Witness Name: Charles Hay
Statement No: WITN3289028
Exhibit: WITN3289029
Dated: 09/7/2020

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.

I, Charles RM Hay, will say as follows: -

Section 1: Introduction

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

Senior Lecturer in Haematology Liverpool University and Director Liverpool Haemophilia Centre, Royal Liverpool Hospital 1987-1994.
Professor of Haemostasis and Thrombosis.

Director UK National Haemophilia Database since 2002.
Member UK Haemophilia Centre Doctors Organisation ('UKHCDO') Regional Committee from 1987 and then Advisory Committee since 2007 (the committee changed names).
Vice Chairman UKHCDO 1997 to 2005.
Chairman UKHCDO 2005-11.

1. In 1994, when I took up post at the Manchester Royal Infirmary ('MRI'), I was single handed, but with the passage of time we acquired more consultants specialising in Thrombosis and Haemostasis, first in 1999 and then in 2003, when the patients were divided up and reallocated amongst first two and then three

consultants. Unit policy, for example around management of liver disease, was reached by consensus amongst the consultants in Thrombosis and Haemostasis and, until about 2010, the Thrombosis and Haemostasis Consultants did all of their ward rounds together. In this way, all of us knew all of our regular patients and were able to pick up their management on call and to comment on their management. By 2013, the Wednesday ward round was the only ward round attended by all the consultants together, the other ward rounds being conducted by the consultant on-call for that day.

Section 1: Clinical Review

2. My statement has been formulated with reference to the Manchester Royal Infirmary notes. These appeared complete to me. I produce collectively as **WITN3289029** those entries and items of correspondence to which I have referred in this statement. I did not have access to Mr Theaker's notes from the Blackburn Hospital.

3. Mr Theaker is a pleasant and chatty man. I would agree that we have a good and continuing professional relationship. I am very sorry to read Mr Theaker's concerns about the care he has received, and I was not aware of them until I received his statement. If he had raised them with me at any stage, I would have been happy to address them.

4. Mr Theaker was born on GRO -C /64 was diagnosed with Haemophilia in 1965. He has been managed by Manchester Royal Infirmary (MRI) or the Haemophilia Centre in Blackburn since that time. Originally, he would have been under the care of Dr Israels and then Dr Irvine Delamore and then Dr Richard Wensley, Consultant Haematologists at Manchester Royal Infirmary (MRI).

5. From the 1970s Mr Theaker was principally managed in the Haemophilia Centre in Blackburn Hospital, by Dr David Newsome, Consultant Haematologist, until his retirement in the mid-1990s. During that time, Mr Theaker was seldom seen at MRI though there is a normal liver function test in his notes from MRI in 1992.

6. Blackburn was geographically more convenient for Mr Theaker and continued as a Haemophilia Centre until Dr Newsome retired, after which all the patients came to MRI.

7. The hepatitis C antibody test became generally available in 1992 and almost all of the patients under our care at MRI were tested in 1992 and 1993.

8. Dr Newsome referred Mr Theaker to me for a second opinion on 6/12/95 WITN3289029 page 1. Dr Newsome specifically asked me to review his factor VIII prophylaxis regimen because Mr Theaker was on a very intensive factor VIII regimen but despite that was bleeding relatively frequently into his joints and, in particular, was suffering a good deal of pain in his right ankle. Mr Theaker failed to attend any of the five appointments to see me he was offered that year on 31/3/95, 26/4/95. 7/6/95, 16/8/95 and 8/11/95 (I believe because of his work commitments), and so I was unable to review him in 1995.

9. On 22 August 1996, Dr Newsome referred Mr Theaker again - WITN3289029 Page 2, noting that the patient had not attended the previous year, but reiterating his concern about his joints, the frequency of his bleeding, and the intensity of his factor VIII regimen. Mr Theaker first attended an appointment with me on 25/9/1996 – WITN3289029 page 3.

10. Mr Theaker's Treatment regimen was not, in fact particularly unusual and there was no reason to change it. As would be my practice when seeing any patient with a long standing bleeding disorder for the first time, I evaluated his potential past exposure to blood born viruses. There was no reference to this in Dr Newsome's two letters. He had not been seen at MRI since early 1992 and the advent of HCV testing in later 1992, and so there were no HCV test results and only a single set of (normal) liver function tests from 1992 in his MRI notes. Based on his previous treatment history, I would have assumed that he would have been exposed to hepatitis C either from his treatment with cryoprecipitate or from his first exposure to factor VIII concentrate. Since about 30% of patients clear the virus spontaneously, I would not have known whether he had active hepatitis C infection.

11. For that reason, I had to investigate and evaluate him from scratch and hence told him that he had *probably* been exposed to HCV. I did mention possible treatment but we were not in a position to know if that would be required at that time. I arranged HCV antibody and RNA tests on 9/10/96. I enquired about alcohol consumption and advised him to moderate this. This is general advice to

anybody with any form of liver disease and was not meant to imply that he was drinking above the general "safe limit", something I would have reassured him about at the time. This would have been the first time he was tested at MRI. I wrote to Dr Newsome and to the GP.

12. The tests showed normal liver function but showed a positive HCV-RNA test, confirming that he had been exposed to the virus and had not cleared it. I think he then continued primarily under the care of Dr Newsome.

13. Mr Theaker then missed several further appointments, and I saw him next on 11/11/98. He was very concerned about losing his job because of the I problems he had secondary to haemophilic arthropathy. We also discussed his HCV. This was difficult to evaluate because of his infrequent attendances, but as far as I could judge there was no indication for treatment at that time. I emphasised the importance of keeping this under observation. I wrote to Dr Newsome and the GP and we retested him. WITN3289029 pages 5-6.

14. Over subsequent years, we monitored his liver disease using LFTs and ultrasound.

15. His liver function tests were mildly abnormal from 1998, though his elevated gamma GT on 12/4/2000 and his history, suggested that this was partly attributable to a combination of HCV and moderate alcohol consumption. Dr Bolton had first raised the possibility of treatment for HCV on 12/1/99 – WITN3289029 pages 7-8. She quoted him a 30% dose response and ran through the treatment side effects. The criteria for offering treatment for HCV, the treatment available, and the tests available changed over the years. There has never been any suggestion that Mr Theaker has serious liver disease however, and his most recent fibroscan is normal, indicating that he does not have liver fibrosis or cirrhosis. Consequently, there has never been any clinical urgency to offer him treatment.

16. On 27/3/2002, I discussed treatment for HCV further - WITN3289029 page 14. I had a long discussion with him about this and about the likelihood of success and the treatment side effects. I suggested that we should evaluate the situation further (i.e. check his LFTs and test him to see which HCV genotype he had). I

explained to him that I would undertake the administrative steps we were obliged to take to get the health authority to pay for the treatment, and that before starting treatment it would be advisable to see him with his wife, so that I could counsel the whole family about the treatment side effects, since these are commonly severe, with depression, general malaise, general tetchiness and weight loss, amongst others. This can cause severe depression and I felt it important that his wife and family were aware, in case he developed any of these symptoms.

17. I arranged HCV genotyping, a test that had earlier been unavailable, and which provided a guide to the likely treatment response and the treatment duration required. This showed that Mr Theaker had HCV Genotype 1a, a genotype relatively resistant to the treatments available at that time, which were Peginterferon and Ribivarin. The likely response rate with this treatment was 40% after treatment for 12 months.

18. Mr Theaker asked that his treatment be deferred until after the summer holidays and so that his treatment was not subject to any short administrative delays that might otherwise have resulted from the process of asking the health authority to fund the treatment. Funding was in place well before the summer.

19. I subsequently saw Mr Theaker again on 9/8/02, this time accompanied by his wife and son. We discussed the side effects of treatment and the prognosis in some detail WITN3289029 page 15. We started him on Peginterferon and Ribivarin on the same day and arranged to review him 7 days later. This was the best treatment available at that time and would remain so until the advent of newer, far better tolerated and far more effective treatments that have only become available in the last 4 or 5 years.

20. Mr Theaker experienced side effects with the treatment which made it difficult to tolerate. However, he managed to complete the 12 month course. Unfortunately his HCV relapsed soon after finishing the treatment. I would have told him that we would keep his liver disease under surveillance and offer him further treatment should significantly improved treatment emerge.

Section 2: Response to specific Criticisms:-

21. In paragraph 8 of his statement Mr Theaker says:

"Professor Hay told me of the HCV diagnosis in or around 1999 or 2000 during what seemed to me nothing more than an "oh by the way" type of chat at a routine appointment. He asked me how I was and I responded with words to the effect that I was a "bit knackered, feeling low, working lots of hours and doing a lot of driving". The advice I received was "well you've probably got Hep C…… He then arranged some blood tests and said I would receive a letter asking me to return with Denise to discuss treatment……..
The discussion was all very casual. I get on very well with Dr Hay but I do not believe that the manner in which I was informed of my diagnosis was serious enough given the deadly nature of the disease."

22. I think Mr Theaker may be conflating several consultations that took place over the period 1996-2002. I am sorry that he considers the way in which I first broached the subject of hepatitis C with him to have been too casual.

23. When I first saw Mr Theaker, on 25/9/96 he was primarily under the care of Dr Newsome in Blackburn and I had been asked to see him by Dr Newsome to offer a second opinion on his orthopaedic problems and factor VIII replacement regime.

24. Since the HCV test had been available for four years, I believe I would have assumed that he had been tested and counselled by others. I would not have expected him to be unaware of the problem or for this to be his first consultation at which it was discussed. The referral letter did not mention HCV at all, and since he had not been seen in MRI since the advent of HCV testing, I did not know his HCV status and was therefore not in a position to discuss it in detail. For that reason, I would have told him he had probably been exposed, based entirely on what I knew of his treatment history. The disease usually has a good prognosis and Mr Theaker has never had serious liver disease. When I first saw him, the only liver function test I had available to me was completely normal. I was not in a position to discuss his prognosis, or indeed treatment, without further evaluation. Treatment was not discussed until 1999. His wife was not invited to attend until 2002.

25. In paragraph 8 of his statement, Mr Theaker observes:

"I am unclear as to when I first tested positive for HCV and/or whether Dr Hay knew for sure that I had HCV when he said to me that I 'probably' did."

26. I do not know (and did not know then) when he was first tested for HCV because I do not have access to his Blackburn notes. The first time he was tested for HCV in MRI was on 25/9/96, when he first saw me. I arranged HCV antibody and antigen testing and liver function tests. I thought it was overwhelmingly likely that he had been exposed to HCV, but I did not know if he had cleared the virus or was still infected, since his liver function test had been normal.

27. In paragraph 9 of his statement Mr Theaker comments:

"In subsequent conversations Dr Hay intimated that I had had HCV for some time"

28. Based on his treatment history and the known epidemiology of the disease, I would say that he was probably infected from cryoprecipitate as a boy, since he was always quite a heavy user of replacement therapy. Each bag of cryoprecipitate is now known to confer a 0.5-1% risk of HCV, though that was not known at the time. A typical dose would be 6-12 bags. At the latest, he would have been infected from the first treatment with concentrate in the 1970s. He has no history of jaundice that I am aware of and has always been asymptomatic from his liver disease and there was no test for HCV until 1992, and so it is impossible to be more precise.

29. Mr Theaker states that he should have been treated sooner for his HCV, although he did not raise this with me until after his GP had referred him to a local Gastroenterologist - WITN3289029 page 16. I believe he is specifically referring to the new generation of drugs to which he eventually responded.

30. He responded to Peginterferon and Ribivarin in 2002, but relapsed after treatment. That treatment was the best available in 2002. There was no further therapeutic advance until about 2015, when triple therapy became available. Mr Theaker has understandably indicated that this, being an interferon-based regime, would have been unacceptable to him because of its side-effect profile.

31. About that time new drugs were becoming available, but were initially used in combination with Interferon. Subsequently, interferon-free regimens emerged that were much better tolerated by the patients and had a far better response-rate than earlier treatments. These drugs, such as Sofosbuvir were initially so extremely expensive ($88,000 for a three month course of treatment), that their introduction into the UK was severely delayed. When first introduced, they were reserved for patients with very severe liver disease and controlled by the hepatologists, who were providing all the antiviral therapy for our group of patients by that time. As time passed, drug costs declined, further new drugs became available, and the criteria for selection for treatment was relaxed progressively. During that time, I met regularly with our hepatologists to discuss my patient cohort and for them to update me on the latest guidance on eligibility for treatment. It was my aim to eradicate HCV from all of our remaining patients as quickly as possible. In late 2016, it became possible to treat all remaining patients, including those who, like Mr Theaker, had previously had low clinical priority because they were asymptomatic and did not have severe liver disease.

32. After discussion with our hepatologist, we conducted a computer search to compile a list of all remaining HCV positive patients, most of whom had either failed to respond to earlier treatments, or had refused treatment. This was submitted to the hepatologists on 12/12/16. I have reviewed that list. There are only 15 names on it (about 5% of the patients we would have had listed in the mid nineteen-nineties), and for some reason Mr Theaker's name is not amongst them. We are at a loss to explain this. I can only apologise to Mr Theaker for this, but it delayed his treatment by only a year. Since he has a normal fibroscan and liver function tests, I do not believe that he has come to any harm from this delay.

33. I may have mentioned clinical trials to him in general but have not recruited him to any and would certainly *not* have offered him gene therapy since we have only been conducting such trials in the last two to three years and he was not eligible for such studies prior to HCV eradication. Past experience also leads me to believe that his job would have made it difficult for him to comply with the protocol. Active liver disease and difficulty complying with the twice-weekly hospital visits required by the protocol are exclusions from such trials.

34. It is my practice to 'horizon scan' with my patients and discuss with them future treatments that are in the pipeline (whether I am trialling the product or not). An example of this is my discussion with him of Emicizumab in April 2019 – WITN3289029 page 20.

Statement of Truth

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

Signed GRO-C

Dated 9/9/2020