Wednesday, 16 March 2011

(9.30 am)

THE CHAIRMAN: Good morning.

MS DUNLOP: Yes, sir. As indicated yesterday, today we are going to try to get some sort of sense of the size of the problems. We are going to hear from two witnesses, Dr Gillon and Professor Goldberg, in that order.

Our first witness for today is Dr Gillon.

DR JOHN GILLON (affirmed)

MS DUNLOP: Good morning, Dr Gillon.

A. Good morning.

Q. You are John and Jack. Is that right?

A. Yes, that's right.

Q. What do they call you at work?

A. They call me Jack.

Q. So you are Dr Jack Gillon. On your CV you are Dr John Gillon?

A. Indeed.

Q. We are going to start by looking at that. It is WIT0030281.

The first page is giving your basic biographical details and we can see that you studied medicine at Edinburgh University and you have a number of postgraduate qualifications, including an MD and you are a fellow of the Royal College of Physicians. What did

you do your MD on?

A. The MD was on the intestinal immune response to a gastrointestinal parasite called giardia lamblia. That was because I was training in gastroenterology at the Western General Hospital under Dr, later Professor Anne Ferguson, who was a world expert on GI immunology.

Q. I think she has given her name to part of the Western, hasn't she?

A. She has indeed. The Anne Ferguson building.

Q. Can we look at the next page, please. We see that you worked under Professor Girdwood at Edinburgh Royal Infirmary. He is a name we have come across in the field of blood transfusion?

A. Yes, indeed.

Q. Who was he exactly?

A. He was professor of therapeutics and had a big interest in the use of blood products, among many other things, and was a member of the committee on the safety of medicines and so on. I went to his unit as a general medicine senior registrar. So while I was a lecturer in gastroenterology at the Western, I also, as we all did, to become internal physicians, had to do general medicine as part of that. So I was looking after the generality of patients on that ward at that time.

Professor Girdwood, just to add to that, was for many years the chairman of the Blood Donor Association. So he had great interest in blood donors as well as in transfusion as a clinical topic.

Q. The Blood Donor Association was a charitable organisation?

A. Yes, in a sense I think it goes back to the earliest days of the Scottish Transfusion Service, around the time of the Second World War when it was founded by John Copland. I think it was continuous from that time, and still exists.

Q. Right. We see also that you became a senior registrar in Edinburgh and Southeast Scotland Blood Transfusion Service in September 1984. There are basically five regional sections of the Blood Transfusion Service in Scotland. Is that correct?

A. That's correct, yes.

Q. This is obviously one of them. Then from April 1985, until now indeed, you are a consultant physician in the Edinburgh and Southeast Scotland Blood Transfusion Service and the department of transfusion medicine. Where is the department of transfusion medicine based then?

A. Well, basically that is the name that we use for the clinical side of blood transfusion. The term

"transfusion medicine" was one that was coined many years ago. We have always been keen on that term, because we are very keen to be involved in the clinical care of the patients who are receiving transfusions, and have been actively in Scotland since that time. Since before the time that I first went there.

Q. On page 3 we can see that your current areas of responsibility look to be really 1 and 4. One, you have consultant responsibility for medical care and selection of donors and you are also responsible for the autologous transfusion service. What is autologous transfusion?

A. It is actually now defunct to a large extent, in one of the senses in which I was involved. Autologous transfusion refers to a patient donating his or her own blood for use in a planned surgical intervention. That's one form of autologous transfusion. That is one that we set up and was the first in the United Kingdom in 1987. It was popularised in the United States in the years preceding that because of the fear of HIV. There was some demand for it in this country and we felt we should be involved in that and worked with the clinicians to establish a service in the southeast of Scotland, which later was rolled out to the rest of the country. But it never really lived up to its promise.

It was something that was useful for some patients but didn't always succeed in avoiding transfusion in the sense that there is a limit to how much blood you can give in the run-up to an operation and if you need blood you are likely to need quite a lot.

So worldwide it went out of fashion in the mid 1990s and by early 2001/2002 we had pretty well ceased that completely. Now it is still theoretically available in the Glasgow area but it is very rarely used except for very specific indications.

Q. Like many doctors, we see you have a number of administrative roles, responsibilities and you have listed those under the heading "administrative and management contributions". Then on the following page you have listed educational and other academic activities and we can see that these have extended beyond the United Kingdom and you have had some involvement in a WHO project as well in the West Bank. What was involved there?

A. That was quite a short course -- I can't remember exactly how long we went for, a week or ten days I think -- to deliver to the hospitals in the West Bank. One was in Bethlehem. I think the other one was in Jerusalem itself. Yes, there were two hospitals. We visited one or two others to do a series of lectures and

seminars on transfusion for the doctors and nurses working there.

Q. This is the first item on the list: you have also spent three months in a community blood bank in the United States, which has presumably given you first-hand experience of the very different way in which blood collection is organised there?

A. Curiously enough, it wasn't all that different. I think the misconception still existed at that time -- probably in some quarters it still does -- that all blood in America is paid for by paid donors, but even then, and in fact for about ten years, since Harvey Alter and his colleagues showed that paid donors were much more likely to carry infectious diseases, transmissible viruses, the whole blood donation in the United States was almost entirely volunteered by that stage.

In fact the structure of the blood bank and how it delivered blood to the hospitals and how it interacted with transfusion services in the hospital was remarkably similar, but of course that was not uniform across the United States and I did travel to various other types of centre to see how they did things. It was very interesting and obviously a fairly crucial time in the history of transfusion here.

Q. I suppose the clue is in the name "community blood

bank"?

A. Indeed, and you know, the blood banks in America and the blood collection centres had various different organisations. Most of it was Red Cross but this in fact wasn't a Red Cross blood centre. There was a council of community blood banks, which was another separate organisation from the Red Cross.

Q. Lastly, you have a very lengthy list of publications, either in your own name or to which you have contributed. I certainly notice some on Hepatitis C, if we look for example, at 76, which is page 10.

You contributed to a workshop on Hepatitis C virus in 1995, and what's that, a paper? "Epidemiology of Hepatitis "C?

A. Yes, that was a paper I gave describing our experience and what we found in blood donors once we started testing for Hepatitis C. It largely was similar to work that was published, I think, the year before that, in fact, under the first author of Crawford, which really described the risk factors and other features of blood donors who were found to be positive for Hepatitis C.

Q. Number 68. I think we are actually going to look at that, where it crops up in the preliminary report as well, but that's on the page before, the last reference there. Is that the one you are referring to?

A. Yes, indeed.

Q. Then I also noticed, I suppose because it has a slightly catchy title, number 92. You have written something called "Look-back on HCV look-back" in 1999.

A. Yes, Transfusion Today -- this was not a peer-reviewed journal by any means, I think it is the journal of the International Blood Transfusion Society and it was a fairly informal sort of journalistic look at the whole HCV look-back process and some of the issues that surrounded that.

Q. Thank you, Dr Gillon. I think now we need to look at your statements. You have provided two statements to the Inquiry, both to assist us in our task of trying to get some numbers together, to get some sort of understanding of how many people may have acquired infection through transfusion properly so-called, or through blood products.

In broad terms, is it fair to say that one can either go about the task by trying to count the numbers of people who have been identified and doing that by asking people who may have been in a position to identify those with Hepatitis C, acquired by transfusion, or one can start with known prevalence of Hepatitis C, say among blood donors, and try to do various, more statistical exercises, modelling exercise

to try to work out how many people are likely to have become infected?

A. Yes, and both are fraught with difficulties, slightly different problems in either approach. What I have been doing in these papers is describing the numbers of people who have been reported to us as individuals, as patients -- well, some of them as donors, presenting to us as donors who had a history of transfusion -- and trying to establish the truth or otherwise of that mode of transmission.

So these are really quite restricted numbers. They depend to a large extent on clinicians, who have identified a patient with Hepatitis C thinking about the possibility of transfusion. The patient may mention it spontaneously but may not. Then doing a further leap of logic of saying, "Well, let's see if the Blood Transfusion Service can either verify that or tell us whether or not it is a real issue".

The other approach is simply to collect data, as HPS does, and of course it is not simple. Again it depends on having a reporting system. The advantage they have is that they can put in place a systematic reporting system. The first such I think was -- and it is described in Professor Goldberg's statement and I'm sure he will be talking about it later -- when HIV testing

was on the horizon, they put in place, through the virus, virology laboratories throughout Scotland, a single unified referral form for the clinicians to use to request the test and HPS -- or SCIEH as it was then called -- automatically received a copy of the referral forms for every positive. So they got to know about every positive HIV identified in Scotland at that time.

I am sure Professor Goldberg can elaborate on that, but you can see that that is a much more secure way of getting some feel for the numbers of people out there with a positive test result.

We depend much more on a logical deduction from a clinician. Perhaps it is something that needs to be explored further with the transfusion service.

Q. There were two points really that arose from that, Dr Gillon. The first was, and I think you have really dealt with this, but as far as the epidemiological modelling and the use of statistics is concerned, we should really ask Professor Goldberg more about that, I think?

A. I think that would probably be appropriate. There are a lot of assumptions in any modelling to try and work backwards from those raw numbers that they get and the information that comes with it. Because they do get some information about the patient's risk factors, the

age, the sex and so on, and geographical locations.

So they have a certain amount of good hard information to go on but to transform that into real numbers in the population is really very difficult. In fact, for any of these viruses it's difficult to get good data on prevalence in the population. I'm sure that is going to be an issue that we will explore.

Q. The second point I was just going to confirm with you was that there isn't any obligation on a clinician to report to you as a blood transfusion service that they have come across a patient who may have acquired Hepatitis C via transfusion. Is that correct?

A. Historically there was no sort of legal obligation, if you like. It was certainly a recommendation in, for instance, I think, publications like the notes for transfusion that, you know, clinicians should report but not many surgeons would have read that, I wouldn't have imagined.

So it has always been informal until fairly recently, and I forgot to mention this in my statement, I think, that that position changed drastically in 2005 with the European Directive On Blood Safety, which was transmitted into UK criminal law, as the Blood Safety and Quality Regulations 2005.

One of the main focuses of that had come out,

I think, largely from what we knew about these transfusion transmissible viruses and the issue of look-back and the difficulty in tracing patients who had had transfusions many years previously and differing standards in, for instance, peripheral hospital blood banks and in transfusion centres and so on.

In 2005 BSQR, the safety and quality regulations, stipulated that for the first time the hospital blood banks had to come into the MHRA regulatory framework -- that's Medicines and Healthcare Regulatory Authority -- whose role was beefed up. They had to take on board inspecting hospital blood banks as well as transfusion centres, which had been inspected through the medicines inspectorate system for many, many years. Largely that was because of this gap in what became known as the traceability of previous donations and therefore the patients who received them.

So traceability became entrenched in that and an obligation on clinicians, blood banks and transfusion service, obviously, to report very formally to MHRA when we had a serious adverse event of transfusion, which obviously these cases would be defined as.

Q. If we have in front of us [PEN0010043], we can see, I think, from this, as we work through it, an illustration of the type of exercise I was describing

earlier about counting the number of people you know about. Indeed, you have described that for us in this statement, grouping people into three categories. Just looking in your preamble, you say that some of the information you have included in a paper on look-back, and you have also obtained information from the national Hepatitis C register, which is at Colindale. That's based in England. That's the Communicable Diseases Surveillance Centre; is that correct?

A. That's right. Also known as the Health Protection Agency.

Q. So HPA is in England and HPS is in Scotland?

A. Yes, that's right.

Q. Yes. That HPA, there are data on 103 patients identified through the targeted look-back procedure required by the Department of Health in 1995. Can I just ask you to take us through 1.1. This is people --

THE CHAIRMAN: Sorry, before we go there, could I ask about the Colindale data.

Is it dependent upon reporting by clinicians as your data was?

A. No, this grew out of the formal look-back which is described in 1.1, when, in the run-up to that, it was really our colleagues in the Blood Transfusion Service

in England and Wales who had the idea of trying to establish a cohort of these patients who would be followed prospectively and anonymously to get information about the natural history of the disease.

The great advantage of these patients for this being that the exact date of the transfusion was known, so that the length of infection was clear.

THE CHAIRMAN: At the moment my interest is in knowing whether the data is homogeneous or whether you are now putting together our data from the Colindale type look-back exercise and data of the kind you have described as collected by your department, which depended upon a clinician finding a significant interest in hepatitis and reporting it. Is the data homogeneous --

A. It is homogeneous and all derived north and south of the border from patients identified through look-back which starts from a donor with a positive HCV test.

THE CHAIRMAN: Thank you.

MS DUNLOP: Dr Gillon, the first of your groups of people are those who have come to the attention of the Blood Transfusion Service because they are themselves donors and it has turned out, since the introduction of screening in 1991, that they have been identified as Hepatitis C positive. So that's, if you like, the first

way in which a group of people have come to your attention.

A. Yes.

Q. The number of that group of people -- and you have told us this -- is 59?

A. Yes.

Q. Is that right? You say that people who have been identified in that way have themselves been investigated to try to establish whether a source donor could be identified, but in only nine of the 59 cases was a date of transfusion given. So that looks like that wasn't a particularly fruitful exercise. Is that correct?

A. I think that probably gives too negative a view of it. What I have presented here was what was reported on our routine epidemiology reporting forms back to Dr Brian Dow, who was responsible for liaison with HPS and for collecting the data for SNBTS. It is quite likely that more of those 59 cases in fact, on further investigation, were shown to have been transfused and may in fact have sparked a second round of look-back as a result of finding the donor who was responsible for the donation which led to them being infected with Hepatitis C. So these are just the raw data that were given to Brian Dow quite early on in the process of sorting this out.

Because of the numbers and the shortage of time, we simply haven't been able to go back to all of those 59 cases to examine them individually and bring that information to the Inquiry.

Q. I take it that what's reflected by this number, 59 people, is the fact that people who themselves receive a blood transfusion sometimes are motivated to go on and become blood donors. Is that correct?

A. Indeed, it is a very powerful motivation and we don't have very good data for the prevalence in the general population of a past history of blood transfusion but, as you can see, the overall 6.8 per cent of these donors is a reasonable percentage. It is very powerful as transfusion in a close relative is also a very powerful motivating factor.

Q. In a close relative, did you say?

A. Yes.

Q. This is really, sir, an issue which has come to the attention of the Inquiry and we are hoping that Dr Gillon will be able to cover it a little more fully when he returns next week because I appreciate it certainly arose in connection with one of the cases we looked at last week.

The second of your three groups of people -- and this is on the next page, if we look at 1.2 -- is

a group of people who were identified through the targeted look-back procedure required by the Department of Health in 1995. That number is 133 people.

I wanted to digress slightly at this point and ask you about look-back exercises in general, in the first place. I gather that "look-back" is a term that was really coined in connection with AIDS. Is that accurate?

A. Yes, people had been doing look-back without calling it that, for quite some time. We know that, for instance, when the test for Hepatitis B was introduced in 1970 and they started looking at it in the West of Scotland, they made a recommendation, the very first paper, that there should be an attempt to trace previous recipients of anybody who was found to be positive.

So the procedure was already there and I'm not sure to what extent it had been pursued in the case of Hepatitis B. But it was first described as "look-back", as far as I can find, in a paper in 1984 in the United States, which was in relation to HIV.

Q. Yes. There are, as I understand it, certain difficulties with any look-back exercise. I wonder if I could try to run some of them past you and obtain your comments, Dr Gillon.

First, potential difficulty is how long the

infectious agent has been present before a test becomes available. I think it is probably reasonably self-explanatory why that is difficult, why that can be difficult but perhaps you can give us that in your words?

A. I can. The contrast between Hepatitis C and HIV is revelatory here because when we started testing for HIV in 1985, the disease, the virus had only been present in the general population, and therefore the donor population, for two or three years really, perhaps slightly longer than that in the United States. Therefore, when the test was introduced, the numbers of previous donations from any individual donor that were dangerous were limited to that two to three-year period.

So the donations prior to the virus arriving in the population weren't significant in that sense, whereas in the case of Hepatitis C, we knew then and we know now that it had been present for many years before a test came along, which meant that some donors who had been donating for a long period had in fact been carrying the virus and were therefore capable of transmitting it. So the impact of that on a look-back exercise means that it is hugely more complicated and more difficult and vastly more patients potentially are affected.

Q. I suppose, if one were doing look-back in general on the

whole of Hepatitis C, you would find secondary and even tertiary levels of infection. If you imagine someone who perhaps acquired it from a blood transfusion and then, as we have said, passes it on through themselves becoming a blood donor; it is almost like a kind of family tree, is it?

A. Yes, indeed. There are various methods of spread. They could become an organ donor for instance, or there could be secondary sexual transmission or whatever. So there are ramifications, yes.

THE CHAIRMAN: I wonder if I could ask a question at this stage. Doctor, I'm interested in two aspects, I think, of this matter. One is the change over time in the means of transmission, because I assume that people's habits change and the behaviour that can lead to spread will not be the same over history. And perhaps you will see the other aspect is whether one can, in any way, limit retrospectively the point in time at which what we now know as HCV entered the world?

I have seen reference to HCV not being known before a certain period. Does that bear on where it may have come from in time? Has it always been here?

A. It is certainly a very ancient virus, an ancient group of viruses. In broad terms, we think of it as having been an issue since the Second World War but that's

partly, I think, because transfusion has really only existed since the Second World War.

Professor Peter Simmonds, who worked with us on Hepatitis C and is now a world expert on Hepatitis C as a virological problem -- he is an expert in the genetics of Hepatitis C or the virus itself -- they have developed methods of looking at the subtypes of virus and how they have diverged and effectively constructing an evolutionary tree for the virus. I think it is thought to be quite an ancient virus that has been around for a long time.

Having said that, the impact on the human population is fairly recent, I think, and the data that we have -- and again Professor Goldberg might be the best person to speak to this -- studies from America and modelling studies from France, some data from the HPA, suggest that the incidence -- in Western countries, certainly -- ramped up through the 70s and 80s and peaked around about the mid 1980s. Largely that was to do with intravenous drug use.

THE CHAIRMAN: Thank you very much.

That was the sort of distinction that I was interested in. We have something of indeterminate age that affects the population from time to time in ways that will vary according to factors such as the

introduction of intravenous drug abuse or perhaps other forms of conduct, and certainly because of the way the transfusion services themselves may have operated from time to time.

A. Yes.

THE CHAIRMAN: Thank you.

MS DUNLOP: Doctor, I was asking you about some of the factors that can make look-back either more straightforward or more difficult, and another one I was going to put to you was the virulence of the agent.

A. Yes. I think the truth is that if you have a very virulent agent, by the time you do a look-back, if a long time has elapsed before you manage to get a test to identify the people who had been carrying it, if it is a very virulent agent, a large proportion of them may be dead as a result of that.

We find in any look-back, depending on the length of time before you start doing it, that, of course, a significant proportion of patients will have died from the presenting disease or indeed from some other disease.

Q. Yes. Perhaps the point you are making is summed up by the propositions that I have had from you before today, that the number of patients traced and found to be alive in a look-back exercise is inversely proportional to the

time elapsed between transfusion and the discovery of the patient via the look-back, which may sound a bit indigestible but I think is probably common sense?

THE CHAIRMAN: Perhaps if we take it again --

MS DUNLOP: I think Dr Gillon can express it more simply today; I think he already has.

A. Well, I think the simple fact is that the longer time that has elapsed between the putative transmission event and the point at which that is identified, the more likely it is that that person will have died, probably of some other cause but possibly of the infection itself in the meantime.

Q. Yes.

THE CHAIRMAN: Only survivors can be candidates for examination.

A. Indeed.

MS DUNLOP: And the number of those candidates that one will be able to find is inversely proportional to the time which has gone past or the time which has elapsed between the transfusion event and the person being identified.

Another difficulty, I think, with look-back exercises and one which I think may be present here, is that records from the early 1980s -- that is blood transfusion records, bloods bank records in hospitals

and so on -- and before tend to be in paper form. Is that a problem?

A. I think the past tense is more a problem. They tended to be in paper form and many of them were lost or destroyed or discarded and it also depended on how paper records were kept, how easy it was to relate back to an event that had happened a long time ago.

For instance, let's say in Edinburgh, Edinburgh Royal Infirmary or whatever, in 1979 a patient had been transfused. We didn't get to know about it until the 1990s. We would be entirely dependent, first of all on the hospital records still being in existence. That would be necessary to give us the unique donation numbers that could link that unit of blood to a given donor; but secondly, also to our own record-keeping system. Even with a donation number, at that distance in time it would be virtually impossible for us to link the donation number to the donor because it was a simple card system that we had for the donors in the 1970s, which were not stored alphabetically, were not searchable in the modern sense of, you know, putting a donation number into a computer and linking it to the donor who gave that. Different transfusion centres had different systems for how they kept the donor records, whether it was a card system or -- well, they were all

by definition card systems in those days.

Q. This is really the pre-computer era, you are describing?

A. It is the pre-computer era. The first computerisation was in 1983, which was one of the first in the world, if not the first in the world to link that specific donation number to the identity of a donor and to the identity of the patient who got it. Since then we have been able to do that.

THE CHAIRMAN: Doctor, I think I have seen reference to a system operating in the Glasgow and South Western area very early on that used computer tape, what was then called computer tape. It was really a form of machine accounting. Do you remember that?

A. I do.

THE CHAIRMAN: How long back did that go?

A. I am afraid I can't answer that but that was largely a laboratory-based method. So that was about the donation and the tests on the donation. What was different in what we developed in Edinburgh in 1983 was the link to the patient.

THE CHAIRMAN: I see. Since I remember these systems operating in the late 1950s, when I was an auditor assistant in a firm of chartered accountants, they go back an awful lot longer than the period you are talking about.

Ms Dunlop, I wanted to interrupt. There is quite a bit of interference coming through from time to time which must be due either, I'm told, to someone having left their mobile phone on for text messages and emails coming into laptops. If it is the latter, I don't know that there is anything that can be done about it but I would like people to make sure that they don't have their phones on. It is quite annoying getting the feedback and if anyone can switch off their inbox, I don't know whether they can, that's going to help too.

So ladies and gentlemen, if you would just make sure you are not the cause of the interference, I would be obliged.

MS DUNLOP: I think, Dr Gillon, we can imagine the problem you are describing because we have seen, even from the 1970s, that people's medical records may very faithfully document a batch number of a pack of blood that was transfused to them, but the question of whether one can do what is really the reverse exercise, and go to some sort of record, see that batch number or that pack number and work out where it went, is really the problem that you are describing, I think, is it?

A. Well, it can go either way. It can be difficult in either direction.

Q. So it can be very difficult to discover to whom

a component was transfused and then I expect there are the additional human difficulties when one tries to track patients, that people move and become lost or even emigrate -- women marry, change their names, all of these -- which make people difficult to trace.

A. That's correct, we could have quite a clear track back to identify an individual patient but then find it quite impossible to trace where that patient now was or who -- you know, people move, they may not register with a new GP.

One of the ways we used to track patients is through the GP tracking system, which in those days wasn't particularly good. Nowadays, it is really relatively easy to find people almost anywhere in the UK quite quickly, provided they have registered with a GP.

Q. I wanted to ask you still in connection with the notion of look-back, if you could look at another document, [PEN0020803]. This is a report to the health committee in the Scottish Parliament and the interesting feature of it is that it contains within it a little more information about the numbers traced by the look-back exercise.

We need to look at page 2. This is a report for which the then health secretary, Andy Kerr, was, I think, responsible. It was submitted to the health

committee on 31 January 2006. If we look at page 2, you see there that the results of the look-back for Scotland have been tabulated. I think the number we need to look at to link into your statement is the 133, which we see a little more than half way down. If we look at the table, it is basically a sequential progression through the different steps; is that right, doctor?

A. Yes, that's correct.

Q. Once screening of donated blood comes in in 1991, it is possible to identify blood donors using that test and if they have given before 1991, you can work out how many donations they have contributed. That would be the 1,658 number?

A. Yes.

Q. Then that 1,658 has become 2,026 components. What do we mean by "components"?

A. That refers to the process whereby a whole blood donation is separated by centrifugation into its cellular components, which are red cells primarily. That's what we think of as a blood transfusion, red cells, and tiny cells called platelets which form a separate layer in centrifugation. They can be separated out and used as a concentrate for patients at risk of bleeding because of a low platelet count.

These are cells which are crucial in the coagulation

process and the supernatant, the clear fluid, which is the plasma, which until 1998 was used for fractionation into further blood products such as Factor IX, immunoglobulins and so on, or can be used as fresh frozen plasma and given to an individual patient.

Q. Yes. But when you are talking about the number of components, the 2026, which breaks down into 1,356, which were traced in the 60s and 70s, which were not traced, that presumably doesn't include the blood product concentrates made for people with haemophilia?

A. No, that's correct. When we talk about components, we are usually referring to the fresh blood components, which would be red cells, platelet concentrate and fresh frozen plasma.

Q. Then we go further down the sequence of events. Of the 1,356 components that were traced, it was possible to identify 880 recipients, and the number of people potentially eligible for counselling and testing perhaps at first sight looks a small fraction but there is some explanation of that, if we look in the box below, where 536 people are described as being deceased?

A. That's right, it is slightly out of sequence there, yes.

Q. So the point that his Lordship made earlier, that obviously, to be eligible for counselling and testing, you have to be alive.

A. Yes.
Q. It is true, is it, doctor, that a number of those who receive a blood transfusion don't survive more than, what, one or two years after the transfusion?
A. Yes, there are various studies from various parts of the world, and I think it has improved with time in the sense that the medical care for the primary condition that the patient has has improved, but when this started to be looked at in the late 80s and early 90s, the survival of patients who had had a transfusion was really surprisingly poor. That was not to do with the transfusion or viruses but with the severity of the illness that led to the transfusion. Within a few years you could expect to find 50 per cent of people had died.
Q. Out of the 266 people, exactly half in fact had been counselled and tested positive. 133. Then 70 of them had been counselled and had tested negative. The other group of 63 people, there are some different explanations for that group: people who didn't want to be tested or where the results for whatever reason haven't made their way back to the Blood Transfusion Service, or a group of people described as not appropriate for testing. What might that cover?
A. That would be a small group of patients usually very elderly, maybe in a care home, perhaps with dementia,

with a low life expectancy who were not able to consent appropriately to being tested. It was usually the GP's decision that they didn't want to inform the patient.

Q. Finally, there were 78 recipients who were just not traceable and that perhaps reflects the points we made earlier about the human factors.

A. Yes.

Q. People moving away or moving abroad even, and other circumstances like that. So that's where the second of your three numbers comes from, the 133 people who were identified through the look-back. I wanted actually just before we leave the topic of look-back, to look at what is said in the preliminary report about this. If I could ask that Dr Gillon has in front of him page 321 of the preliminary report, please. It is chapter 9 and it is paragraph 9.295.

It's probably slightly slower, sir, because each page doesn't have its own identifier. We have chapter 9. It is a long chapter, I am afraid. It does have its own number. I just didn't know it, sorry.

Mr Mackenzie's suggestion is that you should go to the back because it is quite near the end of the chapter. So that might be quicker. Here we are, 321, thank you.

Here you are, Dr Gillon, in the preliminary report in paragraph 9.295, and this is actually a description

of an earlier, I suppose, look-back, that you had done within Scotland. Is that right?

A. That's correct. We started doing look-back as soon as we started Hepatitis C testing. In fact this became our contribution to the look-back. So our patients identified through that are part of that 133.

Q. Right. That, I think, we can see from the footnote, which is 344, I think. It is actually from the previous paragraph but the footnote, 344, is the reference to the paper that we found in your CV. I think it was number 68. Is that right?

A. Yes.

Q. Crawford and others?

A. Yes. That doesn't refer to the look-back as such. That's just the characteristics of the donors. The look-back is described in a following reference, Ayob et al.

Q. Yes, thank you. I did want to look also at the extract from your paper, the Ayob report, which is quoted on the following page, 322. We find there, Dr Gillon, really a summation of the views of the authors as to the, I suppose, ethical reasons for undertaking the exercise. Is that what's being described?

A. Yes. I felt very strongly that the look-back was ethically -- well, more than desirable. I felt it was

a responsibility that we should take on board.

Q. You felt an overwhelming responsibility to the individual patient?

A. Yes.

Q. Thank you. The third and final group in your group of three different categories of individuals is back to your report and back to your statement at PEN0010044. This is the group of people who have been reported to SNBTS by clinicians. So for the most part hepatologists, gastroenterologists who have come across a patient who has Hepatitis C and they think that the likely mode of infection has been transfusion. Is that right?

A. That's correct, yes.

Q. You give us a breakdown for the five Scottish areas of that group of patients who total 28.

Then you provide a slight reservation. You say:

"It is not always possible to establish the diagnosis of transfusion transmitted Hepatitis C with certainty as frozen sample archives mostly date back only as far as 1986 and it is often not possible to trace the implicated donors for testing."

That's frozen sample archives of blood?

A. Well, of plasma really, and in some cases serum, taken from the original donation and frozen at the time.

Q. Right. Plainly, if a person is mentioned as a likely case of transfusion transmitted infection and you can go to a sample and test it and find that the sample did contain antibodies to the virus, then you would, as it were, have found the reason for the infection?

A. Yes.

Q. But where that's not possible, you draw conclusions, as you say, from circumstantial evidence. You say that can be because no other risk factor was declared. I think you are saying that this figure reflects the fact that some people have not been included, if there was, I suppose, no evidence. You instance documentary evidence of transfusion, absence of blood bank or donor records. Would it be enough if you had one of these? Would it be enough support for transfusion as the mode of infection if you had documentary evidence of transfusion, say?

A. We haven't really tried to define this. I should say that this statistical exercise is entirely retrospective and generated purely to answer the question from the Inquiry. In other words, these were individual records of individual investigations and we have tried to find out such records in the five Scottish centres that still exist.

Since 1998, which coincides with the so-called

completion of the look-back -- and obviously we still look back if we find any reason to do so -- for the statistical purpose, the counting of the look-back ceased in 1998. But that coincided with the UK transfusion services setting up an informal system of reporting adverse events, called the SHOT system, Serious Hazards of Transfusion. That was a voluntary service to which we contributed and since then Dr Brian Dow has collated all look-back cases and reports from Scotland to forward them on to the SHOT system and we therefore do have a database of this information from 1998. Prior to 1998 we are dependent on finding individual case records in the transfusion centres and that's an amalgam. The pre-1998 stuff and the post-1998 stuff is included in that 28. But including cases, particularly these older ones, but some of the more recent ones as well, the information is open to interpretation. It can be difficult to know whether to say, "Well, we accept this as a case of transfusion transmission. This one probably is but we really don't have enough documentary evidence to say with certainty".

On a number of occasions we can rule it out. We can say the blood that this person received was tested, all the donations were negative, all of the donors have come back and tested negative subsequently. We can be

confident that transfusion did not transmit that infection. In fact, what the data post-1998 demonstrates very nicely is that the majority of reports that we received turned out not to be transfusion transmitted infections. I have looked at Brian Dow's data and in fact, since 1998 we have had 58 cases reported to us. 11 of them have resulted in a concrete diagnosis of transfusion transmission. In other words, we have identified a donor who was positive.

Sorry, I think an extra 12 come into this interpretable category of: we know they have had a transfusion but we can't identify a donor, either because there is no archive samples or there is a piece of information missing somewhere but we are confident the patient does have another risk factor and it looks as if that might have been a transfusion transmission.

Interestingly, the case that illustrates that very well is Mrs GRO-A because we do not have confirmation that that's a transfusion transmission. So we can never be 100 per cent certain.

Then there is the other category of patients, the majority, where we can rule it out. So out of 58 we had 23, I think, that we would accept either categorically transfusion transmission or possible transfusion transmission. I think Mrs GRO-A would fit into that

second category.

There are some cases where you can feel that the information that we have is a bit skimpy and therefore you would hesitate to say that this is likely to be a transfusion transmission. Some, like Mrs GRO-A, you would be fairly confident to say, "This is probably a transfusion transmission". But we know there are other ways of picking up transmissable viruses in hospital environments, as we will see in some of the data from the renal units, for instance. Therefore, unless we can identify a donor and establish that link with certainty, there is always a bit of interpretation that's necessary here.

As I think I have said, I have tried to be inclusive here. In other words, not to wish to minimise the figures in any way, but there is this caveat that, we can never be certain unless we make the link.

Q. Dr Gillon, you were asked some further queries by the Inquiry team after you produced this statement and you provided a further written response.

THE CHAIRMAN: Ms Dunlop, before you go on, could I go back to the first page of the document that's on the screen, please, just for a moment?

I would just like to understand the figure of 867 and the 59 a little bit better, Dr Gillon. You say that

the number of patients treated and known to have contracted Hepatitis C includes, 867 blood donors that were HCV positive to the end of 2009. That's a fairly precise figure, 867. That's just the accumulation of data from records, is it?

A. Yes, these are realtime accumulating data on the numbers of blood donors identified through our routine testing system to have antibodies to Hepatitis C.

THE CHAIRMAN: Those 6.8 per cent, 59 people, have been identified by what one might now call a conventional testing mechanism.

A. No. Those are people who on counselling, usually by a transfusion service doctor but sometimes by their GP or the clinician who was responsible for the initial transfusion, have said that they think they got it from blood transfusion.

THE CHAIRMAN: I see. So there is not an objective verification of that. It depends upon the reporting by the individual?

A. Yes, entirely.

THE CHAIRMAN: I see. If we go back to the following page, please, just to get a clearer picture of the nature of the data. The second class, I think you have identified fairly clearly as the people who are identified positively by the look-back.

A. Yes.
Q. The third class of people who have been fully examined, fully followed up, as it were, and overall we have got a number that comes to 220 people.
A. Yes.
THE CHAIRMAN: Would it be right to look on the candidates -- just among those numbers, and forgetting the background -- as really coming to about 1,000? The 867 plus the other two categories. Or is the class much wider than that?
A. No, the 867 are the seropositive blood donors identified on routine screening. If you take 59 away from that, the rest are the other blood donors who had a risk factor such as drug use or some other means of transmission. A substantial proportion, 20 to 30 per cent -- and every transfusion finds this -- report no risk activity whatever.
THE CHAIRMAN: But again, that depends on the subjective material provided by the individual being studied?
Yes. Is there any sense of underreporting by people who are asked questions or is it just a case that the individual may not know?
A. There is a sense of underreporting. Certainly in my own experience, I have encountered donors who on meeting them for the second or third time -- and we sometimes

follow them up, depending on how much support they need -- will say, "Well, I didn't tell you this the first time I saw you but I used drugs once at a party in 1978" or something like that.

THE CHAIRMAN: Quite apart from drugs there would be those who had piercings in their youth and may have forgotten?

A. Well, one of the things that people don't realise is how dangerous homemade tattoos were. There was a bit of a fashion for that in the 70s and 80s.

THE CHAIRMAN: I suppose something like that will almost certainly not have had any symptomatic signs at the time.

A. No, the vast majority of non-A non-B hepatitis as it then was, Hepatitis C, have no symptoms whatever.

THE CHAIRMAN: So we are dealing with people who may not have attributed any significance at all to this event in their past.

A. That's correct.

THE CHAIRMAN: It may not necessarily be wilful withholding of information in some cases.

A. Yes. I think at times there is denial going on about behaviour in the past.

THE CHAIRMAN: Well, there is a natural explanation of denial, isn't there?

A. Yes.

MS DUNLOP: Just another short point, Dr Gillon, and then I think it is probably time for a break.

You were posed some further queries by the Inquiry team.

Sorry, but this is one occasion where I would like to juxtapose the two documents, if I could, please. The follow-up queries of [PEN0131557]. We need to go to the second page, please, to PEN0131558. Thank you.

Just in case people are wondering what the question was, firstly you were asked: could there be any overlap between those three groups? You have answered that there. I think in short you are saying: well, you can't rule it out but you don't think it is very likely.

A. Yes, I think that's exactly right.

Q. Can we go down that answer a little bit, please.

You go on to say what you have really just said in your evidence. You have explained a little bit more of the detail, particularly of the third group; how you went about deciding whether or not to include a particular individual in your third group of people. We find that set out there. Is that correct?

A. Yes, that's right. I think there is no doubt that a total of 28 is way off what is the reservoir of such cases in the population. We don't know by how much, of course.

Q. Yes, because -- and we really come full circle here but we are right back to what you said at the beginning about clinicians -- it is really dependent on the clinician having the thought and reporting somebody to you as a possible case of transfusion transmitted Hepatitis C.

A. Yes.

MS DUNLOP: I think, sir, that would be quite a good moment to stop for a short break and then we can take the rest of the Dr Gillon's evidence.

THE CHAIRMAN: Yes, I should ask one question about that last category which is interesting me. Where one is dependent on a clinician making a report, is it likely that the clinician will report only where he sees some significance in the context of his operations for the factor or feature, or do people report just casually if they come across it?

A. No, I think the first is the correct interpretation, which is that we would tend to see reports historically from, for instance, haematologists, who are dealing with patients all the time, who are requiring multiple transfusions. We have fairly close and constant interaction with them. So the link is there. Similarly, in units who use a lot of blood, like cardiac surgery -- and again we have a lot of clinical links --

that would make them more alert to the possibility. Also renal units, kidney diseases.

THE CHAIRMAN: That's what I wondered.

A. Your jobbing surgeon at the district general is much less likely to think of it.

THE CHAIRMAN: It rather suggests that those who don't have a focus on the problem are much less likely to respond.

A. Yes.

(11.02 am)

(Short break)

(11.33 am)

MS DUNLOP: Dr Gillon, we had got to 220 people. We need to go back to your statement, which is PEN0010045.

There we see the 220 people are at the top of the page. You then tell us that you added in another 18 people. Can you just explain to us where these 18 come from?

A. These were when we were trying to compile the list of people reported by clinicians. I learned about this for the first time, that in the west of Scotland the renal unit had identified some patients -- after they started testing for HCV in 1991 -- who had had large numbers of transfusions, whom they had identified to be HCV-positive.

Indeed, some had been identified prior to the

introduction of testing. The reason they were interested in this and monitoring it was that it was well-known that hepatitis transmission in renal units.

THE CHAIRMAN: Ms Dunlop, I have to interrupt. There is something wrong with the sound system. No one is hearing what's being said.

(Pause)

Can we start again.

A. Is that better?

MS DUNLOP: Yes. I think you are going to have to start again, Dr Gillon, with your explanation of where the 18 people come from.

A. Right. Well these were patients who were reported by the renal units in the West of Scotland as having been identified to have Hepatitis C when they started routine testing for that after the test became available, and who might have been infected as a result of a transfusion.

Hepatitis of all sorts really -- it started with Hepatitis B -- does have a high prevalence in patients on chronic dialysis. That had been known for many years. The reasons for that weren't entirely clear even to this day. Some of it was transmitted by the process itself, by the difficulty in sterilising dialysis machines, particularly the earlier ones. It's not

a problem now. But there were probably other routes of spread within dialysis units as well.

On top of that, patients with chronic renal failure have sometimes quite severe anaemia as part of their condition and require regular transfusions. So many of these patients will have had many, many transfusions and most of those would probably have gone back beyond the time when the sample archive was started, which in the West of Scotland, was 1986, I think.

So I don't know who made the decision but at some point it was decided that these cases should not be investigated or could not be investigated further to try to identify a source. But they were there. They were known as potential transfusion transmissions and that's as much as I know about it.

Q. Right. You have explained to us about the renal dialysis unit. Is that the second paragraph? The paragraph beginning "A small number ..."? You talk about the risk of nosocomial infection, which goes with dialysis.

A. Yes. That's what I was talking about, either through the equipment itself, through, conceivably, reuse of other types of equipment which shouldn't have happened, like, you know, reuse of needles. It just shouldn't have happened, that kind of thing. There is still some

doubt about exactly how the prevalence in some units got to be as high as, in some places, 20 per cent of patients.

Q. We did, I think, ask you about this too in our follow-up document, Dr Gillon. If we could again see that beside Dr Gillon's statement, please. The document is [PEN0131557].

If we could go to the next page, please and the page after that too. There is a paragraph with the number 4, where you say the high prevalence of parenterally transmitted virus infections. Parenterally infections are those which are essentially blood to blood or wider than that, bodily fluids?

A. Bodily fluids yes.

Q. Rather than enterally which would be things you eat and drink?

A. Yes.

Q. Yes?

A. Can I apologise for the typo, which a colleague gleefully pointed out to me, of "incompletely misunderstood".

Q. I was just about to take you to that, yes. There is an extra "mis" in there. We will take that out. I think that's the explanation you have just given us. I'm also interested in the word "nosocomial" which we have seen

before. That probably has classical etymology but could we just say hospital-acquired infection?

A. Yes. I am afraid I can't enlighten you about the derivation of the term.

Q. I think the other people I was interested in were the bone marrow transplant recipients. They number 18, I think. Are those people with leukaemia and other --

A. Yes, they would be people with primarily leukaemia who had had multiple transfusions over many years, resulting in bone marrow transplantation.

Q. They have been added in but as I understand it, you are saying identification of the donations which infected them was considered impossible because of the number of transfusions involved. So they have had a lot of transfusions and you presumably do not have test results which would enable you to pinpoint any one transfusion in relation to an individual.

A. I imagine that was the case. In such a case, really the only way you can find if there is a donor who transmitted would be to have archive samples which you can go back and test. We have certainly in Edinburgh done occasional look-backs where we have tested between 100 and 200 samples, which is a very big exercise. Occasionally it does provide results. But some of these patients will have had hundreds of individual units of

transfusion and many of them will have had these transfusions before the archive samples started, which would make it impossible.

Q. I see. You were also asked, if you could, to address the date of transfusion -- and this is back to your report -- which resulted in each patient -- I think it is probably contracting Hepatitis C. You have told us, first in relation to the second of your three groups -- that is the look-back people, if we can call them that for shorthand --

A. Yes.

Q. You have set out in your report, beginning at the bottom of page 3, the numbers identified for each year. The subset is in fact 103 of the 133. I think you were asked, "Well, why is it only 103 of the 133?" And that's in the further queries at the side. You have dealt with that too. Paragraph 5. The goal was to include all 133 but for various reasons 30 of them dropped off the survey really.

A. Yes.

Q. You say you have discussed this with Dr Helen Harris, who is the coordinator of the register from CDSC or HPA at Colindale. She made a number of visits to the transfusion centres and she included all the patients whose details she could get. You say:

"The reasons why some patient records were not available ..."

This is looking at your paragraph 5:

"... are not clear. It is likely work was ongoing in certain cases, ongoing correspondence ..."

But you say:

"The 103 patients can be taken as unselected and therefore representative of the 133."

So you think they are a reasonable spread?

A. I'm sure they are representative having spoken to Helen Harris. There was no selection for entry into the study, other than that the records should be available. She, like me, cannot explain why on the day she got some records and not others. But she certainly got most of them. The only real entry criterion was that it should be transfusion transmitted -- which it was by definition -- and a known date of transfusion.

Q. In appendix 1 to your report, page 50, PEN0010050. There is a tiny typo here, Dr Gillon. If we look at the right-hand column and count four from the bottom, that should obviously be 1985?

A. Oh, yes, I do apologise.

Q. Just so we know. You have actually produced the same data in the body of your statement, showing us the numbers in each particular year. So if we go back to

page 45. It is PEN0010045 on the left.

It looks actually, give or take one or two years, a pretty steady rise to 1991. I wondered if that was just because in the nature of the exercise you are looking at blood donors who have been picked up after screening in 1991 and you are more likely to find donations that they have given that have been transfused in the years closer to 1991 than if you were to go further back. Is it as simple as that?

A. Probably not quite as simple. It will also illustrate the point that we made earlier that, the further back you go, the more likely it is that the patient will have died of some other problem or indeed that it will be harder to trace the person -- well, no, obviously these are people who have been traced.

Q. Thank you.

A. I think your point is valid.

THE CHAIRMAN: If we treat the 103 as truly representative of the whole group, then one would expect the spread to be similar for the unknowns as it is in the 103? Or not?

A. I'm sorry, the "unknowns"?

THE CHAIRMAN: You have indicated that Dr Harris' study identified 103 of the 133.

A. Yes.

THE CHAIRMAN: Whose data could be analysed and you and she agree that they are representative of the class as a whole.
A. The 133, yes.
THE CHAIRMAN: Yes. So if we take 103 from 133, we get an answer that so far doesn't have a date of transmission attributed particularly.
A. Well, no, I don't think she excluded them on that basis. I think for some reason the records were not there when she --
THE CHAIRMAN: I appreciate that. I'm just wondering whether the chronological spread can be taken to follow the same curve --
A. I would expect so.
THE CHAIRMAN: -- as the 103.
A. I would expect so.
THE CHAIRMAN: So one would have again a concentration in the second half of the overall period.
A. Yes.
MS DUNLOP: Then the same exercise, Dr Gillon, for the people who have come by the clinician reporting route. This is paragraph 2.2.
You say that those transmissions -- transfusions ranged between 1979 and March 1991 and that's in appendix 2, which should be page 52.

One of the things that struck me about this, Dr Gillon, was that if you, perhaps slightly laboriously, go through and list the period between the date of transfusion -- so column 3, and column 2, the date when this person's infection was reported to SNBTS -- you produce quite a spread but the shortest seems to be number 9, who has a transfusion on 23 October 1987 and is then reported to SNBTS as a possible case of transfusion transmitted infection in March 1988. Do you think it might be somebody who has been acutely ill with hepatitis? Might that be ...?

A. I think it must have been. I don't recall the individual case. It's certainly a short period of time. And the occasional patient who is infected with Hepatitis C will develop jaundice in the early part of acute hepatitis. That is relatively rare but it does happen. I think that must have been the case. But then we have gone back to that when testing became available.

THE CHAIRMAN: I'm a bit worried about 1988. If one looks at all the dates of reporting down that column, it stands out as being very, very early.

A. It does.

THE CHAIRMAN: Could it be 1998?

A. It could be 1998. It could be another typo. I would have to look into that. I think that's the most likely

explanation actually.

MS DUNLOP: If you could, perhaps, Dr Gillon, and let us know.

At the other extreme there is number 15. Patient number 15 appears to have had a transfusion in 1980 and is then reported to SNBTS in 2007. So 27 years have passed. I think, apart from people who have received multiple transfusions, these seem to be the opposite ends of the spectrum.

A. Indeed, and again, I don't remember the individual case and I would guess that we certainly wouldn't have had an archive. It is unlikely that we would have been able to trace that donor or test them.

Q. I think if we were to take out the six-month person, the next shortest would be two years, which would be the second patient. Had a transfusion in 1990 and is reported as under two years in May 1992. Anyway.

A. Indeed.

Q. Largely speculative to study this for too long, I suspect.

A. I could identify that patient. I think that's almost certainly Mr GRO-A

Q. Oh. Would it help us to know why?

A. Sorry, why ...?

Q. Why you think that's misleading?

A. It can't have been, no, I beg your pardon.
Q. Sorry, my ears are going. It is the wrong date, I am afraid.
A. It's the wrong date.
Q. It was 7 August 1990?
A. And it's the wrong category of patient, I do apologise.
Q. Going back to the body of your statement, you have also given, for the 103 patients -- and this is paragraph 3, I think on page 46 -- the Blood Transfusion Service region in which the blood for each transfusion was collected. The breakdown really accords with the population spread, does it, more or less?
A. Yes. Relatively speaking, Aberdeen seems low there. Whether that means that there were more records unavailable on the day in Aberdeen or what, I'm not sure. But otherwise it does seem to represent populations.
Q. If we can look over to the next page, please, that of the 103. You are able to tell us that only 49 of them were known to be alive at January 2011. I suppose, obviously, more of them may be alive; it is just that the data that's held by HPA is only able to tell you definitely in relation to 49 people. Is that right?
A. Well, no, they know for sure that 53 -- sorry --
Q. Yes, sorry, I'm forgetting. Of course, you are coming

on to that.

A. They have a report of the death and a copy of the death certificate.

Q. Yes. In fact, most of the reports in this area that one reads seem to deal with patients known to have died rather than patients known to be alive.

A. Yes.

Q. That's presumably a slightly safer measurement with which to work, is it?

A. It is, yes. I think they enquire into the status through the register office annually, I think -- maybe six monthly. So they are getting very regular updates on these patients.

Q. In relation to the patients known to have died, the 54 patients in the next paragraph, you mention that one of those was a consular notification for which no cause of death was known but you have a spreadsheet. I'm not sure how easy this is going to be to display.

THE CHAIRMAN: CinemaScope.

MS DUNLOP: Yes. Your appendix 3. I think we were hoping to display those by alternative means.

I'm sorry, it is only available in hard copy but perhaps I can allow everyone else to have a look at the hard copy when we stop for lunch and let people see that if one goes through the spreadsheet with the 53 people

on it, there seem to be 14 deaths which mention Hepatitis C at some point in the death certificate. I don't know if you have a copy with you, doctor.

A. I don't have a copy but I do remember that. I think the surprising thing is that only 14 mention Hepatitis C, when they all had Hepatitis C in fact.

Q. Yes. Then you yourself examined the 53, and you say your interpretation is that Hepatitis C was the cause of death or contributed materially to the cause of death in eight of the 53 patients for whom the causes of death are known. So in very general terms I take it that where somebody had, for example, died of a myocardial infarction but was known as also having Hepatitis C, you didn't count them --

A. Yes, I think that would be correct.

Q. -- as people for whom Hepatitis C was the cause of death or a material contribution.

THE CHAIRMAN: Ms Dunlop, I'm anxious that there should be some record of methodology so that, quite apart from the examination of the document over lunch, there is something in the transcript that tells us what Dr Gillon has done. Might it help if he had it and just gave a brief description --

MS DUNLOP: Yes, certainly. I'll pass it over. I have highlighted all the Hepatitis Cs, Dr Gillon. (Handed)

A. Thank you.
THE CHAIRMAN: I think, doctor, if you could identify the data, describe the methodology and explain the selections, first of all, then we might get a basis for going forward.
A. This, in a sense, is the essence of the study that was set up, in that -- well, this is the Scottish contribution to the data. Of course, there is a much bigger number which includes data from England and Wales too, but by knowing the date of transfusion they then planned to follow the outcome in these patients, not only by looking at the register of deaths but also by regular reports from these patients' clinicians. The clinicians were asked to sign up to this at the beginning and give access to the hospital records so that clinical data could be obtained right from the start and then regularly throughout.
THE CHAIRMAN: Does that mean that the death certificates are not the exclusive source of information in this case?
A. They are not the exclusive source but they are in a sense. This is the hardest end point, which is about mortality. I should also add that these data are so far not analysed by HPA and haven't been published. So this was kindly made available to us by Dr Helen Harris for

the purposes of the Inquiry. The interpretation which I have given is a personal interpretation, and may not reflect what they finally decide when they analyse these things formally through HPA and publish the data.

THE CHAIRMAN: It is the exercise of your judgment for the time being.

A. As counsel said, I really categorised people into those who had clear evidence that their final demise was fairly directly attributable to hepatic disease. In other words they have liver failure or a complication such as sepsis, or they had hepatocellular carcinoma. Again, there may be a primary cause of death such as bronchial pneumonia, but hepatocellular carcinoma, Hepatitis C. That is clearly attributable to the Hepatitis C.

So my reading is that eight of these 53 had a final illness where Hepatitis C was the significant factor.

THE CHAIRMAN: So that in the balance of 45, Hepatitis C is certainly an aspect of their general medical history.

A. Indeed.

THE CHAIRMAN: But in your view, looking at the totality of the information available, it was not a significant contributor to death.

A. Yes. I think that by and large at this sort of length of follow-up, which is 20 years plus now, that's broadly

in line with what's in the published literature.

THE CHAIRMAN: Ms Dunlop, I don't know if it is necessary to go beyond that.

MS DUNLOP: No, I don't think so, sir. It gives us an impression. Perhaps an interesting feature is the feature Dr Gillon has highlighted, that all 53 of these people had Hepatitis C and it has only been recorded in relation to 14 of them. Then the next stage is, as your Lordship put it, the exercise of judgment that Dr Gillon carried out. He thought that it featured prominently, if one can put it that way, in eight of the individuals.

Dr Gillon, that really concludes the information that you included for us in this first statement. You, I think, really refer us to Health Protection Scotland for some of the other data and obviously we have got Professor Goldberg coming to help us with that.

But you also provided a statement in relation to transfusion transmitted HIV, which is [PEN0010038], and I don't want us to lose the further queries either, please, if you could hang on to them for the moment.

You tell us that 18 patients are known to SNBTS and HPS to have contracted HIV as a result of a blood transfusion in Scotland. Ten of these patients were identified through the targeted look-back by SNBTS. Am I right to think that that was in 1985?

A. Well, 1995 was --
Q. Sorry, this was the HIV look-back.
A. I'm sorry.
Q. There was an HIV look-back in 1985?
A. Look-back was instituted as online as soon as we started HIV testing in 1985 and that was a UK Transfusion Service's policy decision.
Q. Through that mechanism you identified ten people and then another eight patients were reported, presumably to you, to the Blood Transfusion Service?
A. Yes.
Q. By their doctors as possible transfusion transmitted infections.
A. Well, actually, only 17 of those were reported to us. There was one patient whose data was on the HPS database, about whom we knew nothing.
Q. That's patient 9. We are coming on to patient 9, sorry.
You say in four cases it was possible to identify a blood donor as the probable source of the infection, whereas in three of the remaining four, the evidence for transfusion transmission was circumstantial. You explain that, that you couldn't get actual evidence because transfusion had occurred before testing for HIV and there was no archive sample that you could go back and test. I think you have already said to us that the

systematic storage of archive samples began in 1986?

A. Yes, a little earlier in Edinburgh.

Q. Sorry. When was it in Edinburgh?

A. It was mid 1984.

Q. Then the remaining case, you say, was reported to Health Protection Scotland by clinical staff in The Western Infirmary, Glasgow, but is not known to SNBTS. Then you were asked some questions about that person. If we could go to the further enquiries document. Look at the first page. You were asked some questions about this patient: whether you accepted that this patient did acquire HIV from transfusion, which number in the table the patient was and how the patient had been missed really. You have explained this. You have said:

"The patient was patient 9 in the table."

We will look at the table in a minute:

"...of Dates of transfusion. The patient was reported to Health Protection Scotland which was then ..."

Do you pronounce it SCIEH?

A. Yes, SCIEH is what people tend to say.

Q. SCIEH. That stood for?

A. The Scottish Centre for Infection and Environmental Health.

Q. Thank you:

"... by the consultant virologist at the Western Infirmary, Glasgow, in 1986."

The only additional information you had was that the patient had sickle cell anaemia. Can you explain sickle cell anaemia, please?

A. Before that, it may be worth saying that this was through the mechanism I identified earlier, of automatic reporting of the referral form back to SCIEH when a positive was found. As far as I or anybody now working at HPS knows, that is the only information that was obtained about this patient. So what was on the referral form.

Sickle cell anaemia is an inherited disorder of red cells, which is commonest in black African and black American/Caribbean populations and is a major source of serious disease which can result in what's called a sickle cell crisis. The red cells have a structural defect which in certain circumstances can cause them to be distorted and to effectively clog up the smaller arterials and capillaries. It is a very serious and very painful disorder. To prevent that they receive frequent top-up transfusions of normal red cells. So you have to keep a proportion of normal red cells in the blood to prevent this.

Of course, transfusion is one of the treatments of

a sickle cell crisis.

So from an early age people with severe sickle cell anaemia are heavily transfusion-dependent.

Q. I see. You say in relation to that patient -- and I'm reading from the follow-up queries:

"It is not known whether these transfusions were given in Scotland or elsewhere."

The only reservation I had about that was that you go on in your statement to list for all 18 individuals, transfusions in various different parts of Scotland. So we can take it that this person had had at least one transfusion in Scotland. Is that right?

A. I think it is an assumption but, yes, the patient was being treated at the Western Infirmary in Glasgow. So we have to assume that transfusions took place there then.

Q. Right. Then --

A. And in fact there is the date of, I think it was, May 1984, which is also on the HPS database and that's what was on the database.

Q. In your follow-up queries, you have explained what I think you have just alluded to, which is the reporting mechanism, that sometimes it was possible to get further information but the basic information that came from the virology laboratory was just whatever had been on the

test request form. Just sticking with the response to the further enquiry, you say it suggests that the clinician involved did not report the case to Health Protection Scotland nor to the SNBTS.

So didn't report the case. Whoever was looking after, the consultant who was looking after the patient did not reported the case to Health Protection Scotland, it just came from the lab.

A. I think that's what happened. At this distance in time and given that HPS is an anonymised database, I don't think there is any way of investigating that further.

Q. I think where you say why you included this case, you say that the circumstances were compatible with the relatively high risk of exposure through -- I think that should probably be transfusion, should it, rather than "transmission"? That's the end of the third paragraph, on the right-hand side.

A. Yes, of course; yes. Apologies.

Q. Not at all. Then you say:

"There has never been an agreed policy nor a legal requirement for clinicians to report possible transfusion transmitted infection to [you], although you have sought to encourage that."

A. Yes. In fact HIV was not a reportable disease. So in that sense there was no legal obligation on the

clinician to report to HPS either.

Q. Going back to your main statement on this topic, Dr Gillon, you then cover the date of the transfusion which resulted in each patient contracting HIV, saying no date can be established for three patients. In fact, we can see from the follow-up query that the three you have in mind when you say this are 2, 9 and 12. So if we look at the table, which is on PEN0010042, you regarded it as not possible to establish a date of transfusion for three patients. Just to look again at the table, that's 2, 9 and 12.

But for the remaining 15, appendix 1 has shown the most accurate available information. And we can do this exercise ourselves but you say the earliest was in August 1983. So out of the remaining 15, the earliest was August 1983, the latest in August 1986 and for the years between, it would be three for 1983, nine for 1984, one for 1985 and two for 1986.

Right. Can we go back to the body of the report, page 44, it will be. 38 I think it was or 39. PEN0010039. You were also asked how many patients were under 16 when the transfusion took place. You have said four, with one possible extra in the group who had no confirmed date of transfusion. 4 and 5. In 4 you tell us where the transfusions took place and these are

health board areas.

A. Yes.

Q. So eight in Lothian, six in Greater Glasgow, three in Tayside and one in Lanarkshire. Then for question 5 or paragraph 5, you have given us geographical data but related to the blood transfusion areas, which are different, and that's the five regions that we spoke about earlier.

So you have the sources of the infective donations for all 18 being Edinburgh and the south-east, eight, Glasgow, seven, and Dundee, which is the east of Scotland area, three. So your five regions, also including the Highlands and Grampian, we have none for either of those areas.

A. That's correct, yes.

Q. Then if we turn on to the next page, please, paragraph 6, you are asked about how many patients in this group had died of HIV/AIDS. You say you don't know about the cause of death for these people but you do know that 15 of them -- that's 15 out of the 18 -- were known to have died as at the end of last year.

A. Yes.

Q. I suppose, though, we should bear in mind the piece of information you gave us earlier about the number of people who may die quite shortly after a transfusion

anyway because of the illness or concern that led to them needing a transfusion in the first place?

A. Yes. HPS does have some clinical information on some of these patients and many of the comments really are AIDS-defining illnesses. So many of these patients will have died of AIDS.

Q. Then in conclusion, you were asked the number of partners who contracted HIV, and you weren't able to answer that because you don't collect that information. In view of that you weren't able to answer question 8 either.

Allow me a moment, sir. (Pause)

Yes, there was one other document I wanted to put to you, Dr Gillon. It is [PEN0010053].

We can actually see Dr Dow's name on this, if we look at the second page. This is just to tell us about the two viruses in relation to the question of how many infected donations have been detected since testing came in.

First of all, we can see that it's a tabular summary of data submitted to the SNBTS NMRU?

A. National Microbiology Reference Unit.

Q. It is, funnily enough, on the front as well, sorry.

Infection surveillance system by 12 July 2010 about positive donations. If we look at the first table,

table 3 tells us that since screening for antibodies to Hepatitis C began in relation to blood donation on 1 September 1991, 867 donations have been collected, which have been positive, and that in fact takes us right back to the beginning of your earlier statement, where you had that 867 figure. The detection of positive donations is also shown for us in a graph at the bottom. Very crudely put, we can see that the trend, both in relation to new donors and repeat donors, has been one of diminution and that's what you would expect, is it?

A. Yes, it is certainly what we would expect to see in repeat donors. The drop in prevalence of new donors, can reflect two things broadly speaking. It can reflect a change in the population prevalence, which may well be happening. It could also represent better donor selection, to put it broadly. It is impossible to separate out the two really, I think.

Q. If we turn the page, we can see the same sort of table but in relation to HIV. We can see, if we look in the same place on that table -- that is the bottom right -- that since testing began on 1 October 1985, a total of 95 positive donations have been detected. That one, from the graph, is bouncing around quite a bit more, isn't it, Dr Gillon?

A. Yes, the one in new donors. It is really very low numbers, though. That reflects the left-hand column in the table and as you can see, you are dealing with tiny numbers: 1, 0, 2, per annum.

Q. In fact a number are none.

A. In some years none. I'm sure there is no statistical significance there.

Q. There is, presumably, quite a lot of resource which goes into the screening programme?

A. There is indeed, yes.

Q. Yes. Do you have any --

A. I assume by resource, you mean pounds, shillings and pence.

Q. Yes, I do. I just wondered if by any chance you had any sense of a figure?

A. Yes, I looked at this and in crude terms, just in terms of the cost of tests itself, we are currently expending roughly three quarters of a million pounds on our first line screening and what's called NAT testing, nucleic acid testing, which is also known as PCR, for these viruses. It is a little difficult to separate out the individual components because the machines which do this use combination tests now. But as far as we can separate it out, it is costing us about £750,000 a year for HIV testing.

THE CHAIRMAN: Who are "we" in this context, Dr Gillon? Is it the whole of Scotland or --

A. The whole of Scotland, SNBTS, yes. So that's for 250,000 donations a year, approximately. The actual HIV component of the screening process is only 57 pence. The nucleic acid testing, which is the testing directly for the virus genome, costs around £2.20 per HIV test.

THE CHAIRMAN: £3 a cycle, is it?

A. It is about that. Then there are repeats that you have to do and follow-up samples and so on. Overall the laboratory manager's best estimate is three quarters of a million for that.

MS DUNLOP: The only reason for asking, Dr Gillon, is that in a book which a number of us have looked at, Douglas Starr's book on blood, there is a quote for how much the testing programme is costing in the United States and over a period of years it seems to be in the region of several hundred million dollars but perhaps they are doing more extensive testing than we are doing here.

A. No, they certainly aren't. That would be hundreds of millions of dollars over years for the American population.

Q. So it is just a reflection of the different --

A. It is just the different scale of population.

Q. Right, thank you. Thank you very much.
THE CHAIRMAN: Mr Di Rollo?
MR DI ROLLO: Mr Dawson has one or two questions.
MR DAWSON: I think my voice is being picked up.
THE CHAIRMAN: Your voice is very quiet here.
QUESTIONS BY MR DAWSON
MR DAWSON: I'll speak up.
Dr Gillon, perhaps we could have Dr Gillon's statement, which is number [PEN0010043] on the screen. This is your statement to the Inquiry, Dr Gillon, which relates to transfusion transmitted hepatitis.
You work for SNBTS. Is that correct?
A. That's correct.
Q. As I understand it from the first paragraph that one sees under "preamble", in providing information to the Inquiry, you have collated information from a number of different sources on this topic. Is that correct?
A. The collation refers to an attempt -- not an attempt. We did sit down with HPS, with Professor Goldberg and others, to make sure that we each knew about the data that the other held.
Basically, all of this information is from patients who have either been identified by SNBTS or reported to SNBTS from clinical sources.
Q. I'm interested to know what the relative

responsibilities of the various bodies that you identify in the preamble are with regard to the compilation of statistical information such as that you have provided to the Inquiry. Can you help me?

A. Well, as I say, this is all primary SNBTS-derived data. So when we talk about HPA data, this refers to the study which was set up by HPA in combination with the transfusion service in England and Wales to which Scotland agreed to contribute after a lot of deliberation and discussion to make sure that we were satisfied that (a), the data were going to be sufficiently well anonymised, that no patient could be identified through that, and (b), that those data would remain available both to SNBTS, to clinicians throughout the United Kingdom for further research.

In other words, it wasn't going to be put in a box and never made accessible to other researchers. Those criteria have been fulfilled. In fact, I know that Scottish clinicians have applied to and been granted access to the data for follow-up information.

Q. Are there any other bodies, other than those you have mentioned in that preamble paragraph, who would have statistical information that might be useful to the Inquiry on the issue of the number of people who may be infected with Hepatitis C as a result of blood

transfusions?

A. Other than the data on haemophilia patients, which would be treated separately in the Inquiry, I'm not aware of anything else.

Q. I'm obliged.

Could I just ask you briefly about paragraph 1.1, which appears in the bottom half of that page that we are looking at. As I understand it, what you are recording here is, first of all, the number of HCV-positive blood donors that SNBTS had on its records. That's 867. You are then identifying a percentage of those, 6.8 per cent, who are a cohort of that larger number, who have identified blood transfusions as their only risk factor for contracting HCV. Is that correct?

A. That's correct.

Q. Could I ask you whether the greater figure that is produced there represents the number of people who have been diagnosed as having Hepatitis C in Scotland or whether it represents the number of people who have received blood transfusions in Scotland, who have been so diagnosed?

A. The figure of 867 refers to blood donors who have presented themselves as blood donors and undergone testing of that blood donation. They are the ones who have been found to be positive for HCV antibodies,

presenting as blood donors, not presenting as patients with clinical illness.

Q. So these patients appear on your list because they have been tested as HCV positives in Scotland?

A. By the SNBTS, yes.

Q. Do you know, in deriving the 59 figure from that, whether or not the 59 received blood transfusions in Scotland?

A. We know that they declared that as a risk factor. We will have investigated them individually. As I explained earlier, for the purposes of presenting these data, we have not been able to go through these individually. Some may have been transfused outside this country, some, as we found with the clinician-reported cases of HCV, could be excluded on the basis of investigation. They may never have had a transfusion.

Q. So the position is that these are people who have tested positive in Scotland, who have identified blood transfusion as the only risk factor but they have not indicated blood transfusion in Scotland as the only risk factor?

A. Not specifically, necessarily.

Q. I think you have accepted that that might mean, if one were to investigate it more thoroughly, that some of the

59 may have received their blood transfusions outside Scotland?

A. That's entirely possible.

Q. Is the converse also true, that there may be people who perhaps are identified outwith Scotland because that's where they were tested as blood donors, who received transfusions in Scotland? Would those people be identified by this process?

A. Not by the process that we went through to do this, no. It is entirely possible. I'm sure there have been cases who have presented in England, for instance.

Q. That's precisely what I was thinking of, Dr Gillon. It may be that, on the assumption that a similar process has been gone through in England, there may be people who, because they are currently in England or gave their donations in England, are identified as English positives, if you like, but they may have received their transfusions which give rise to that in Scotland.

A. That's entirely possible.

Q. Thank you. If I might ask you, could we go over to the next page? I have a brief question on the issue of the look-back exercise, which is the second of the categories you have taken into consideration. I think you have identified in your evidence already that the look-back exercise which was undertaken has some

limitations on it, insofar as it might be taken to be a representation of the total number of people who are transfusion-transmitted HCV patients.

You have mentioned, I think, that it is dependent on good record-keeping and you have mentioned that there are a number of people who, for various reasons, are not able to be tested, be it because they were deceased or were simply unavailable for some other reason.

Could you explain how it is that people come to be traced by the look-back procedure and could you confirm my understanding that it is only on the basis of repeat donations that one identifies what I think counsel for the Inquiry has called the "infection families"?

A. That's a new term to me. I'm not sure what's meant by "infection families". But, yes, the look-back, as a starting point, takes those donors who are now known to have Hepatitis C, who have donated in the period prior to the introduction of testing in 1991. When such a donor is identified, the process that we go through is, of course, initially the informing and counselling of the donor, which is done through the clinician responsible for the transfusion, or the GP and the SNBTS. Any of those three might undertake the counselling and that is where the information about the donor history and donor exposure is obtained.

In terms of tracing the recipients, the next step is to identify what happened to each previous donation; in other words, what components were made from it, where were they sent to, what do we know about the fate of those individual components. Some of them will have been time-expired without being transfused, some will have gone off to quality control, most will have been shipped to hospitals.

You then, through formal routes, contact those hospitals, informing of this and asking them for details of the fate of that individual component; in other words, where it did it go, was it time-expired, did a patient get it and, if so, can we identify the patient.

Q. Am I correct in saying that the donors who were identified in the look-back exercise were repeat donors who were tested between 1991 and 1997?

A. Yes.

Q. 1997 being the date when the look-back exercise came to an end?

A. 1998 really but, as I mentioned earlier, that is ongoing. We would still, if we identified a donor now who had donated, and indeed I think Lord Penrose pointed out one who had donated in 2007.

Q. If that were to happen, would those numbers be included

within the figures you produced here?
A. Yes.
Q. Thank you.
There are, of course, a number of epidemiological studies which would suggest that there are considerably higher numbers of people who have been infected with HCV as a result of blood transfusions in Scotland, and I understand we will be going to those with different witnesses later, but no doubt you will be aware that such studies exist?
A. Yes.
Q. And they use different methods to arrive at their conclusions than your methods. Is that correct?
A. Yes, entirely different.
Q. Would it be fair to say that, given the way in which you have compiled your data, your final number, which I think is either 220 or 238, could be categorised as probably a minimum number of infections in Scotland through this route?
A. Yes.
Q. Thank you. I have one further question for you, which straddles over the two areas on which you have provided reports. You have provided information relating to transfusions in general, but both Hepatitis C and HIV. Do you have information about co-infection rates, ie

people who have been infected through this route with both types of infection?

A. I'm not aware of such a case.

Q. Would it be possible to compile such data?

A. It would be possible. I'm not sure exactly how difficult or otherwise it would be. I simply can't remember any such case myself. It is possible that other witnesses could help with that, specifically Dr Brian Dow.

Q. On the basis of the material you have looked at, you are not aware --

A. I'm not aware of it, yes.

Q. Thank you very much, Dr Gillon. Thank you.

THE CHAIRMAN: I take it that cases of co-infection must have happened, Dr Gillon.

A. It is quite likely but, as we have seen, the number of HIV cases from transfusion transmission is small and they occurred in an era when there was no Hepatitis C testing.

THE CHAIRMAN: Whether you could, from collected sources, identify these might depend on whether the data was anonymised before being passed on to a central agency. Do you know whether Health Protection Scotland data is anonymised or not?

A. It is anonymised.

THE CHAIRMAN: And what about your data? Is it anonymised?
A. Well, no, our data -- the data that we hold within SNBTS is all patient-specific or donor-specific.
THE CHAIRMAN: Mr Dawson, that might lead to question as to whether, on SNBTS data, there is any example of co-infection. I don't know whether you want to ask that or not.
MR DAWSON: Well, my understanding of the evidence that has been given is that, on the basis of the data to which Dr Gillon has access, he is not aware of any. It may be that other people have access to different data which would give different (inaudible).
THE CHAIRMAN: Speculation won't help at this point.
MR DAWSON: Absolutely.
THE CHAIRMAN: Mr Anderson?

Questions by MR ANDERSON

MR ANDERSON: I'm obliged, sir.

Can I ask you about a matter to which you alluded earlier this morning but which has not been ventilated; that is to say, the prevalence of Hepatitis C in the general population.
A. Yes.
Q. I ask you this, Dr Gillon, because of what might be thought to be a slightly mixed message coming out of a passage between one of the experts we have heard from

previously, a Dr Colvin, and the chairman. I don't know if you have seen this. Have you seen this discussion, which took place on 9 March?

A. I haven't read the transcript of that, no.

Q. All right.

Sir, for your information and for others, it may be helpful. It was within the evidence of Dr Colvin and it can be found on the transcript of 9 March at pages 111 and 112?

THE CHAIRMAN: 9 March is which day?

MR ANDERSON: Wednesday.

THE CHAIRMAN: It helps me to know the day of the Inquiry since that's the way I've noted it.

MR ANDERSON: Sorry, day 2.

THE CHAIRMAN: Day 2.

MR ANDERSON: Can I just take you through this, Dr Gillon?

Dr Colvin, in his evidence-in-chief, suggested that the prevalence of Hepatitis C in the general population was 0.1 per cent. All right? Now, if we just pause there, I think I'm right in saying that 0.1 per cent is one tenth of 1 per cent. Is that right?

A. That's correct, which translates to one in 1,000.

Q. All right. The chairman then asked Dr Colvin -- what he said was this:

"I can understand that the prevalence in the

population is 0.01."

Which is a different matter, isn't it?

A. Yes, that would be one in 10,000.

Q. All right. Dr Colvin replies to this and appears, I think, to seek to correct matters by saying:

"With respect ..."

But he then, rather strangely, goes on to say, "I think it is 0.01," which is indeed precisely what the chairman says. But he then says:

"I think it is a tenth of 1 per cent."

Just to clear up this confusion -- and it may be as a result of the transcription, we don't know -- what is your understanding? Is it a tenth of 1 per cent or not?

A. I think even that is too low a figure, to be honest, but certainly 0.01 per cent is far too low for the prevalence, as I understand it, in the British population.

Q. Where do you get this understanding from? What is the basis of the understanding?

A. I think I mentioned in passing earlier that good population data -- in other words, data which you derive from taking a representative cohort of the population in terms of demographic age, sex and so on and get them to agree to testing is very difficult to do and to my knowledge hasn't been done in the UK. The only

population data as such that I know of in the UK was a paper by Balogun et al from HPA in 2002. I'm sorry, I don't have the reference but I'm sure that this paper is known to the Inquiry.

They based this on taking the results of samples referred to the public health laboratories in England and Wales and came up with a prevalence figure of 0.7 per cent in the British population. That's probably too high because these were, by definition, clinical samples of some sort.

If you look at population data, some of which is based on speculation, I suppose, to put it broadly, in terms of trying to extrapolate from clinical data, some of which is very good, from the United States, where they have a rolling programme called the "NHANES": National Health and Environmental Nutrition and Environmental Studies" -- they do massive surveys I think it is roughly every seven years. In one fairly recent study, I think around about 2001/2002, they managed to get over 20,000 people, representative of the population across the United States, with one or two exceptions -- with the exception of people living in hostels and people in prison, I think -- I think everybody else was tested -- and they acquired vast amounts of information about these people, as well as

getting them to agree to give blood tests, and their information in the United States suggests that the population prevalence is something like 1.6 per cent.

In France they estimate it is 1.1 per cent. I have seen figures for other north European countries of around 0.6 to 1 or 2 per cent, with high prevalence in certain areas, like south Italy. American and northern Europe is considered low prevalence. In other words, anything under 2 per cent is considered a low prevalence area, but I think 0.1 per cent is too low for the United Kingdom and 0.01 per cent is unrealistically low.

Q. It is perhaps a rather rough and ready approach but would it be reasonable to suggest that it is somewhere between 0.1 per cent -- that is to say a tenth of 1 per cent; and 0.7 -- that is to say seven tenths of 1 per cent?

A. I think so, yes.

Q. You have mentioned the United Kingdom. Would I be right in thinking that that would apply to Scotland as well?

A. Yes, I think there is pretty good evidence that Scotland has a higher overall prevalence than England.

Q. I think you will see that the paper by Balogun and others has helpfully been put up on the screen and we see, for the purposes of the transcript, that it is [PEN0020822]. Is that correct?

A. That's correct, yes.
Q. I'm much obliged to you, doctor, thank you.
PROFESSOR JAMES: Lord Penrose, can I interpose one brief thing? Would you mind?
THE CHAIRMAN: Of course.
PROFESSOR JAMES: I believe that Dr Colvin was quoting as a prevalence in the United Kingdom the prevalence of HCV in the general population to Lord Penrose. Actually, the figure, which he got from the blood donor population, was very, very close to the figure that Dr Gillon had found from his original HCV blood donor population study, which we have already heard about this morning. So I think this difference between the 0.1 per cent, quoted by Dr Colvin, and the 1 per cent, seen in the Balogun paper that we have had on the screen, is due to a misapprehension by Dr Colvin, rather than a sort of mysterious difference.
THE CHAIRMAN: Well, I have no doubt that we are going to get lots of mysterious differences but if you are looking at the transcript on page 112, Mr Anderson, there is actually a word missing. What I was putting to Dr Colvin wasn't really concerned with a figure but with something else and it should be:
"I can understand that if the prevalence is 0.01 -- and that doesn't much matter -- then a single treatment

from a single donor will reflect that prevalence."

We then go on to the person with multiple transfusions. That was my interest, not in getting an actual figure.

MR ANDERSON: Sir, I think it would be remarkable if there wasn't the odd glitch in the transcript, I have to say. But perhaps I should explain. The reason is I think certain parties outwith this room have misunderstood and misinterpreted that passage, and I was simply --

THE CHAIRMAN: That will happen also.

MR ANDERSON: Indeed. I was simply anxious to clear up what appeared to be a misunderstanding.

THE CHAIRMAN: You can take it that I will not find myself bound by anything I said at that stage in arriving at the prevalence in the UK as a whole.

I think that we have actually seen a number of different figures. I seem to remember a figure of 0.088 as a prevalence in Scotland at one stage.

A. I think that would be a population prevalence based on blood donor prevalence. Blood donors are not a good way to assess population prevalence.

THE CHAIRMAN: I think I would like to follow that just a little. If one carries out a full epidemiological study, it would require a random selection from the population generally.

A. Yes.
THE CHAIRMAN: Is that right?
A. Analogous to what the Americans have done.
THE CHAIRMAN: Analogous to --
A. Yes.
THE CHAIRMAN: And that on any view would be an extremely difficult exercise even in a place the size of Scotland.
A. Yes.
THE CHAIRMAN: Partly because of the probability of different prevalences across different parts of the country.
A. And that apparently is the case. In the Balogun paper, for instance, London has a much higher prevalence than the rest of England and Wales, as judged from those public health laboratories.
THE CHAIRMAN: Once one introduces an element of self-selection into the definition of any statistical population, what happens?
A. You get a skewed result.
THE CHAIRMAN: And the probability of a good fix between the data obtained from the self-selected population and the general population reduces very considerably?
A. Yes.
THE CHAIRMAN: So if one looks at the blood donor population, one starts, by definition, with

a self-selected population?

A. Yes, and then we add the selection process which we impose.

THE CHAIRMAN: And some of the selection criteria relate to age, general fitness, medical history and so on?

A. Yes.

THE CHAIRMAN: All of which restrict even further the general applicability of the data obtained from the exercise?

A. Yes.

THE CHAIRMAN: Is that a correct understanding of the problem?

A. That is correct, yes, and also blood donors demographically do not represent the general population, by definition, because of age restrictions, and new blood donors tend to be in the younger age group of eligibility for blood donation, whereas regular donors tend to be in the older age groups. So there are all sorts of subtleties in there.

THE CHAIRMAN: If one then ends up with a data set that is skewed, is there anything one can do with it in the way of general application, by the application of judgment, or does it just represent what you find?

A. I think it is what you find. Given these subtleties about demographics, I don't see that it is valid to

extrapolate from blood donor prevalence data to the general population.

THE CHAIRMAN: Mr Anderson, I don't know if you have any follow-on on that?

MR ANDERSON: No, sir.

THE CHAIRMAN: Mr Sheldon?

MR SHELDON: No questions, sir, thank you.

THE CHAIRMAN: Ms Dunlop, does anything arise out of this that is of interest to you at this point? Sorry, in the sense of provoking additional questions.

MS DUNLOP: It's all of interest, but, no, I don't need to ask any more questions, thank you, sir.

THE CHAIRMAN: Thank you very much.

A. Thank you.

(12.51 pm)

(The short adjournment)

(2.08 pm)

MS DUNLOP: We have Professor David Goldberg this afternoon.

PROFESSOR DAVID GOLDBERG (sworn)

Questions by MS DUNLOP

MS DUNLOP: Good afternoon, professor.

A. Good afternoon.

Q. I'm going to ask you first of all one or two questions from your CV, which we have. That is PEN0020646.

In fact this is a short form. I have a much longer

CV and I realise that I'm not sure I have the number for the much longer CV. It is obviously not going to come after this page because this is only 1 of 1. Perhaps I can just ask one or two questions anyway.

We can see from this that you are a consultant in public health medicine at Health Protection Scotland and we heard the HPS. You are a honorary professor of public health at Glasgow University.

Thank you very much. I'm obliged to Mr Dawson for giving me the right number.

You are administrative head of the group for blood-borne viruses, sexually transmitted infections...

Could I get that back for a second until we have finished looking at the short one. PEN0020646.

You obviously have a group within Health Protection Scotland, which deals with blood-borne viruses, sexually transmitted infections, vaccine preventable diseases, respiratory infections. So they are all organised together from a statistical or epidemiological point of view. Is that the case? And then you are the lead on Hepatitis C and HIV programmes of work.

A. Correct. Yes, that's right.

Q. We see you serve on several United Kingdom and Scottish committees, are involved in academic supervision, author

of many peer reviewed articles and the holder of many grants. You have a team which secured a £43 million investment for Scotland's Hepatitis C action plan. You are chair of the action plan governance board.

If we go to your longer curriculum vitae, which is PEN0020671, to the second page, you tell us about your education and your medical qualifications. We can see you are a fellow of all three of the colleges of physicians, Glasgow, Edinburgh and London, and you take us through your medical career and your move into epidemiology. You started in the area, I suppose, when you were a registrar in infectious diseases at Ruchill; that right?

A. That's correct, yes.

Q. And rather developed your interest from there on. Then if we turn to the next page, we see your honorary appointments, and then you list the public service, that is within Health Protection Scotland. Is that right? Were you there when it was called SCIEH?

A. Yes, and before that CDSU. Communicable Diseases Scotland Unit.

Q. Right. We also see from the next page -- and this is about the middle of the page -- that over the last 12 years, much of your work has focused on leading a team and describing the epidemiology of the

Hepatitis C epidemic in Scotland and developing prevention, diagnosis and treatment services for Scottish people. Then you talk about the action plan on Hepatitis C, phase 1. What was involved in phase 1?

A. Phase 1 was essentially generating the evidence and from the evidence identifying proposed actions and costing these actions out. Effectively a business case for phase 2. Phase 2 was about the actual implementation of the action plan to improve services in all the areas that I have described. So phase 1 is this sort of business case development period between 2006 and 2008, involving evidence generation and the actions themselves.

THE CHAIRMAN: Ms Dunlop, I seem to be hearing the professor naturally and not through the transmission of rather peculiar sound.

A. I'm -- that's because I'm --

THE CHAIRMAN: It is more likely to be the machinery.

A. I was too far back.

MS DUNLOP: Phase 1 of the plan was really evidence gathering and phase 2 is presumably about implementation of the plan you had drawn up.

A. That's right. In phase 1 there was also a, I suppose, co-ordination component, getting some co-ordinating infrastructure into place. We had an action plan

co-ordinating group and in terms of the relevance to this Inquiry, we did have a patient representative for haemophiliacs with HCV and HIV.

Q. Yes, because, as you have told us, you have a great deal of experience as an epidemiologist in both Hepatitis C and HIV.

A. Yes. I mean, it is HIV which really, from a clinical perspective, inspired me in the mid 1980s when I was at Ruchill Hospital doing infectious diseases. That's why I moved into the epidemiology of HIV at that time and then that moved on to Hepatitis C.

Q. I think we have covered this already. From page 6, you talk about being head of that particular group within Health Protection Scotland, but I noticed from page 7 that you still perform on-call duties and this is really as a public health doctor.

A. That's right, yes.

Q. You make a contribution on a one in five consultant basis?

A. That's right, yes.

Q. What are the sorts of things that crop up?

A. Oh, just anything. I mean, what we tend to deal with are problems that health boards find difficult to deal with. So it is a hub and spoke mechanism. We have at the centre Health Protection Scotland. That's the hub.

Then there are spokes out into each of the health boards which have their own health protection units. So they are very much more reactive than we are and they deal with the day-to-day sort of issues, gastrointestinal infection outbreaks, respiratory infections, meningitis, that sort of thing. They tend to deal with it without our support. But if a problem is complex or involves more than one health board, we are brought in to advise.

I mean, recently, for example, we were involved in an incident -- and I led this -- involving two haemodialysis patients in the West of Scotland, who'd gone to Mallorca. They were Hepatitis C antibody negative before they went and they came back Hepatitis C antibody positive on screening. You know, they get screened every three months. We investigated that incident along with individuals in Spain and we identified that they did indeed become infected. They became infected through haemodialysis in Mallorca. So I can just give you that as an example but it also, of course, is relevant to Hepatitis C.

Q. Thank you. On the following page you have a list for us of the different pieces of work in which you participated for the Scottish Executive. We can see really quite a number which are relevant to the viruses that we are considering. Perhaps we can highlight the

AIDS co-ordinating group and the expert advisory group on HIV/AIDS treatment, the chief medical officer's committee on Hepatitis C and then all the ones which relate to the action plan that you described.

A. Yes.

Q. Then you are also, beyond the Executive, you are involved in -- or you were involved in -- the Scottish hepatitis working group. I think we have actually already heard about that. Is that now defunct?

A. Yes, that's defunct.

Q. Then looking at the next page, you are involved in the SIGN group on Hepatitis C infection. Then you have a list of United Kingdom committees as well and then international representation of SCIEH. Then a number of guest lectures, many of them obviously on Hepatitis C. The supervision you carry out of postgraduate work and the grants you have received for research. Finally a long list of publications of various different types, in which again, a great deal of the matters in which the Inquiry is interested feature, and even a list of book chapters as well.

A. Hm-mm.

Q. So you come here today from Health Protection Scotland to contribute to our attempt to get some feel for the size of the problems and to do that by looking at

epidemiological data.

You have prepared two different statements and I would like to start with [PEN0130014]. You have helpfully repeated, as question 1, that you were asked about the number of patients treated by the NHS in Scotland known to have contracted Hepatitis C as a result of a blood transfusion. Before we look at your answer, professor, I wanted to ask generally as an exercise in epidemiology, is this quite difficult?

A. This is an extremely difficult question because not only are you asking if an individual has Hepatitis C, you are asking if they have had a blood transfusion, and then you are also asking if there is an association between the blood transfusion and the infection. So effectively I didn't answer that question.

Q. Right.

A. Because the question is "known to have contracted". I think initially, in discussion with my colleagues at SNBTS, we felt that it would be best to concentrate on the look-back data, which are far more accurate in this respect. Of course, Jack has spoken to you this morning. Then I was asked to also just provide data that HPS holds in relationship to blood transfusion and Hepatitis C and I have done that. But the word "may" is in there:

"... may have resulted in the acquisition of Hepatitis C infection."

As you can see.

Q. Yes.

A. So I think that applies essentially to all the questions being asked.

Q. I suppose some of the reasons why this is a difficult exercise would be that the problem appears to have been occurring over a long period of time and there may be quite an interval between somebody having a transfusion which may have caused them to become infected and that infection coming to light. Am I on the right lines?

A. Yes. Obviously pre-1991 is the issue and Hepatitis C has been around for a long, long -- probably going back into certainly the middle of the last century. To what extent we are not sure but I think it would be reasonable to assume that it was around. Discovered of course in 1989, the test available in 1991. So I suppose the factors which influence matters is the prevalence of Hepatitis C in the general population and then that influences the prevalence of Hepatitis C in the blood donor population. Okay?

So you have got the blood donor population, and because you don't know for sure what the prevalence of Hepatitis C in the general population was pre-1991, you

wouldn't know for sure what the prevalence was in the donor population.

Then, of course, there is the issue of epidemiological screening that took place in the early 1980s and beyond. Screened out people at high risk, principally for HIV, at that time. So I think there is a huge amount of uncertainty here and we can estimate the size of the problem. I believe you have asked us to do that and we will generate some estimates of the number of people infected through blood transfusion during 1970 to 1991.

Q. Yes. You are hoping that that work will be available for the Inquiry towards the end of April. Is that right?

A. That's right.

Q. Thank you. You referred in your answer, professor, to epidemiological screening. I take it that what you are talking about is that in the early 1980s, there was quite an intensive focus on perhaps not accepting blood from particular groups of donors?

A. That's right.

Q. Even though it was directed towards AIDS, may as a side effect have also withdrawn from the pool people who had hepatitis?

A. That's right.

Q. But then on the other hand it might be -- I suspect possibly is the case -- that the actual prevalence among a group of people, maybe those using drugs, injecting drugs, might have been increasing. That would affect an assumption that you might choose to make as well. So these are, I suppose, the imponderables of the situation, are they?

A. So, on the one hand you have measures that were being introduced to make blood supply more safe, the epidemiological screening, culminating in the testing.

Q. Yes.

A. But on the other hand you have an increase in the prevalence of infection. Our work at HPS indicates that there really was quite a dramatic increase in that prevalence towards the end of the 1970s and beginning of the 1980s, contemporaneous, of course, with the increase in injecting drug use that we saw.

Q. You say in your answer that:

"Health Protection Scotland is aware of 304 individuals known to be antibody positive in Scotland for whom information indicated that a blood transfusion ..."

And I take your point. You say:

"... may have resulted in the acquisition of Hepatitis C infection."

You are excluding people who looked to have received a transfusion outside Scotland and also other individuals who are also known to have injected drugs. Then you say:

"Transfusion should only be regarded as a possible and not a definite or confirmed route of acquisition for these cases."

I think we will come to look at the notes which were attached to the statements that you provided. But two questions I wanted to ask you. I wanted to ask you both the how and the why of a report coming to you -- perhaps we could take the why first: why does a report come to Health Protection Scotland if someone is identified as having Hepatitis C? Is it just -- well I don't want to say "record-keeping". That sounds pejorative. But there must be more to it than that.

A. Yes. I mean, we established our Hepatitis C diagnosis database in 1996 and we acquire data from Hepatitis C testing laboratories in Scotland. They hold information that has been obtained through the test request form that accompanies the blood sample to the lab. In the instance of Hepatitis C, clinicians were and still are using a general request form. It varies actually in different parts of the country, the type of form that's used. It is not uniform in this respect. Of course, on

that form the clinicians document demographic information, identifying information. But also on that form is an additional information or clinical information space and sometimes they provide the laboratory with relevant information. It is by interrogating those forms at the laboratory that we are able to access the information that we need for our surveillance, epidemiological purposes.

Q. You are both monitoring what is happening and also -- and this links back to your description of the action plan -- trying to draw up the most suitable services for the population, based on the information you have about the extent of the problem?

A. Absolutely. This is just one source of information that we use. So we use this information in association with other pieces of information that we obtain through surveys of various population groups and also clinical information that we get through a national clinical database. So a whole number of information sources. We use all this information to answer some very key questions about this infection.

Q. Yes. I don't want to lose site of the statements; this is another occasion, if we can, for putting documents side by side. I wanted to ask you about a paper, which is [PEN0120066]. I'm hoping that the other parties also

have a hard copy of this. It is a paper about notifiable diseases.

Professor, I just want to ask you about this mechanism first before I ask you about its efficacy. This is a summary of the legal position that has been prepared by a member of the Inquiry team. It sets out, sir, the legislative provisions governing the position insofar as notifiable diseases are concerned. I think we can see that the legislative regime has quite recently changed in form of the public health et cetera Scotland Act 2008, but of course it is the position a little further back in which we are mainly interested.

You see that viral hepatitis -- the inclusive name for various types of hepatitis, including Hepatitis C -- became a notifiable disease under the 1975 regulations which came into force on 2 April 1975. I should say, sir, that we have looked at the position from 1975 onwards because that is the period of time in which the Inquiry is interested.

That, obviously, corresponds reasonably well with the time when the Inquiry is really beginning its examination. We can see viral hepatitis listed in the left-hand column. So that's something that certainly was in the 1975 regulations, and was also in the 1988 regulations which replaced the 1975 ones.

THE CHAIRMAN: I have seen the background correspondence in the files that show how this came about and what was understood by viral hepatitis at the time.

MS DUNLOP: Yes. Well, I think --

THE CHAIRMAN: If necessary --

MS DUNLOP: -- the professor has certain views about the efficacy of all this. But just looking at what the mechanism is, we can see from the next page that there is an obligation, or there was an obligation on a registered medical practitioner to notify, in this case viral hepatitis, if they had a reasonable suspicion that a patient whom they are treating has the disease:

"The disease is reported to the health board who in turn notify Health Protection Scotland."

Then there was a style form and a copy of that is there, shown on the next page.

This is scrolling on to the next page as well, please. We are told that:

"Health Protection Scotland have been in charge of retaining this information since 2007 and notifications are recorded in weekly and annual databases."

I think those of us who have gone on to your website, professor, are aware that you publish a lot of weekly reports of information. The paper also notes that HIV is a notifiable disease in some countries but

has never been a notifiable disease in the UK. And then there is a copy of a form which actually, I think, comes from the haemophilia centre doctors' organisation for their own system of reporting of a case of AIDS or HIV.

You were explaining to me before lunch, professor, that the system in the 2008 Act has changed slightly in that hepatitis is now not a reportable disease but a reportable organism. Is that right?

A. Hm-mm.

Q. What's the distinction that's being made there?

A. I think prior to the recent Act, as you can see, it was viral hepatitis that was the notifiable condition and it is not specific. There are different types of viral hepatitis. There is A, B, C, D, E, and there are other infections, viral infections, associated with hepatitis, such as Epstein-Barr virus, the virus that causes glandular fever, that can be associated with hepatitis, cytomegalovirus, that can be associated with hepatitis.

So you can see the difficulty here. It is not specific in any way and in reality, clinicians rarely reported the clinical entity, viral hepatitis, to health boards. So as far as we were concerned, the data that were ultimately collated by the information and statistics division of NSS, we ignored that information because we got better information by going direct to the

hepatitis testing laboratories. You cannot be diagnosed with, for example, Hepatitis C, unless you have a positive test for Hepatitis C.

So by going direct to the laboratories, using this voluntary approach, reporting approach, we managed to get the information that we actually needed.

That's for national epidemiological purposes. I think for local purposes the importance of this, in terms of the new 2008 Act, is that the laboratory is now being required to report the information to the health boards on individuals testing positive for these viruses, the hepatitis viruses. Certainly for Hepatitis B and Hepatitis A, it is important because there is a vaccine available for these infections and it means that you can go out and identify close contacts and vaccinate them. That's why it is important that these two infections are notifiable in this respect.

For Hepatitis C, I'm not so sure because there is no vaccine available. Certainly from my own knowledge of what happens at a health board level, the close contacts of individuals who are Hepatitis C infected generally speaking are not contacted; simply because I think there is no vaccine intervention available that can be implemented.

Q. Might that also be something to do with the relative

ease or difficulty of transmission of the virus just from person to person in ordinary contact?

A. I suppose that's a reasonable point. It is more difficult to transmit Hepatitis C in that way but I suppose, if the individual identifies as an injecting drug user, then the chances of the individual's partner being an injecting drug user might be quite high. But there is no vaccine anyway. So there is not much you can do in that respect.

Q. Just looking back at your statement, your figure, we can see, for the number of individuals about whom you have information is 304.

A. That's right, yes.

Q. But when you are asked if you can supply information about dates of transfusion, you tell us in answer number 2, that's not something that you hold.

A. No.

Q. Indeed, we have heard some evidence from Dr Gillon about that and he is no doubt in a better position to provide that information.

THE CHAIRMAN: Could I ask a question at this stage about language?

In your answer to question 1 you say that in the cases involved blood transfusion should only be regarded as a possible and not a definite or confirmed route of

acquisition. I'm a bit concerned about the extremes since I would have thought that a possibility was a hypothesis that couldn't be excluded. At the other end you have got something from which all alternatives have been excluded. Are we talking about possibilities here or probabilities?

A. Sir, are you talking about greater than 50 per cent in this respect? Probability being greater than a 50 per cent chance?

THE CHAIRMAN: Yes. Take that as a test.

A. I really don't know.

THE CHAIRMAN: Possibility was not much good to anyone since it simply means that it is the hypothesis that you can't exclude.

A. Do you know, I think that, because there is so much uncertainty about these cases, I mean, what we are doing here is just taking some information that has been recorded on a request form. We did not seek additional information. We didn't clarify whether indeed that information provided was accurate. So that was why the word "possible" was provided here.

If I had to put money on it, I would say that less than 50 per cent of the 304 contracted their HCV through blood transfusion.

THE CHAIRMAN: That's perhaps a much more valuable approach

for me because that means I take it that looking at the totality of the information available, you are in a position to offer a judgment as to the prevalence among this group.

A. Partly based on additional information that we have about these individuals, their age at the time of diagnosis, for example. But it is a difficult area and, as I say, I would put money on it. I don't know how much money I would put on it.

THE CHAIRMAN: We don't have the mechanism to test the odds on that approach. The other thing that I would like to ask, just before we leave the material that you have been dealing with, is just exactly what is meant over time by "viral hepatitis". Because I think that there may have been a period at the beginning of the reference period when, in the minds of many clinicians, viral hepatitis and jaundice were not seriously distinguished.

A. I think it's a fair point because I remember even at the time in Ruchill Hospital we had admissions in the mid-1980s of individuals with jaundice and abdominal pain. They clearly clinically had hepatitis. They were then tested for Hepatitis A, negative, Hepatitis B negative; and in the case notes it was recorded as non-A non-B hepatitis. I think the other thing to point out was that other possible causes of hepatitis were

excluded in this respect.

THE CHAIRMAN: Cytomegalovirus and Epstein-Barr or others?

A. I think sometimes -- whether they had a full viral screen is difficult to remember, but there were no other clinical indications that they had these viruses. There was no indication that the hepatitis could be caused by a drug, a medication, which is often a common cause of hepatitis. There were no other causes of hepatitis which might have explained the condition.

So you were left with hepatitis from unknown causes but having said that, the individual probably had injected drugs and that information, coupled with the fact that there was no other explanation, led to a diagnosis of non-A non-B hepatitis, which was probably correct in most instances.

THE CHAIRMAN: My problem at the end of the day, professor, will be to decide what inferences I can draw from the totality of the information I get and perhaps the more one undermines the foundations, the less likely it becomes that there will be much reliable information that can be used. Where do I stand with your 304 individuals?

A. I think as I said to you, to colleagues in CLO, I couldn't answer this particular question in the first place. You asked how many were known to have contracted

Hepatitis C as a result of blood transfusion and as I say, all we can do is provide you with what we have. You have asked me to estimate what proportion of the 304 I think actually acquired their infection through blood transfusion. I really don't know the answer to that question but when pressed, I would say in my judgment, from looking at other information available to me, that we are in the less than 50 per cent category.

THE CHAIRMAN: Thank you.

MS DUNLOP: Sir, it is probably only fair to put up at this point the notes to which Professor Goldberg refers -- at least I think they are the right notes -- which appear at the end of the other statement. Again, if we could have these side by side. If we go to PEN0010212 at the end of the statement ... (Pause)

THE CHAIRMAN: I think while this is being looked for, Ms Dunlop, it occurs to me that it might be a great advantage to me, and perhaps to others, to have hard copies of some of this material because looking from page to page on screen is fine when the pages are there but they tend to escape. If there is anyone else who would benefit from having a hard copy, if they would let us know now, we can perhaps get them all at once.

MS DUNLOP: Yes, by all means, sir.

It was just, professor, that I think this paragraph

really should be read along with your answer to question 1. That's really a summary of the reservations that you have expressed in your evidence, I think, isn't it?

A. Indeed, yes.

Q. So you tell us in short, in the middle of that paragraph, that:

"The lack of confirmation associated with blood transfusion is a weakness in the system. However, it is not much better in other places."

A. Yes. I mean, I'm pretty confident that the information that Scotland has on Hepatitis C is as good as, if not better than anywhere else in the world.

Q. Thank you.

THE CHAIRMAN: I'm tempted to say that if all the apples in the barrel are bad, it doesn't help one to find good fruit.

A. I think, that comment -- I wasn't being in any way flippant there. I would hold by that statement. I think there are weaknesses in our information base with respect to blood transfusion and blood factor, but in general our information about Hepatitis C is pretty good. If you want to compare our diagnostic information, ie numbers of people known to be infected, with the information available in England, then we are in a far superior position in terms of the completeness

of our data and indeed its accuracy. That probably doesn't help matters.

THE CHAIRMAN: No. I look forward to the April elucidation of all of this.

MS DUNLOP: I think the April exercise will be the other kind of exercise, as I understand it. It will be a modelling exercise rather than an attempt to count heads, as it were.

A. It will be a modelling exercise and it will be done in the context of the time that we have available to do it.

Q. Yes. We will come on to that kind of exercise in a minute. Just now we are still at the counting of heads.

A. Indeed.

Q. I mean, perhaps one can say that the fact that there are difficulties in other countries may reflect the inherent difficulties in the issue.

A. I think there is also one other point to make and that is that much of the data that we collect, it's about preventing future infection and disease. So when we started this exercise in 1996, ie the creation of the diagnosis database, I think it was felt that, as far as blood transfusion is concerned -- and indeed blood factor is concerned -- these were not serious issues in relationship to preventing further transmissions. But

there were other areas which were very much more important and indeed are still very important.

We still estimate that around about 1,000 to 1,500 people are becoming infected every year with Hepatitis C in Scotland. So a great bulk of our energies are focused on the behaviours associated with transmission of HCV in that respect.

Q. Yes. By far the most common means of acquiring Hepatitis C, as I understand it, is by injecting drug use.

A. Absolutely.

Q. That has been so really for as long as the virus has been identified?

A. In this country but not in every country.

Q. Right. Let's stick to Scotland just now.

A. Sure.

Q. That statement we should just look at. You have said that in relation to those 304 people -- that is answer 4:

"219 of them are not known to be dead as at December 2009."

Then if you turn the page, again at that point, 85 of the 304 cases were known to have died.

A. Yes.

Q. "Of these, 18 had a primary liver-related cause of death

on the death certificate and 13 had a secondary liver-related cause of death. It is not possible to conclude from this information alone if Hepatitis C materially contributed to death in these instances."

Professor, we looked this morning at a different cohort of people. 53 people who have all died and who are all known to have had Hepatitis C. It was interesting to see that hepatitis was only recorded on the death certificates of 14 of them. So in very broad outline, this seems to be a similar sort of picture but I think you are not yourself enthusiastic about using death certificates as a reliable guide. Is that fair?

A. That is fair. It's not a reliable guide and that's one of the reasons why we use the Hepatitis C diagnosis database in association with the death register, to identify individuals with Hepatitis C who have died, rather than going straight to the death certification register and just relying on that source of information. It just is completely unreliable in that respect.

Q. Certainly I think this is a vivid illustration of the degree of underreporting we would have encountered if we had simply tried to count the number of people who had it on their death certificate. That looks from these two exercises as though that would have been really quite unreliable.

Can I ask you to look at the --

THE CHAIRMAN: I don't think you got an answer.

MS DUNLOP: I think there was a nod.

THE CHAIRMAN: I think the point is that simply if the death certificates were the only sort of information, the sum total produced would be a gross underestimate.

A. It would be a gross underestimate and that's why having the diagnosis database is so important in this respect. Very few other countries in the world have a diagnosis database and even if they do have one, it is very difficult for them to do this type of linkage exercise. We are able to do that and therefore are able to identify the number of individuals known to be infected with Hepatitis C and who have also died, but not necessarily as a result of Hepatitis C. That's where we get into difficulties. What is the actual cause of death in those individuals can be difficult.

MS DUNLOP: Yes. I suppose, in theory, there is the possibility of error in the other direction, but that perhaps is more theoretical than anything else, that somebody could have Hepatitis C on their death certificate but for whatever reason they have never been reported to you in their life as having hepatitis. But that's presumably a very small number.

A. It's a very small number and indeed we don't collect

data on individuals who are diagnosed in the private sector. It is a small number of individuals who will be diagnosed with Hepatitis C in the private sector. We think it is a small number. So these individuals would not be included on our database.

Q. We should look at your tables which follow on on the next page. I'm hoping we will have hard copies. I think it must be [PEN0130016] that is the tables. Possibly not actually, because I can see it says "2 of 2". [PEN0130016] might be it. No?

THE CHAIRMAN: Which tables are these?

MS DUNLOP: There are three tables. [PEN0130024]. This actually goes with your statement, I think, professor, doesn't it?

THE CHAIRMAN: This is not ...

MS DUNLOP: No, well, it is just a breakdown of the 304 really, isn't it?

THE CHAIRMAN: All right. It is on one page and we do have the hard copies of the other materials so we can cross-refer.

MS DUNLOP: Just to look very quickly at it, professor. Perhaps unsurprisingly the greater number of people are on the Greater Glasgow and Clyde Health Board area, followed by the Lothian Health Board area. Then Grampian, Grampian Highland, Lanarkshire and Tayside all

quite similar and then a big drop. Then you have given the deaths, the 219 and the 85, then the figure that you have also included in the text about the causes of death. Then I was completely thrown by the column at the bottom that says "cumulative per cent", but you have told me that I can completely ignore that.

A. Yes.

Q. It seems to have got on by some mechanism that we are not very sure about. I'm glad to learn I don't have to try to understand it.

The other statement is [PEN0010206]. This is your statement in relation to the number of haemophilia patients infected with Hepatitis C. We can see that initially you were asked to do this by Haemophilia Centre, which you were not able to do, but you could do it according to NHS board of residence or the board of the source of the original specimen. You are aware of a total of 351 individuals who have received blood factor and have been diagnosed as Hepatitis C antibody positive. For all those 351 people, there was no information to indicate that the blood factor was received outside Scotland.

I'm hoping that the table is the next page, which will be PEN0010207. Yes, it is.

There is a slight difference, professor, from the

figure that was on your website for the period to 30 September 2010. There seems to have been some adjustment of the figure since then. Is that something you know about? Presumably the refinement of the accuracy of certain figures.

A. Yes, the figure you are making reference to was -- I don't have that in front of me.

Q. Right. It was just that, for example, the table up to last September, the figure for Lothian was 77 not 71. I mean, it is quite a small changes but I just wondered if you had been doing some work on the figures in recent months.

A. What I have in front of me -- Laura, are you saying that the figure that I provided in the witness statement is different from another figure?

Q. Well, it was just that on the Health Protection Scotland website, as it happened, there had been a slightly higher figure and it was 361 a few months ago. That was all. It just made me wonder if there had perhaps been some further investigation.

A. I see. I think again it is going back to the original question. I mean, we have excluded individuals for whom information indicated that treatment, ie blood factor treatment, was received outside of Scotland. So that will explain the differential between the 361 and the

351.

THE CHAIRMAN: I wonder if I can help. Professor James suggests that we look at the reference date, that what appears in this table is correct as at December 2009.

PROFESSOR JAMES: What Ms Dunlop referred to was a website dated towards the end of 2010.

MS DUNLOP: Yes, I'm obliged.

A. The 351 is up-to-date. The December 2009 date relates to dead. The 351 is up-to-date and applies to individuals for whom there is no information to indicate that they received blood factor outside Scotland. The 361 includes ten individuals, okay, for whom that applies. That's my reading of the situation.

THE CHAIRMAN: It is not that some people have died and disappeared off the list.

A. No, the deaths are a separate entity.

THE CHAIRMAN: A separate entity.

MS DUNLOP: Right. And you have also broken down the information according to the different reported blood disorder that each individual had. Then 3, the year of the earliest specimen positive for Hep C antibody. We can see, completely unsurprisingly, that there is a big group in 1991 in association with, obviously, the availability of a test.

Then the information is shown in the same manner in

these tables as in the previous statement about the number of people who are known to be dead and then the causes of death. You have also repeated that in the text, of the 351 people, 78 of them were known to have died by December 2009; then in relation to those, 15 had a primary cause of death which was liver-related, 15 had an secondary cause which was liver-related and the rest, at least on the face of it, didn't. You referred to the ICD9 and ICD10 codes. That's the international classification of diseases, is it?

A. That's correct.

Q. Right. Where does the international classification of diseases come from? Is that an American publication or is it truly international?

A. I think it is World Health Organisation.

Q. Thank you. I was trying to remember. I think there is one system which is an American one and one which is WHO.

A. I can't be absolutely certain about what I have just said there but certainly WHO are involved in the whole process.

Q. I also wanted to ask you about the work that was carried out by Kate Soldan and others. Dr Soldan, I think, is an epidemiologist based at CDSC in London. Is that correct?

A. That is correct, yes.
Q. Right. Just in a nutshell: she researched the number of transfusion transmitted Hepatitis C infections in England and then she was asked to do a similar sort of modelling exercise for Scotland. I think she was asked by Dr Brian McClelland. Is that right?
A. That's right. I mean, I'm looking at a letter from 2002.
Q. We should have that. That's [SGH0057201]. Is that the letter you are meaning?
A. Yes.
Q. The one that says:
"Dear Bob ..."?
A. Yes, that's right.
Q. And this was all done in connection with Lord Ross's enquiry into the extent of the problem in Scotland.
A. Hm-mm.
Q. There was an attempt made to gather some data and that's the context in which Dr Soldan was approached. Sorry, did you want to draw our attention to the letter?
A. I was just making that comment because I think that's the only reference to Kate Soldan I actually have in my notes.
Q. Actually the work relating to Scotland is appended to that letter and we see it at [SGH0057203]. Her name is

on SGH0057204. I didn't want not to go to the main publication, professor, so even though it is late in the day I would ask you to look at her principal article and she refers to it on [SGH0057203]. Do you see at the top she says:

"Estimates of the contribution of transfusion to HCV infection in England have been made and are in press [with Epidemiology and Infection]."

That article was duly published. It is entitled "The contribution of transfusion to HCV infection in England". We have that at [PEN0131580]. There are some hard copies of it as well, sir. I don't know if you would want to have one. Would that help? We have some extra ones. (Handed)

THE CHAIRMAN: If it's a pre-condition that I'm convinced it will help. I'm not sure that I shouldn't resist. I will have a look. I have seen it before.

MS DUNLOP: I just thought it was easier to understand the Scottish exercise if you looked at and even, on a good day, understood the English exercise as well. I mean, I think as we said when we were discussing this before lunch, if we look at the chart, just two pages on, I certainly find the middle column perhaps the least difficult. The right-hand column is slightly more difficult and the one on the left is really quite hard

to follow, but if we can just look at it and hopefully not spend too long on it.

Dr Soldan appears to have been the coordinator of quite a large group of people conducting this exercise and she tells us in the text that a large number of people from different blood centres in England supplied data. She was able to get data for eight blood centres which had handled 80 per cent of all blood components which had entered the look-back programme in England. So she had information from them and then in fact she had information on all tested recipients as well.

This is, I guess, the way an epidemiologist works, is it, this kind of chart? Not all the time. It is one of the things an epidemiologist can do. Is that accurate?

A. I suppose so, yes. It is a fairly standard, I think, approach to try and make things as easy as possible to understand. A flow diagram.

Q. Yes. The word "path" is used a lot in the article. I suppose "path" is slightly confusing because if you are on a path you can both join it and leave it whereas you have used the word "flow". I wonder if it is better to try and think of this as a flow, because certainly people do leave the flow but nobody joins the flow.

I suppose there is one group of people who join the

flow but generally you do not join the flow half way down. But just look at it, if we start in the middle at the top, "The observed path of components that entered the look-back programme and resulted in known Hepatitis C status of recipients," she tells us firstly that there were 9,222 components which entered the look-back programme. We have already heard about the look-back programme. It was UK-wide but this is in relation to England. Components are the different parts that can be used from a donation of blood.

Then the first group which leave the flow, the 2,119, these are components whose fate is not traced. Of the remaining components, 4,586 were transfused, but -- and again, some are leaving the flow to the right -- 154 of the components which were transfused didn't have an identified recipient. 2,711 of the recipients had died and then there is another group of people who were eight, who were known to have been infected. I take it from the way it is written that they are dead as well.

So that reduces the number quite a bit. There were 1,713, if we go back to the middle of the flow, 1,713 identified recipients, assumed to be alive. Of that number 1,062 were tested, 651 weren't. I should say that every so often she has to scale up her number

because she only had 80 per cent of the components, data for 80 per cent of the components. So she scales it up to 100 per cent to give her a more accurate number, a more complete number.

Then, of those who were tested -- and at this point 271, who come from the other 20 per cent of the components -- 271 tested recipients join in. They are in italics and the total number found to be infected was 677.

A. Hm-mm.

Q. So 677 infections were identified during the look-back. Then if we look on the right-hand side, she has charted all the components which dropped out of the flow for whatever reason and worked out a number for the likely infections from those. Then finally the exercise on the left-hand side, as I understand it, is that she had to come up with a figure for the HCV-positive components which would have entered the system, as it were, between 1980 and 1991 but which had not come to light in the look-back exercise.

A. Hm-mm.

Q. So she works out -- and this is really pretty complicated, and I find it difficult, so we will not bother going into it -- a figure of 19,525 for the additional components entering the system. Then she

applies the same percentages that she has established from the middle column to that figure as well.

A. Hm-mm.

THE CHAIRMAN: 19,000 -- you have lost me at that point. Up there, I see, yes.

MS DUNLOP: Yes. It is at the top on the left-hand side.

THE CHAIRMAN: Let me see whether I do understand this. I'm not sure that the persons and the figures are always necessarily presented in the same way but if we look at the top of the middle column, 9,222 is an absolute number, which represents 80 per cent of the total components entering the look-back. Is that right?

A. Hm-mm.

THE CHAIRMAN: Then 2,119 components had a fate that wasn't traced.

A. Hm-mm.

THE CHAIRMAN: If we look below that and find that 7,103 is the difference between 9,222 and 2,119, so that at that point she has not grossed up to 100 per cent. I'm not quite sure why. We then get to the next stage, 65 per cent of the 7,103 -- one full figure and one not grossed up -- is carried down to the next line, where there are two alternative blocks to the right, and where, regretably, I can't get the arithmetic work. Because if I take 7,103 and subtract 2,649, the

grossed-up figure, I get to 4,454 and not 4,424. Which is the figure she has used. Then it goes down.

I regret, at that at the moment, I can't quite follow in detail the logic of the presentation. It may be me simply not getting it right, professor. Can you help me understand?

A. Certainly. I have been put on the spot here because I knew of the existence of this paper and I was given a copy about just over an hour ago. So what I would have to do is go through it, preferably with Kate, because I'm seeing Kate next week. The reason I'm seeing Kate next week is to help me generate the Scottish estimates that you want for the period 1970 to 1991, and of course this period that we are talking about just now is 1980 to 1991.

So, sir, if you would like me to spend some time sort of adding things up just now and also sort of going back to the text, I can do that, but I'm sorry, I'm not in a position to --

THE CHAIRMAN: I can assure you I don't want you to do the arithmetic. It happens to be something I tend to do and play with the arithmetic. I'm much more interested in the process. It looks to me as if she starts with a gross number of people and then adjusts that number down for a series or sequence of factors that she can

identify as affecting the general development of her thesis. Some components' fate was not traced. Exactly what the arithmetic is is less important than knowing that that's why she discounts the figure for that. Then we have unidentified recipients and she discounts her exercise for that. Then there are people who are not tested and they are taken out of the loop, and finally we get down to 677 infections identified, of whom 1 per cent are known to be dead.

What interests me is that if one has once done that and progressively reduced the relevant focus for enquiry into a relatively small number of people, what's the next step? And how do you get up to a much larger figure, for example at the top of the left-hand column? What's the significance of the reduced figure in the overall picture, when it has been reduced by so many factors to such an extent? Sorry, that's rather a complex point, I suppose. But what can one make of this?

A. I mean, what she has done here is she has got some pretty solid data on the 9,222 and then she has extrapolated the findings to those components for which she doesn't have solid data, and I think that's a reasonable thing to do.

I'm not convinced this is the only way to estimate

the size of the infected population. I think there are other ways of doing it. As I explained just before coming in here, if you use a combination of approaches, then you do reduce uncertainty. But that all takes time and much, of course, is dependent on the information that's available to you. So for Scotland we have information generated through the look-back, but we also have other information about the size of the infected population, ie Scottish population, during the 1980s but also during the 1970s as well. So I would expect to use these data.

I have probably not answered your question properly but I hope what I have just provided you with is of relevance.

THE CHAIRMAN: Perhaps I can put it quite briefly. We start off with 9,222 pieces of hard information, pieces of hard data. By the end of the exercise, 73 per cent of those cases have disappeared because of a number of factors which mean that they can no longer be taken into account.

What is the confidence one can have in inferences drawn from the 27 per cent that are left?

A. I'm just looking through this chart just now to try and answer that question. I mean, I think the information is pretty solid in that -- for example, you have

infected components but not all of these have been transfused. You have to eliminate, obviously, individuals who have died. You then test the 1,713 living individuals who received components, so you test two thirds of those individuals.

The question is: is there bias here? Are the 651 individuals who were not tested more likely or less likely to be infected?

THE CHAIRMAN: That involves a stage beyond what I would want to pause at. If one reaches the stage at which 677 effective tests are available, and 651 persons who would qualify for testing have not been tested, you are getting down pretty well to 50 per cent of the relevant population at that stage. Indeed, if we gross the 651 up to the full 100 per cent, more than 50 per cent of those who would have qualified, as it were, who would have been within the class for testing, have not been tested.

I don't know, I'm not a statistician. I don't know what the statistical validity of a percentage of this kind might be, but I think it is legitimate for me to ask you what confidence one can have in extrapolations based on this about the general population.

A. For me it is the 651 recipients who were not tested. So they were alive and they were not tested. The other

factor, of course, is the 2,711, those who had died further up, were they more likely to be infected. After all they had died and so Hepatitis C may have been a contributing factor in those instances.

I know what you are driving at: the potential bias, all the way through here. I think there clearly must have been reasons why, for example, of the individuals who were alive, 651 were not tested. What were the reasons for them not being tested? I presume in most instances it was just failure to get access to them. One possibility is refusal. Why would somebody refuse? Would that make them more likely to be infected or less likely to be infected? I don't know if you can make any assumption, other than there is potential bias in the system.

THE CHAIRMAN: I think I could almost forecast that a point Mr Dawson will want to ask you about is the net result of this, that this is a minimum number.

A. The 651?

THE CHAIRMAN: Not the 651, the 677 that one ends up with.

A. Absolutely. It is a minimum number and that's why the overall estimated number is 13,500. But the thing is that the extrapolations appear to be based on the middle column and the question is: can you extrapolate? Because there may well be biases in the system which

mean that the numbers infected, or the expected numbers infected may be an underestimate or an overestimate.

I don't know if this is discussed by Kate in the paper but I would be surprised if it wasn't.

MS DUNLOP: I don't know if it helps, sir, to note that she says:

"We may have underestimated or overestimated the infections --

THE CHAIRMAN: I noticed that. I'm not sure it helps at all.

MS DUNLOP: It certainly shows she is frank.

THE CHAIRMAN: Mea culpa, mea culpa, mea maxima culpa is frank. It doesn't necessarily enlighten one.

I don't want to play this down at all. It looks like a serious exercise carried out in a very difficult area in the absence of hard data, but somehow or other, professor, I have to try to get a measure of numbers. There is a great danger in taking a figure that may be fundamentally flawed. From my point of view it may be that one just has to have a general estimate and perhaps you can help us as best you can for Scotland, but not go beyond that. I don't know.

MS DUNLOP: The main problem perhaps, professor, is that out of the limited group she did have, the people who made it to the end of the flow, certain results have been

obtained and then, no doubt because there isn't any better way, after that the team applied the findings from the group of people who made it to the end to the people who dropped out. As you have said, that may not be valid because of the people who dropped out, there may have been more Hepatitis C in those groups or, I suppose, less Hepatitis C for particular reasons.

So you are starting with a disadvantage because you are assuming that, as his Lordship would say, the people are really homogeneous. So if you were able to get the people who dropped out, they would produce the same kind of results if you tested them and so on, and that may not be right. Then the problem is no doubt magnified when you try to work out the much bigger number, which is the components that didn't get into the look-back exercise in the first place --

A. Hm-mm.

Q. -- and apply the same assumptions to them because there might again be all sorts of reasons why these assumptions are not very accurate. But perhaps there isn't really any other data that the team could have used.

A. I think that's why I was saying that, you know, if you just use one method, you are opening yourself up a little. I mean, you know, this is actually a very

good piece of work undertaken by Kate, who probably knows more about this field than anybody else in the UK. But it does have its limitations and I think we just have to acknowledge these limitations.

Q. Yes.

A. By and large, when you are doing this sort of work, if you use maybe two or three methods -- but much depends of course on the information you have available to you -- I think your confidence in your final outcome is very much greater because if you have considerable differences in your results, you can get an average or you can take what's regarded as the best or whatever. That, I think, would help you, sir, in feeling confident that one actually had the best estimate possible.

THE CHAIRMAN: I just introduce a word of caution at that. I understand that. If there is variation in the basic data that is used, but if the basic data happens to be common to all three methods and particular numbers are dominant, then it may be that simply having different approaches to analysis should not give one greater confidence.

A. I think that's right. But it may be that, you know, you are using different data sources. So in this instance we are just using look-back data but there are potentially other sources of information that one might

use.

PROFESSOR JAMES: Lord Penrose, can I point one other thing out? Would you mind?

THE CHAIRMAN: No, anything that helps.

PROFESSOR JAMES: Professor Goldberg, of all the 13,500, the big, big number at the bottom, the biggest number comes from the left-hand column, the 9,455 infections due to components not entering the look-back. That is based on the assumption that the observed prevalence of anti-HCV during the first four months of donor testing, in England 0.066 per cent existed from 1980 through to 1991; in other words, for every year. Then they have extrapolated against the number of units of blood transfused over that decade, 25 million donations, et cetera, et cetera, and made some extrapolations. So that's just another set of assumptions --

A. Absolutely.

PROFESSOR JAMES: -- to add to the mix and it does actually produce the biggest number, if you see what I mean. No doubt you will be examining that number, particularly since the Inquiry has asked you to look at 1970 to 1980, which is even further away from being able or justified to make an assumption on a prevalence made in 1991, but you will be looking at that with various caveats in order to help the Inquiry as best you can.

A. We will certainly do our best. What you can do is you can do what's called a sensitivity analysis, which means that you can vary various assumptions to see what impact that variation actually has.

So you might generate a range of estimates. As I say, this has not been done in this particular instance. I mean, I suppose as far as Scotland is concerned, if we are going to do this work, these things do take time and my understanding is that you want something as soon as possible, clearly, and so the complexity of the work that we undertake has to be considered in the context of the time available to do the work.

THE CHAIRMAN: Professor, I think you should take it that you have made one important point today, as far as I'm concerned, and that is that your primary focus is on those who are infected now and how matters can best be adjusted to protect people in the future.

You may take it that I don't want to see this becoming a massive statistical exercise that may end up with less confidence in it than your own judgment can provide. I think that one is entitled to look to a practitioner in the field, like yourself, and ask for your best estimate, making use of such data as you have and such methods as you think are right. For my part,

although others may have a different view, I will not be disappointed if I get your best judgment.

A. Thank you. Thank you, sir.

MS DUNLOP: Professor Goldberg, I just wanted to go back to [SGH0057203]. We looked at this before. This is Dr Soldan's short paper on the exercise she did for Scotland. If we look at that table in the middle. We can see that she did the same thing. She had the middle column and that's the 106 people. So she started --

THE CHAIRMAN: Sorry, Ms Dunlop, before you get into it, I understand that we really must give the stenographer a break. We should stop at that point briefly. I should also say that I have a commitment at half past four, which is not inelastic but I wouldn't want to stay too much beyond that.

MS DUNLOP: Thank you.

(3.46 pm)

(Short break)

(4.01 pm)

MS DUNLOP: Can we just have [SGH0057203] again, please? This is Dr Soldan's exercise for Scotland. Just to look at the table, if we might, for a moment. In short she did the same exercise but with Scottish data. So the top row in the table -- that is the row that's described as identified by the HCV look-back programme

in Scotland -- that would be the middle column in Dr Soldan's paper.

So that's all the people who made it to the end, as I said a short time ago. Then the next row is people who received components that entered the look-back but did not receive testing in that programme. That's the column on the right-hand side of her chart. So the components that dropped out of the flow along the way, for whatever reason.

Then the last row, those who received components issued between 1980 and 1991 is the column on the left-hand side. So the estimate that one has to prepare for the components that didn't enter the look-back programme. In other words, from donors who were positive but who were not picked up via look-back. The figures that Dr Soldan was able to use were the actual figures that had been ascertained in the Scottish look-back. So, for example, the unidentified recipients, the people who were dead, the people who had declined testing, the people whose test results were insufficient and so on. They could be used for Scotland and they didn't have to use the English figures.

The only thing that was different, or the figure that was different for Scotland, which she used, was the prevalence in the donors. We should just go back to

remind ourselves that the prevalence among donors in the first four months of testing in Scotland was not 0.066 per cent, as it was in England and as Dr Soldan used, but 0.088 per cent. We find that in the preliminary report, if we can go back to chapter 9, please.

If we can go to chapter 9 -- are you better with the paragraph number or the page number? Again starting at the back, we are going to page 318.

Paragraph 9.285. This is Dr Crawford reporting that in the first six months of testing in Scotland -- so between September 1991 and February 1992 -- donors were tested and the prevalence rate amongst Scottish blood donors was 0.088 per cent. The reference for that is given in footnote 332.

So we have looked at all of that material, really, professor, just to try to get in very broad terms an understanding of the sort of exercise that Kate Soldan did for Scotland and how she produced the figure she produced. We can see the conclusion of her table. She got, in round terms, 3,500 for the total number of infections, and she says again in the note at the bottom of -- sorry, this is going back to [SGH0057203]:

"Many assumptions were used, some of uncertain validity."

Do you see that?

A. Hm-mm.

Q. There has been another figure mentioned recently, professor. You may know nothing about this and that is no reflection on you, certainly, but in the recent review conducted on the instructions of the Department of Health, the review of the support available for individuals infected with Hepatitis C and/or HIV, a figure is given for the whole of the United Kingdom for the period 1970 to 1991 of 28,043 infections.

The thing about that is that from the Soldan paper she had 13,500 for the 1980 to 1991 period. I'm not going to go back to it but she added on 10,000 for the decade of the 1970s. So that paper would be 23,500, and that in the Department of Health paper it says that the 28,000 figure has been arrived at by "correcting Dr Soldan's paper for the UK." But there is no explanation of how they got their additional figure. In fact, if it is only the addition of 4,500-odd for Wales, Scotland and Northern Ireland, that looks quite low.

A. Certainly in the context of that 3,498 for Scotland for the 11-year period.

Q. You haven't been involved in any work leading to the Department of Health's figure then?

A. No, nor was I or any colleague in HPS involved in this

piece of work, the 7203 work that Kate Soldan did. That's one of the reasons why I'm seeing her on Tuesday.

Q. Yes. Perhaps she might be able to enlighten you as to how this most recent figure, which purports to be a figure for the whole United Kingdom, was obtained as far as Scotland is concerned because that's a bit of a poser when you read that report?

A. Absolutely. I think your calculation is a very reasonable one. There really is some inconsistency there.

Q. Yes.

A. Certainly the prevalence of 0.088 versus the English prevalence of, was it, 0.066 is consistent with our estimates for HCV in the general population in Scotland versus England. I mean, in Scotland it is estimated there are probably about 30 to 50 per cent more infected individuals per head of population, ie the prevalence is about 30 to 50 per cent greater, and that is, as I say, consistent with that difference that you observe for prevalence among blood donors.

Q. Yes. We know, sir, that you yourself have looked at this report and it just seemed another possible source of statistics, but the only reference which is given for that 28,000 figure for the whole UK is the 2002 Soldan article, at which we have just been looking, which is

described as having been corrected to the UK but there is no explanation of how.

THE CHAIRMAN: I have a strong suspicion that "corrected" should be expressed as "adjusted" and it may be no more than a crude accumulation of estimates but one can't tell.

MS DUNLOP: It is just another figure, sir, at the end of the day. Another figure which the Inquiry has obtained is that there have been 636 payments from the Skipton fund to people in Scotland and that's obviously not inconsistent with the lower range of figures that have been described at points today.

THE CHAIRMAN: I'm suggesting to Professor James that if one tried to do a stochastic projection of the range of values here, the vector would be nearer to that of a blunderbuss than a shotgun, professor, really, the variables are become becoming so wide.

A. I think that's fair comment.

MS DUNLOP: I don't have any more questions for Professor Goldberg. Thank you, professor.

THE CHAIRMAN: Mr Dawson, are you asking again?

MR DAWSON: Yes.

Questions by MR DAWSON

MR DAWSON: Thank you, sir. I have a few questions. Professor, as I had you noted in connection with the

issue of notifiable disease legislation, your position, as I understand it, was that under certain provisions of that legislation information would be communicated to Health Protection Scotland but that you considered that information to be broadly unreliable. Is that your position and if it is your position, could you explain why that source of information is unreliable?

A. Yes, I mean imported directly to Health Protection Scotland from 2007 onwards but prior to that it was ISD. Unreliable for two reasons. One was that the numbers of individuals with viral hepatitis, unspecified, pretty meaningless to us, and then the number of individuals actually being reported by clinicians bore no relationship to the actual data we were getting from the laboratories. I'm not saying our laboratory data are 100 per cent complete but certainly over 90 per cent complete and so our feeling was that we weren't being just dismissive, recklessly dismissive, in this respect, we were basically saying, "Look, the information we are getting from the laboratories is fairly accurate," and the information that we would have got from the notification system would not have helped in any way at all; it would have actually hindered us in our understanding of the epidemiology of this infection.

Q. Thank you. In your paper on transfusion-transmitted

Hepatitis C you give a figure, which you have discussed in detail, of 304 individuals. That number comes from a number of people in connection with whom reports have been made to Health Protection Scotland.

A. That's right.

Q. And the reason why reports are made to Health Protection Scotland in respect of these 304 people is because they have tested positive for Hepatitis C in Scotland. Is that right?

A. That is correct.

Q. And the way in which one connects that positive test with a possible blood transfusion is that blood transfusion is included in the data with which you are provided and it is the only risk factor. Is that correct?

A. It is certainly a risk factor. We did exclude from that figure 51 individuals for whom injecting drug use was also recorded on the request form because what we were trying to do was get as near to answering the question as possible, ie contracted Hepatitis C as a result of blood transfusion. So our view was that if you had on your request form, "Blood transfusion and injecting drug use," it is much more likely that you would have been infected through injecting drug use behaviour because we knew, certainly in the 1980s, that in most centres

between 80 and 90 per cent of injectors were infected with Hepatitis C. It was a very, very high percentage. But that 304, I suppose, could be increased to 355 if you include those individuals who also have the risk factor injecting drug use as well.

Q. In compiling this statistic, was information available to you as to where these individuals had received their potentially infecting blood transfusions?

A. Our position is that if information indicated that they had received their blood transfusion outside of Scotland, then we excluded them from this particular figure. So often you would find on the request form a person from, I don't know, Zambia, blood transfusion from Zambia. We would exclude that individual from that figure. In total we have 49 individuals for whom there is information indicating, strongly indicating, that the transfusion was received outside Scotland. Of course, that doesn't mean to say that for the 304 individuals they definitely received their transfusion within Scotland; there just was no information indicating otherwise.

Q. So it is an assumption you made --

A. It is really just an assumption because a clinician would only tend to include information if they thought it was relevant, if it was unusual. So, for example, if

they put on the request form, "Blood transfusion," and the person had the blood transfusion in Scotland, they wouldn't say, "In Scotland", they would just say, "Blood transfusion." It would be probably assumed by the clinician that that had taken place inside the country. So I think the problem here is that the absence of information, of course, doesn't mean that in this instance blood transfusion didn't occur outside Scotland.

Q. As I understood your evidence, you expressed the view that you thought that perhaps less than 50 per cent of the 304 actually did get their infections through the transfusion. What was the reason for you expressing that view?

A. I think I answered the question originally by saying that I just didn't know.

Q. Is it to do with the unreliability of the information which --

A. I think it is to do with that, the unreliability of that information. I think the age distribution is a bit lower than I thought it would be for a blood transfusion recipient group. Having said that, individuals, older individuals, who received blood and were infected through blood transfusion, many of these would have not been diagnosed with Hepatitis C simply because they

would have succumbed to other things.

So that in some ways influences matters as well. But, you know, I was asked a question and I said, "Well, if I was putting money on it, I would lean towards possibility as opposed to probability, less than 50 per cent than to over 50 per cent, but that's just a personal sort of judgment.

You have asked me on what basis. I don't think I have looked at this in enough depth to make a proper judgment in this respect but I'm not sure if in fact we have sufficient data to actually make a judgment, and so my actual answer of, "I don't know," I think is probably the correct one.

Q. At the end of your first report, more accurately described as the document at [PEN0010206], the front page PEN0010210, you explain the methodologies that are applied to the compilation of data, first of all in connection with HIV, which I don't think you have been asked to give any information about, but, secondly, perhaps more relevantly for the matters upon which you have given your views on Hepatitis C from the following page.

I just wanted to ask you: my understanding in relation to HIV is that the information with which Health Protection Scotland is provided is likely to be

more comprehensive than the information with which Health Protection Scotland might be provided on the issue of Hepatitis C. I understand that that's principally because of the fact that there is a specific form which asks specific questions about HIV, whereas no such specific form exists in relation to Hepatitis C. Is that an accurate understanding?

A. I think that's fair. We introduced the HIV dedicated HIV test request form in 1989 and so this is really just a prompt list for clinicians requesting a test. So you are absolutely right that the information obtained for HIV is likely to be more reliable than that for Hepatitis C. We did make some attempts to try and get a dedicated Hepatitis C request form but there were obstacles and we didn't manage to achieve that.

Q. Thank you very much, professor, thank you, sir, I have no more questions.

THE CHAIRMAN: Mr Anderson?

MR ANDERSON: I have no questions.

THE CHAIRMAN: Mr Sheldon?

MR SHELDON: Nor I, thank you.

THE CHAIRMAN: Thank you very much indeed for your help. We look forward to hearing from you again.

A. Thank you.

MS DUNLOP: Tomorrow we are going to hear from

Professor Turner and Dr Norfolk, who are going to talk really about transfusion from -- if this isn't too crude -- a sort of seller and purchaser point of view, the people who collect the blood and how they do that and the systems that they use, and then the various uses to which the blood is put in the therapeutic context.

So that is what we have planned for tomorrow, as much to give us general background which should be useful throughout the whole of the rest of the Inquiry as anything else.

(4.27

(The Inquiry adjourned until 9.30 am the following day)

I N D E X