Witness Name: Professor Charles Hay
Statement No.: WITN3289034
Exhibits: WITN3289035-038
Dated: 09/09/2020

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF PROFESSOR CHARLES HAY

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated 26 May 2020, and specifically in response to the statement of Witness W3114

I, Dr Hay, will say as follows: -

Section 1: Introduction and Background

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.
Copies of my curriculum vitae and publications have already been submitted to the Inquiry.

2. The Manchester Haemophilia Comprehensive Care Centre (Adults) is based in Manchester Royal Infirmary (MRI). 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 took up post 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.

3. We now have four consultants with this specialism. In 1994, we had three Haemophilia Nurses, one of whom (Sister Meg Openshaw) 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 the assistance of any junior staff. 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.

4. This group of patients is complex and many need multidisciplinary care and so this situation was unsatisfactory. In the first year in that post, 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 very close throughout this period, but not formalised around a clinic. 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. Almost all the HIV positive patients have remained with me and are joint-managed with Dr Ashish Sukthanker, Consultant HIV Physician.

5. **Section 2: WITN3114 Treatment Overview**

6. WITN3114, had moderate severity Haemophilia A with a factor VIII baseline of 3-4%. Moderate severity haemophilia (baseline factor VIII 1-5%) is milder than severe haemophilia (Baseline <1%) and the bleeding severity varies between individuals. Many will seldom bleed. WITN3114's bleeding tendency appeared to have been more severe than average for someone with 3% factor VIII. In the past, he had bled into his joints,

especially ankles, but by the time I knew him he was essentially bleed-free on factor VIII prophylaxis three times a week.

7. In preparing this statement I have had access to WITN3114's extensive medical records. I have confirmed that these run to 7 volumes, only 3 of which have been located. I have made extensive enquiries to trace the outstanding 4 volumes, but without success. There is no record of them having been destroyed, nor would I have expected them to have been destroyed. I have therefore not had access to the full medical records in order to produce this statement.

8. I was WITN3114's Haematologist between December 1994 and March 2003, after which he was allocated to Dr Paula Bolton-Maggs, who had just joined us as Consultant Haematologist. Prior to my arrival in Manchester, WITN3114 had been managed, firstly by Dr Irvine Delamore, from 1963 until his retirement in the late nineteen-eighties, and then by Dr Dick Wensley until he retired in 1992 and, from 1992-2004, by Dr Guy Lucas. WITN3114 transferred his care to Liverpool in 2008.

9. It is most likely that WITN3114 contracted Hepatitis C (HCV) from cryoprecipitate in the 1960's or 1970's. I base that on the amount of treatment he had, but there can be no certainty of this and he may have contracted hepatitis C as late as the 1970s when he was first treated with factor VIII concentrate. His liver function tests (LFTs) were monitored regularly and were very abnormal already in 1979 (the earliest LFTs I could find). He was tested for hepatitis C antibody first by Dr Lucas on 12/1/93. Fortunately, he was not infected with HIV.

10. He was made redundant from his job in 1984 and was unable to work after that time. From 1991 he increasingly complained of chronic fatigue, which worsened over the years and was ultimately assumed to be post-viral fatigue syndrome secondary to HCV.

11. His HCV was discussed with him by Dr Lucas on 14/5/92 and Dr Lucas suggested he see a Hepatologist, but WITN3114 declined this offer.

12. Dr Lucas discussed WITN3114's HCV with him again on 8/11/93 and told him that his liver function tests were deteriorating. Dr Lucas encouraged him to consider treatment with Interferon, which WITN3114 declined. Dr Lucas again discussed treatment with Interferon and deteriorating liver function and hepatology referral with him on 6/4/94. WITN3114's platelet count was already declining, suggesting the possibility of a complication called portal hypertension, which is often secondary to cirrhosis of the liver.

13. On 21/7/95 Dr Monica Bolton noted the recent discovery of a solitary large gallstone from which WITN3114 was symptomatic, and referred him to Mr GRO-D Consultant General Surgeon.

14. Mr GRO-D saw WITN3114 on 17/10/95 and considered he needed laparoscopic cholecystectomy.

15. In November 1995, he was reviewed by Dr Monica Bolton who wrote: *"I have suggested quite strongly that he should try interferon if the hepatitis C, as he believes, is destroying his life to such an extent. He is so reluctant to take on board any medication because of possible side effects that this prevents him trying many therapeutic options."*

16. On 1/11/95, he underwent laparoscopic cholecystectomy under the care of Mr GRO-D This was complicated by secondary haemorrhage and I will deal with this more fully below. He had a liver biopsy perioperatively, which confirmed our clinical suspicion of cirrhosis. After recovering from this surgery, he remained reluctant to be referred on to hepatology.

17. On 10/6/96, I persuaded WITN3114 to be referred to Dr TW Warnes, Consultant Hepatologist. I reviewed him again on 4/7/96. He was still reluctant to consider Interferon. I mentioned liver transplantation in passing. He was seen by Dr Lipscombe, Senior Registrar in Hepatology, on behalf of Dr Warnes on 30/10/96. He remained under the joint supervision of a Hepatologist ever since.

18. He eventually agreed to be treated with Peginterferon and Ribivarin in 2002, which successfully eradicated his HCV. Whilst this undoubtedly improved his prognosis and general health, he still suffered hepatic decompensation at times of acute illness and his oesophageal varices (varicose veins in his gullet, which may bleed catastrophically) required very active management by Hepatology.

19. In 2003, WITN3114's care was transferred to Dr Paula Bolton-Maggs, who had joined us as Consultant Haematologist.

20. WITN3114 obtained a full copy of his notes in 1995, through his lawyers who appeared to be investigating a claim of "battery" against Mr GRO-D again through the medical records procedure in 2004 and again in 2008. In 2008, shortly after making a complaint (which was admitted) about the cleanliness of the ward on which he had been an

inpatient under Dr Bolton-Maggs care, and examining his notes for the third time, he decided to move his care to the Liverpool Haemophilia Centre. Despite Dr Bolton-Maggs making enquiries, WITN3114 declined to explain his decision.

21. I understand that he has recently, sadly, died from hepatocellular carcinoma, a complication of cirrhosis.

Section 3: Responses to criticism of Witness 3114

3.1: At paragraph 19 witness WITN3114 states that he developed clots after being given 3,500 units factor VIII despite the fact that that he was already at 100%. He states that you were responsible for his care and considers this mistake may have been due to inadequate planning. Please comment on this.

22. This relates to an elective cholecystectomy (gall bladder removal) which WITN3114 underwent on 1 November 1995. Dr Bolton, Haemophilia Clinical Assistant referred WITN3114 to Mr GRO-D Consultant General Surgeon for an opinion on the management of his single large gallstone. This gallstone was unrelated to his HCV. Mr GRO-D considered that WITN3114 should have a laparoscopic cholecystectomy. This is the standard approach to symptomatic cholelithiasis (gallstones) and is safer from the bleeding perspective and generally has a shorter recovery time than traditional open cholecystectomy.

23. In the lead up to this surgery, Dr Bolton suggested that his surgery might be a good opportunity for a liver biopsy to establish if WITN3114 had evidence of liver damage (cirrhosis). WITN3114 refused on the grounds that he did not want to know the answer. He also made this clear to the surgeons in outpatients. An "open" liver biopsy, i.e. a liver biopsy during the course of an operation under direct vision and with the application of diathermy is safer than percutaneous "closed" liver biopsy which is carried out by passing a special biopsy needle through the skin, in to the liver under ultrasound guidance.

24. The role of Haematology in supporting surgery in patients with a bleeding disorder is to act in a supportive capacity, normalising the patient's clotting for a period of up to a week to permit surgery to take place without excess bleeding risk. This is done by evaluating their clotting system preoperatively and in the case of Haemophilia A, by giving regular 12-hourly factor VIII concentrate to keep the factor VIII level in the normal range (50-

200%) for seven days, until wound healing has taken place with close laboratory monitoring of the factor VIII level and titration of the factor VIII replacement therapy as necessary, as indicated by the laboratory values and the clinical situation.

25. These patients are managed perioperatively and postoperatively on the ward that would be usual for a patient undergoing that surgical procedure.

26. The cholecystectomy, took place on 1/11/1995. During the course of the operation, Mr GRO-D saw WITN3114's liver had a "cobblestone" appearance, which is typical of cirrhosis and took a liver biopsy.

27. The patient subsequently had a very significant intra-abdominal secondary haemorrhage the cause of which was never clear. The factor VIII levels and coagulation levels were as shown in the table below, which I have compiled from Exhibit WITN3289035

28. This shows that WITN3114's pre-operative pre-infusion factor VIII level was 4% and after factor VIII infusion and immediately pre-op had risen to 114%. His factor VIII level was consistently maintained in the normal range with the single exception on 22/11/95.

29. WITN3114's surgical course is summarised in the complaint correspondence, which I produce as Exhibit WITN3289036. In summary, although haemostasis was established at the end of surgery and the factor VIII level was in the normal range, he became restless in the evening and it became apparent that he was bleeding significantly.

30. He had become over-sedated with his analgesia and had fallen out of bed and this was suspected to have started the bleeding. His blood pressure dropped. He was taken back to theatre at about midnight, the biopsy site *was* visualised and was not bleeding, and no specific bleeding site was identified. He settled after this.

31. On the 22 November he was readmitted with a fever and a secondary bleed thought to be related to infection. Intense factor VIII replacement was started and the bleeding settled overnight.

32. On the evening of the 22 November he was unstable and it was suspected that he was bleeding and so, on an empirical basis, a further dose of factor VIII was administered and the factor VIII level measured pre and post infusion. Bearing in mind that pre-infusion the patient was cardiovascularly unstable and the factor VIII level was unknown,

I think that was a reasonable decision. This raised the factor VIII level to about 300%. This is arguably an unnecessarily high level, but by the following morning the level was down to 63%. I would suggest that had such a large dose not been given to this actively bleeding patient, then the factor VIII level would have been subnormal, with associated bleeding risk, by the following morning. Furthermore, there is no evidence in Mr Bullen's records that he suffered blood clots to all his major organs as a consequence of his treatment.

33. WITN3114's surgery was well-planned and his haemophilia treatment kept his factor VIII level in the normal range for the whole time, despite significant bleeding.

Date Time	Factor VIII level %	Other tests/notes
30/10/95 16/37	Fibrinogen 2.0 (1.5-4.0) PTT 5.5 (normal) pre-op.	
1/11/95 10.48am	4%	Pre-op, pre-treatment.
1/11/95 10.49 am	114%	Pre-op, post-treatment
1/11/95 16.29	110%	Post-op recover room
1/11/95 9.25 pm	54%	post op. evening. Bleeding
2/11/95 9.20 am	74%	Post laparotomy
2/11/95 15.46 pm	78%	fibrinogen 0.5 ??DIC
3/11/95 09.19	88%	
4/11/95 17.16	68%	
6/11/95 18hrs	112%	
7/11/95 15.03	105%	
8/11/95 09.53	98%	
22/11/95 09.37	116%	pre-infusion Pyrexia. Secondary haemorrhage
22/11/95 09.37	308%	Post infusion
23/11/95 11.24	63%	
24/11/95 11.19	96%	
24/11/95 11.40	70%	
27/11/95 13.31	96%	
27/11/95 13.32	124%	
30/11/95 11.45	47%	

3.2: At paragraph 23, Witness W3114 states that he was prescribed half doses of his medication after full doses caused him side effects. He states that you did not agree with this and wrote to the liver department saying that the treatment should be stopped because it would not work. According to the witness, the liver department

ignored your advice and continued to prescribe half doses of the medication. Please comment on this.

34. What WITN3114 actually says in paragraph 22 and 23 is:

"I was still under the care of Dr Hay and the liver department at the MRI at the time. It was the liver department that prescribed the treatment course. However, almost immediately I was unable to take the full dose of the medication as it became extremely difficult for me to take due to the side effects. As a result, the liver department decided to prescribe half doses but over an extended period of time for the remainder of my treatment course. The course of treatment is normally six months but as I was only able to take half dose I was treated for 11 months. The doctor then wanted me to continue with the treatment for a further two months, but I could not bring myself to take it any longer.

Dr Hay did not agree with the prescription of half doses over an extended period and wrote to the liver department on 22 November 2002 (Exhibit WITN3114003) saying that the treatment should be stopped because it would not work. He used to paint the picture as black as possible. Thankfully the liver department ignored Dr Hay's letter and prescribed half doses of medication. I cleared the virus in 2003."

35. Exhibit (WITN3114003) provides an account of a pre-arranged joint consultation between WITN3114, Dr Ali Aboutwerat, Associate Specialist in Hepatology, and me, to discuss a strategy about which we had already been corresponding for two months. The exhibit describes our discussion of a strategy to *intensify* his treatment in the light of poor treatment tolerance and a poor therapeutic response, in an attempt to maximise his chance of responding. I produce the entire correspondence relating to his antiviral treatment as paginated Exhibit WITN3289037, which will provide perspective.

36. This correspondence shows that in my letter to Dr Warnes, Consultant Hepatologist, dated 21/3/02, WITN3289037 page 1, I acknowledged that WITN3114 was to be referred to Leeds for consideration of Liver Transplantation and I encouraged Dr Warnes to consider treating him with Peginterferon and Ribivarin.

37. The liver disease was being managed jointly by Haematology and Hepatology. The clinics took place simultaneously in adjacent clinic areas so the patients came on the

same day and some joint consultations were possible. We had a very close and collaborative relationship with Hepatology, as was appropriate.

38. Assessment in 2002 and reassessment of his liver biopsy from 1995, led us to believe that he had cirrhosis and showed that his HCV genotype was 3 (letter 27/5/2002) WITN3289xx037 page 4. His genotype was more responsive than the commoner genotype 1 but his cirrhosis was a poor prognostic indicator, partly because cirrhotic patients responded much less well and also because patients such as WITN3114, whose portal hypertension leads to cytopaenias (low blood counts) even before treatment starts, were often unable to tolerate full-dose treatment. This adversely affects the outcome of treatment.

39. The Department of Haematology agreed to pay for the Peginterferon and to do most of the monitoring - see letter 27/5/2002 at WITN3289037 page 5). Haematology had a different funding mechanism from Hepatology and were therefore in a position to start treating our patients with the much more effective pegylated Interferon some months before it became generally available to Hepatology.

40. WITN 3114's treatment with Peginterferon and Ribivarin began on 5/6/2002. Like most patients, he struggled with this and, in my experience, patients like WITN3114, who have post viral fatigue syndrome before treatment starts, suffer particularly badly with fatigue once treated with Interferon-based treatment regimens. His account of his symptoms and side effects from his treatment is not untypical.

41. On 17/9/02, Dr Aboutwerat, Associate Specialist in Gastroenterology and Hepatology, wrote to me to ask my opinion about the use of granulocyte stimulating factors *"to allow him to continue with the treatment of WITN3114 and give him a maximum chance of response"*, dose reductions having been necessary because of the adverse effect on his blood count - WITN3289037 page 9.

42. I responded to Professor Warnes, and to Dr Aboutwerat on 1/10/2002 WITN 3289xxx page , detailing our experience of this approach and encouraging the use of this strategy to give WITN3114 the best chance we could. I suggested a joint consultation, which took place on 22/11/02 -Exhibit WITN3114003. This letter details how WITN3114's liver function tests had failed to normalise on treatment and how, without further intervention, he was unlikely to respond to ongoing treatment using reduced doses and given his background of cirrhosis. It goes on to describe a strategy to use various growth

factors to improve WITN3114's blood count in such a way that it would be possible to increase the dose of WITN3114's Peginterferon and Ribvirin treatment and improve his chance of a good response. This was agreed with WITN3114.

43. WITN3114 was then treated with GCSF (a growth factor that increases white cell count) which enabled hepatology to increase the dose of both his Peginterferon and Ribivarin, which ultimately successfully eradicated his HCV - WITN3289037page 20. His treatment ended after 11 months, against advice from Haematology and Hepatology, because WITN3114 wished to be off-treatment at the time of his daughter's wedding WITN3289037 page 19.

44. This led to a partial remission in as much as his cirrhosis improved to some degree, as did his general health. I am sure his prognosis was much improved though, sadly, he eventually succumbed to hepatocellular carcinoma, a complication of cirrhosis. Had his HCV not been eradicated, I have no doubt that he would have died many years sooner or would have been been transplanted.

3.3: At paragraph 39, witness W3114 states that he believes you wrongly diagnosed him on several occasions and made the wrong choices during his treatment. Please comment on this.

45. I cannot identify any errors of judgement in my management of WITN3114 between 1994 and 2003, when I was his consultant. WITN 3114 suggests that he lost confidence in me and as a result transferred his care to the centre in Liverpool in 2008. By 2008, WITN3114 had not been my patient for five years. His decision to move centres followed a complaint about the cleanliness of the ward on which he had been an inpatient under the care of Dr Bolton-Maggs, which the CEO accepted.

3.4. At paragraphs 41 and 42, witness W3114 states that he recalls another patient asking you for medical records and you refused on the basis that they had been destroyed. He states that you provided these records only after that patient threatened to take matters further. Please comment on this.

46. WITN3114 actually says: *"I think it was only because of this complaint about the cleanliness that I was able to obtain my complete medical records from the MRI. I recall another patient I knew told me that he had asked Dr Hay for his medical records and Dr Hay had refused on the basis that they had been destroyed. It was only after that patient*

threatened to take matters further that Dr Hay suddenly changed his position and provided the medical records. They were in the drawer of his desk. Unfortunately, I have lost contact with this patient so I am unable to guarantee the accuracy of my comment."

47. This is incorrect. I also highlight the final sentence of WITN3114's comment above. The identity of the other patient to which he refers has not been provided and therefore I am unable to comment specifically. I do however make the following observations.

48. WITN3114 accessed his notes on three occasions, in 1996 through his solicitors, and in 2004 and in 2008 through the Medical Records Department and was presumably familiar with the process. He obtained full copies of his medical records on two occasions, in 1996 and in 2008. His application for his notes in 2004 is produced as Exhibit WITN3289038, signed by WITN3114 and myself. I was unable to find the similar application for 2008, presumably signed by his consultant, Dr Bolton-Maggs.

49. Access to medical records is a patient right regulated by the Data Protection Act, and is not discretionary. Application for access is not made to a member of medical or nursing staff but to the Medical Records Department. Should a patient ask a member of medical or nursing staff for access to their notes, they are redirected to the Medical Records Officer.

50. In almost all cases the initial approach is to the Medical Records Officer or a secretary or a member of nursing staff. The only part played by the medical staff is that the patient's main consultant is expected to countersign the form to ensure that there is no reason for refusing access. Exhibit WITN3289038 confirms that the stated acceptable reasons for refusing access to notes are extremely restricted. I cannot remember these ever applying. Accordingly, I have never, refused a patient access to their notes and I never went through the notes before agreeing access.

51. Collecting the notes together and providing them for the patient to view or providing a complete photocopy is managed entirely by the Medical Records Department and does not involve clinical staff in any way.

52. I do not keep patients notes in drawers in my office. If I do have notes in my room they are on top of my desk so that clerical staff can readily retrieve them, if required. This is deliberately kept to a minimum and it would be unusual for there to be more than 3 sets of notes in my room at any one time. There is a tracing system for notes so that they are

traced to the last person, department or clinic that had them. Therefore, if I have a set of notes, they are traced to me and the clerical staff know where to go to get them.

53. Therefore, if a patient requested their notes, as described above through the Medical Records Department, it would be a clerical officer who would be looking around for each component on behalf of the Medical Records Department. Notes from patients who continue under our care or who have died in the previous 6 years are *not* destroyed, in accordance with the hospital Medical Records policy. However, when patients are seeing several different specialties and have notes running to up to 7 volumes, which is not uncommon for our patient group, tracing all of the notes can sometimes be a challenge for the medical record clerks. Where the notes run to many volumes, only the recent volumes may be "pulled" for a clinic, the rest remaining in storage. In that way, components of the notes may become separated.

Statement of Truth

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

Signed _____ GRO-C _

Dated _____9/9/2020_

Table of exhibits:

Date	Notes/ Description	Exhibit number
1/11/95 – 30/11/95	Factor VIII/coagulation levels	WITN3289035
6/12/95 – 11/02/96	Complaint correspondence	WITN3289036

21/03/02 – 6/06/03	Correspondence regarding treatment of HCV	WITN3289037
29/01/04	Application for medical records	WITN3289038