Witness Name: Professor Gordon Lowe
Statement No.: WITN3496036
Exhibits: WITN3496037 – 047
Dated: 19 April 2023

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF PROFESSOR GORDON LOWE

I provide this statement in response to the request under Rule 9 of the Inquiry Rules 2006 dated 05 October 2022.

I, Professor Gordon Lowe, will say as follows: -

Section 1: Introduction

1. **Please set out your name, address, date of birth and professional qualifications.**

   My name is Professor Gordon Douglas Ogilvie Lowe.
   My address is c/o Central Legal Office, Edinburgh.
   My date of birth is GRO-C 1949.

   I hold the following professional qualifications: -
   MB,ChB (with Honours and gold medal) 1972, University of Saint Andrews
   MRCP UK 1974
   JCHMT Completion of Training in General (Internal) Medicine 1980
   MD by Thesis (with Commendation) 1984, University of Dundee
   FRCP (Edinburgh) 1986
   FRCP (Glasgow) 1986
   FRCP (London) 1989
   FFPHM (Honorary) 2001
   DSc by Thesis (Medicine) 2006, University of Glasgow

2. **Please set out the positions you have held in your duties within General Medicine, Vascular Medicine and Thrombosis, your consultant role at Glasgow Royal Infirmary and also as the Haemophilia Centre Co-Director, the organisations in which you held these positions and your role and responsibilities in these positions.**

1 November 1974 – 31 December 1977. Registrar in General Medicine, University Department of Medicine, Royal Infirmary, Glasgow (Professor Edward McGirr). Clinical general medicine (50%), including haemophilia (1% of clinical practice) and thrombosis, endocrinology and coronary care. Teaching and research (50%).

Lecturer in Medicine, University of Glasgow Department of Medicine, Glasgow Royal Infirmary; and Honorary Senior Registrar in Medicine, Glasgow Royal Infirmary; January 1978 – September 1985. General (Internal) Medicine, with interests in thrombosis, haemostasis, and vascular medicine.

Senior Lecturer in Medicine, University of Glasgow Department of Medicine, Glasgow Royal Infirmary (October 1985-1992), promoted Reader in Medicine (1992-1993), then Professor of Vascular Medicine (1993- September 2009); and Honorary Consultant Physician, Glasgow Royal Infirmary (October 1985-September 2009). Teaching, research and clinical practice in general medicine, thrombosis and haemostasis, and vascular medicine.

West of Scotland Haemophilia and Thrombosis Centre, Glasgow Royal Infirmary; – assisting Senior Lecturer/Reader, University Department of Medicine and Honorary Consultant Physician Dr Charles Forbes (who was Co-Director, with Dr George McDonald, Consultant Haematologist) (October 1985- 1987);

Co-Director (1988-September 2009) with Dr McDonald (1988-1990), liaising with Dr John Davidson, Consultant Haematologist with interest in haemostasis and thrombosis and in charge of Blood Bank including ordering of blood, blood products and recombinant clotting factor concentrates; and with Dr Isobel Walker, Consultant Haematologist with interest in haemostasis and thrombosis (including women and children at Glasgow Royal Maternity Hospital); then with Dr Walker as Co-Director and Honorary Professor of Perinatal Medicine (1991-September 2009); and Dr Campbell Tait, Consultant Haematologist with an interest in Haemostasis and Thrombosis (2000 -

September 2009). After Dr Davidson retired in 1996, Drs Walker and Tait were in charge of Blood Bank.

Honorary Professor, Bioengineering Unit, University of Strathclyde; 1998-2009

Emeritus Professor and Honorary Senior Research Fellow, Institute of Cardiovascular and Medical Sciences University of Glasgow, October 2009 - present.

3. **Please set out your membership, past or present, of any committees or groups relevant to the Inquiry's Terms of Reference which can be found on the Inquiry's website at www.infectedbloodinquiry.org.uk .**

I have been a member of the following committees or groups: -

- Scottish Society of Physicians , member 1979-
- British Society for Haematology, member 1980- ; Scientific Advisor 1994-97; member, Working Party on Fibrinogen 1995-2001
- British Society for Haemostasis and Thrombosis, member 1982-; committee member 1987-92; President 1990-91
- International Society for Thrombosis and Haemostasis, member 1982-2009; Vice-President 2001-03; senior advisory member 2003-; Investigator Recognition Award 2009
- Scientific Services Advisory Group, Scottish Home and Health Department, member (1987-88)
- UK Haemophilia Centre Directors/Doctors Organisation (UKHCDO), member 1988-2009; organiser of Comprehensive Care Centre national audit 1992-2000.
- Scotland and Northern Ireland Haemophilia Centre Directors Committee (SNIHCDC), member and co-chair 1988-2009
- Scottish Home and Health Department, Scottish National Blood Transfusion Service, and SNIHCDC - Annual meetings, and meetings of its Coagulation Factor Working Party, member, 1988-2009
- Glasgow Royal Infirmary Stroke Service Development Group - chair 1993-97
- Health Sciences and Public Health Research Committee, Chief Scientist Office, Scottish Home and Health Department, member 1995-99
- Clinical Audit and Resources Group (CRAG), Scottish Office Home and Health Department, member (and member of its Clinical Outcomes Subcommittee) 1996-2002

- NHS Scotland Recombinant Coagulation Factor Working Party, member 1997-2002
- RCPE, Deputy Assessor 1998-98; Assessor and member of Council, 1999-2003
- Scottish Intercollegiate Guidelines Network (SIGN), member of Council 1998-2003; Chair 2002-07
- Clinical Outcomes Group, NHS Quality Improvement Scotland, member, 2003-08

Section 2: Responses to criticisms of W2183

The criticisms I have been asked to respond to are:

4. ***Paragraphs 7, 8 and 9***

I cannot remember exactly when GRO-B: S was diagnosed, but I remember it was around three or four years after he had been transferred to the Glasgow Royal Infirmary. One of the times I had taken S to the Haemophilia Unit, we were pulled to the side and told that they had found something in S 's blood work. It was Professor Lowe that spoke to us and he told us that S had contracted non-A non-B hepatitis. The crisis around hepatitis had not happened yet so at that time it did not seem like something to worry about in terms of the way they told us about the infection. In hindsight, they downplayed it quite a bit.

I cannot remember specifically what we were told about the infection on that day. What I can say is that it was very concerning to hear as S 's mother but Professor Lowe made it sound as if there was nothing to worry about.

I think the information we were given was not enough for either of us to understand the situation. It was not made clear at the time that the infection could get worse or could lead to complications further down the line. It felt like Professor Lowe glossed over a lot of things.

5. ***Paragraphs 11 and 12***

All I can say about how we were told about S 's infection was that the way it was put to us, it was not something we should be worrying about. They

didn't seem to think it was that big a deal, which looking back was completely wrong. They didn't say anything about continuing to monitor S or anything like that. We pretty much heard nothing more about it until S was offered Interferon in his twenties.

At S 's diagnosis there was no information provided about the risk of cross infection. That was something he was told later on but we didn't receive any information about that kind of thing initially.

In preparing this response, I requested viewing of S 's complete case records from Glasgow Royal Infirmary, from 1986 when he first attended the Haemophilia Centre, to my retirement in 2009. The Central Legal Office has provided me with photocopied case records. I am not confident that I have received complete copies of these.

The copies I have record –

S was referred from GRO-B Hospital Haematology Department on 19 February 1986 by Dr GRO-B (Clinical Assistant to Dr GRO-B to Dr Charles Forbes, who was Co-Director of the Glasgow Royal Infirmary Haemophilia Centre with Dr George McDonald (WITN3496037). Dr GRO-B 's letter records that he was diagnosed in 1977 with moderate severity haemophilia (factor VIII level of 8%), after his older brother GRO-B: S2 had been diagnosed with haemophilia, and had a few haemorrhagic problems as a child. I recall that at GRO-B Hospital Dr GRO-B was consultant until 1983, when he was succeeded by Dr GRO-B .

This letter records that in 1981 S received cryoprecipitate for thigh haematoma; then commercial Factor VIII for ankle joint bleed. In 1985 and 1986 he received cryoprecipitate for recurrent ankle bleeds; then DDAVP (desmopressin) after he had an allergic reaction to cryoprecipitate.

The letter also records that S was tested at GRO-B hospital for HTLVIII (HIV) antibody and Hepatitis B surface antigen; both of which were negative; and liver function tests (transaminase levels) which were normal.

I note that in Section 2,5, W2183 states that she cannot say for certain when S was infected (with hepatitis C) but it could have been any time after he started receiving Factor VIII when he turned sixteen. However, it appears more likely that infection occurred from 1981, aged 10, when he received at GRO-B Hospital not only many doses of cryoprecipitate, but also commercial Factor VIII concentrate, which carried a high risk of hepatitis prior to viral inactivation.

I note that in Section 2,6, W2183 states that "no information was ever provided to her or her son about the risk of infection from receiving these blood products…. No one ever mentioned there being a risk of anything". I would be surprised if Drs GRO-B and GRO-B did not advise W2183 about risks of hepatitis and AIDS when performing tests for HIV, hepatitis B, and transaminases as a marker of non A non B hepatitis; given their evidence to the Inquiry on their practice in 1985-86. I would add that during 1985-86 W2183 and her older son S2 were informed of these risks at Glasgow Royal Infirmary, where these tests were performed on her older son S2 (see my written statement WITN3496048).

S's case records indicate that he was treated at Glasgow Royal Infirmary for joint bleeding from 1986 with desmopressin (DDAVP) if minor, or cryoprecipitate, in accordance with Drs Forbes and McDonald's policy for patients requiring occasional treatment. In January 1987 he was admitted with a large haemarthrosis of the knee, requiring cryoprecipitate, joint aspiration to exclude septic arthritis, and a course of antibiotics. He was also treated for nosebleeds with cautery by Ear Nose and Throat staff WITN3496038).

At review in May1987 he remained negative for Hepatitis B surface antigen and HIV antibody, and liver function tests (transaminase levels) were again normal (WITN3496039)

I reviewed him in March 1988, by which time I had succeeded Dr Forbes as Co-Director with Dr McDonald. Hepatitis B surface antigen, HIV antibody, and transaminase levels remained normal (WITN3496040). I requested Hepatitis B antibody level, which showed he was not immune and he was therefore asked to attend for vaccination, which was successful.

S required no further treatment until February 1992, when he was treated for knee joint bleeds with SNBTS Factor VIII concentrate, then from May 1993 with SNBTS high purity Factor VIII concentrate. including some home treatment. I reviewed him on 21 May 1993 with our Rheumatology colleague Dr Canvin, who noted bony changes and referred him to the Orthopaedic department for consideration of arthroscopy and removal of an osteo-chondritic fragment from the left knee (which was performed on 12 July 1993 - WITN3496041 and WITN3496042). At this review, I counselled and advised S of his positive hepatitis C antibody test, including: continuing to monitor his liver function tests (which remained normal), and precautions to avoid transmission by blood or bloody fluids, and limiting alcohol intake (WITN3496041). His general practitioner and Orthopaedic surgeon were also informed of his positive hepatitis C test (WITN3496042).

In May 1994 he was reviewed by Dr Holyoake, Senior Registrar in Haematology, who noted improvement in his left knee, no further bleeds, and normal liver function tests. She repeated counselling and advice regarding hepatitis C, including barrier methods of contraception, and reassurance that the chance of sexual transmission was low. (WITN3496043).

In November 1994 I reviewed S and re-referred him to the Orthopaedic Department. I updated him on hepatitis C, including: that there was no clinical or biochemical evidence of liver disease; advice on barrier contraception and limiting alcohol intake; and that an antigen test was now available which would indicate whether or not he was a carrier of the virus and this was requested. He had not used home treatment for two years but wished to continue having this available (WITN3496044). He was also vaccinated against the hepatitis A virus, as he had no natural immunity.

In 1995 S was confirmed to be positive for hepatitis C antigen by the PCR test, and I referred him to the joint haemophilia and hepatitis clinic which had recently been established by Dr John Morris at the Haemophilia Centre. Dr Morris confirmed that S had no clinical or biochemical evidence of chronic liver disease; and discussed with him the potential long-term sequelae of chronic Hepatitis C infection, the use of Interferon, its treatment and potential side-effects. With S's approval, he started Interferon treatment in November 1995 (WITN3496045). As noted by W2183, he was one of the first people to receive Interferon. I see no record of Ribavirin in the case records.

In September 1997, Dr Morris reported that S "remained PCR negative, six months after treatment and can now be classified as a sustained responder to Interferon therapy…. It would seem, in view of his recent normal liver function tests, that he has been cured of Hepatitis C." (WITN3496046).

At that time, Dr Walker and I arranged that S's treatment be transferred from SNBTS human factor VIII to recombinant factor VIII (Recombinate), as he was a priority patient for transfer according to national criteria (WITN3496047).

6. Response to W2183: 1.1, 7-9; and 1.2, 11-12.

Paragraph 7

At no time did S have clinical evidence of non-A non-B hepatitis (jaundice, enlargement of liver or spleen) or biochemical evidence of non-A non-B hepatitis (elevated serum transaminase levels).

Paragraphs 8 and 9

I gave evidence to the Penrose Inquiry, then to the Infected Blood Inquiry, on the process at the Glasgow Royal Infirmary Centre for informing patients of their hepatitis C antibody results, then antigen results (WITN3496013; 66, p 114-116). Patients were told that a confirmed positive antibody test meant exposure

to the virus; and that subsequent antigen tests (available from 1994) would show whether the patient was a carrier of the virus, and hence at risk of chronic liver disease; or not. They would be informed of current knowledge about the risk of progression to chronic liver disease and cancer; and the current state of development of antiviral drugs. Patients were advised to keep their alcohol consumption low, and to continue the precautions with sexual intercourse and blood which the Centre had recommended from 1985 for patients who had received blood products, regardless of their HIV status. Patients were advised to discuss this with their regular partners, who the Centre would be happy to see for discussion and counselling about testing for hepatitis C. From 1993, patients were given information leaflets on hepatitis from the Haemophilia Society or British Liver Trust; for reinforcement of information given at the clinic.

After S was confirmed to have a positive hepatitis C antibody test in 1993, I counselled and advised him that this test indicated previous exposure to the virus; and that further (antigen) tests would be required to determine whether or not he was a carrier of the virus. If these were positive, he would be referred to the liver clinic for monitoring and consideration of treatment with antiviral drugs. He was informed that he had no clinical or biochemical evidence of hepatitis; but that this did not exclude the possibility of developing chronic liver diseases in future years. At this time, I also informed his brother S2 and mother W2183 of positive hepatitis C antibody tests and their significance (see written statement WITN2185001).

Paragraphs 11 and 12

See response above. S was monitored regularly from 1993; and given information on the risk of cross-infection of hepatitis C, and precautions to prevent this.

Section 3: Other Issues

7. **If there are any other issues in relation to which you consider that you have evidence which will be relevant to the Inquiry's investigation of the matters set out in its Terms of Reference, please set them out here**

None.

Statement of Truth

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

Signed GRO-C

Dated 19 April 2023

Table of exhibits:

Date	Notes/ Description	Exhibit number
19/02/1986	Letter from Dr GRO-B to Dr C Forbes	WITN3496037
14/01/1987	Letter from Dr K Spowart to GP	WITN3496038
11/05/1987	Letter from Dr K Spowart to GP	WITN3496039
10/03/1988	Letter from Dr G Lowe to GP	WITN3496040
21/05/1993	Letter from Dr J Canvin to Mr I Kelly and GP	WITN3496041
21/05/1993	Case record notes – Dr J Canvin and Dr G Lowe	WITN3496042
05/05/1994	Letter from Dr Holyoake to GP	WITN3496043
15/11/1994	Case record notes Prof Lowe	WITN3496044
17/11/1995	Letter from Dr J Morris to GP	WITN3496045
10/09/1997	Letter from Dr Morris to GP	WITN3496046

02/09/1997	Letter from Prof G Lower to Finance Department, Dr I Walker, GP and Dr T Taylor	WITN3496047