticise. It was an error which was made but was not an accident.

**SIR BRIAN LANGSTAFF:** Thank you. On that note, I think we will end for the day and come back at 10.00 am tomorrow, if you would. 10.00 am.

**MS RICHARDS:** Thank you, sir.

**(4.04 pm)**

**(Adjourned until 10.00 am on Friday, 14 October 2022)**



**I N D E X**