Witness Name: Professor Charles Richard Morris Hay
Statement No.: WITN3289181
Exhibits: None
Dated: 6/3/2023

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF PROFESSOR CHARLES RICHARD MORRIS HAY

I provide this statement in response to a request under Rule 13 of the Inquiry Rules 2006 dated 22 March 2022 in relation to the criticisms of Witness W0679.

I, Professor Charles Richard Morris Hay, will say as follows: -

Section 1: Introduction

1. Professor Charles Richard Morris Hay MBChB MD FRCP FRCPath
Consultant Haematologist Manchester Royal Infirmary since December 1994.
Director Manchester Adults Haemophilia Comprehensive Care Centre since December 1994
Professor of Haemostasis and Thrombosis.
Senior Lecturer in Haematology Liverpool University and Director Liverpool Haemophilia Centre, Royal Liverpool Hospital 1987-1994.
Director UK National Haemophilia Database since 2002.
Member UK Haemophilia Centre Doctors Organisation (UKHCDO) Regional Committee from 1987 and then Advisory Committee since 2007 (when the committee name changed).
Vice Chairman UKHCDO 1997 to 2005.
Chairman UKHCDO 2005-11.

I have already provided a copy of my Curriculum Vitae to the Inquiry.

2. The Manchester Haemophilia Comprehensive Care Centre (Adults) is based in Manchester Royal Infirmary. This was the third largest haemophilia Centre in the United Kingdom. It is now the second largest with >2500 patients with bleeding disorders registered. When I arrived in December 1994, I was the only consultant specialising in adult Thrombosis and Haemostasis in the North West Region, assisted by a part-time clinical assistant, Dr Monica Bolton. We now have four consultants with this specialism. In 1994, we had three Haemophilia Nurses, one of whom also did counselling and went into the community. There were no clinical research staff. There were no joint clinics and no formal liaison with any other supporting specialism or profession allied to medicine, such as physiotherapy. All the follow-up clinics were conducted in the Haemophilia Centre without any junior staff support. There was no internal training rotation for junior staff so they spent all their time treating leukaemia. I was on call 1:1 i.e. 365 days a year except when away or on holiday.

3. In the first year, I introduced an internal training rotation for junior staff so that we had a registrar attached to thrombosis and haemostasis most of the time. I introduced weekly multidisciplinary meetings and arranged for Physiotherapy input for our patients. I rapidly established joint clinics for Orthopaedics and subsequently joint HIV clinics and joint obstetric clinics and later joint adolescent clinics with the paediatric service. Liaison with Hepatology was close throughout this period but not formalised around a clinic. Our Hepatologist in the late nineties and early 200s was Dr TW Warnes.

4. As we acquired more consultants specialising in Thrombosis and Haemostasis, first in 1999 and then in 2003 and in 2018, the patients were reallocated among the consultants.

5. I prepared this statement with access to incomplete medical records of Witness W0679. I have asked medical records for two further volumes of records. I have access to correspondence from 2003 and laboratory results from the 1980s to date.

6. Witness W0679 was managed initially by Dr Irvine Delamore and subsequently by Dr Richard Wensley, until 1992. He was then managed by Dr Guy Lucas until late 1994. I was his haematologist between December 1994 and 2003, when Dr Bolton-Maggs took over his care. Since 2011, when Dr Bolton-Maggs retired, he has been managed by Dr Jecko Thachil. Since he underwent liver transplantation in late 1998, he has required very little haemophilia care, since liver transplantation normalises the factor VIII level in people with Haemophilia.

7. Witness W0679 has severe haemophilia A and consequently developed severe arthropathy of his knees, ankles and elbows, which required a series of orthopaedic procedures including joint replacement over the years.

8. He also contracted HCV and HBV, (but not HIV), either from the cryoprecipitate administered in his childhood or from factor VIII concentrate. Fortunately, he cleared HCV spontaneously and his virology tests have shown repeatedly that he has been HCV-RNA negative from the point in time at which those tests became available. He was Hepatitis B e-antigen and surface antigen positive, indicating chronic infection with Hepatitis B. Since he has cleared HCV, his liver disease is attributable to HBV alone.

9. His statement makes it clear that I explained this to him on more than one occasion, but he remains confused about the matter and reluctant to accept that he no longer has HCV infection. I think Witness W0679 has inadvertently confused this issue by asking hepatologists whether they could tell which virus was causing his liver disease, if he was co-infected with both. They answered in the negative, presumably accepting that he was coinfected, which he is not. Matters were further confused by our then Clinical Assistant, Dr Monica Bolton, who told him that he had HCV and not HBV, in June 1993. Her statement was probably based on finding that he was HCV antibody positive. This shows that the patient has been exposed to HCV at some stage in the past but not that they have active HCV infection. The HCV-antigen test was not yet available at that time. When it became available, it proved to be

consistently negative in Witness W0679's case, showing that he had cleared the hepatitis C virus completely.

10. It was clear when I first saw him in early 1995 that he had established cirrhosis of the liver with some degree of liver failure secondary to chronic HBV contracted from blood products. I referred him for joint management with hepatology in early 1996. He had apparently been stable for some time but was beginning to decompensate. From that point onwards he was joint managed by Dr TW Warnes, Consultant Hepatologist. In 1996, he started to develop ascites. By 1998 he was becoming jaundiced and his hepatic decompensation was gathering pace.

11. I discussed the possibility of liver transplantation with Dr Warnes. Initially, Dr Warnes felt that it was too early to consider this, but it was clear that Witness W0679's liver failure was gathering pace and I was aware that if it is left too late the patient has less chance of surviving the transplant, and so I pressed hepatology to refer W0679 to a transplant centre. He was referred to the Liver Transplant Centre in Leeds and went over for assessment. He was very ill. Given his poor clinical condition, he was put on an urgent waiting list. In advance of transplantation, HBV viral reduction was attempted with Lamivudine and Interferon. The Interferon was relatively ineffective and was also associated with severe side effects and was therefore discontinued. He was transferred from Manchester Royal Infirmary to Leeds on 7/10/98 and transplanted on 10/10/98. He has done well subsequently and made very satisfactory recovery.

Response to Criticisms of Witness W0679

12. Witness W0679 is a founder member of the Manor House Group.

13. In relation to being told by myself about vCJD, Witness W0679 says: -

16 "I was going on holiday on the Saturday and I didn't want that man to ruin my holiday. I had always found him very unhelpful and I had never trusted him since he had told me that I wasn't entitled to the Stage 2 Skipton Fund

payment. In that regard he said he had been discussing my case with the Department of Health about the Skipton Fund Stage 2 payment indicating that it was Hepatitis B that had caused my liver disease and so from that conversation I asked one of the liver doctors. At the time I said to him, if someone has been infected with Hepatitis B and C can you tell which one caused liver disease and he stated it is not possible."

14. I am disappointed by Witness W0679's comments, since I have always been truthful and as helpful as I could be with him. It is disappointing to discover that my efforts on his behalf were neither recognised nor acknowledged and that he did not believe what I told him.

15. It is not clear in his statement in what way he regarded me as "unhelpful".

16. In relation to his part two Skipton payment, I would make the following observations. The Skipton Fund was a Hepatitis C compensation scheme. Witness W0679 had cleared Hepatitis C and his liver disease was attributable to chronic Hepatitis B, a relatively unusual situation (see para 7). When setting the Skipton Fund up, I believe that chronic Hepatitis B was not considered. Although it seemed illogical to me, since it was clear that Witness W0679 had contracted HBV from blood products and his cirrhosis was attributable to this, this scenario was not included in the scheme. I met regularly with the DOH (at Skipton House) at that time and took Witness W0679's case up with them directly, to no avail. I explained all of this to him and he refers to this in his statement.

17. In relation to his statement: *"At the time I said to him* [the Hepatologist]*, if someone has been infected with Hepatitis B and C can you tell which one caused liver disease and he stated it is not possible".* The hepatologist gave a correct answer to a false premise put to him by the patient. The premise is that if one is actively co-infected with both HCV and HBV, can one determine which is causing the liver disease, to which the answer is "no". However, Witness W0679 had cleared HCV and was therefore no longer actively infected with HCV, but was actively infected with HBV. This was confirmed

repeatedly by HCV-PCR testing. Therefore, his liver disease was entirely attributable to chronic HBV. I explained this to Witness W0679 on a number of occasions but it would appear that he remains reluctant to accept it.

18. Witness W0679 has also stated:

39 *"I remember on one of the ward rounds Dr Hay came round with an army of medics all staring at me and just came out with something that really upset me and shocked me. He said because I had chronic active Hepatitis B I wouldn't get a liver transplant because the immuno-suppressant drugs wouldn't work. I wasn't on the transplant list at the time. I was really upset and cried a lot that night; I was staring death in the face at this point. When you know you have cirrhosis of the liver and not going to be given the help needed was dreadful. I didn't sleep that night."*

19. I did not say this and, as his statement confirms at paragraph 41, I denied saying it at the time. There must have been some misunderstanding because not only is the statement attributed to me incorrect, but I was actively trying to facilitate his referral to Leeds for liver transplantation at the time. Such a referral must come from a hepatologist. Witness W0679 was unquestionably very ill at that time, with liver failure and jaundice and in a poor psychological state because of his obvious rapid deterioration. It was very clear to me and to my Hepatology colleagues that without liver transplantation he would die within less than a year. Dr TW Warnes arranged a referral to Dr Davies in Leeds and he was put on the transplant list. To facilitate transplantation, the Hepatologists and our team attempted HBV viral reduction in Manchester with Interferon and Lamivudine which took place within a few months.

Section 3: Other Issues

20. None

Statement of Truth

I believe that the facts stated in this witness statement are true.

Signed _____ GRO-C _____

Dated ___6/3/23_____