

Witness Name: Dr Russell Cowan

Statement No.: WITN7734001

Exhibits: WITN7734002 - 022

Dated: 31 January 2024

## INFECTED BLOOD INQUIRY

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### WRITTEN STATEMENT OF DR RUSSELL COWAN

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I provide this statement in response to the request under Rule 9 of the Inquiry Rules 2006 dated 11 October 2023.

I, Dr Russell Cowan, will say as follows: -

#### **Section 1: Introduction**

1. I am Dr Russell E Cowan, date of birth GRO-C 1944, of GRO-C  
GRO-C I am a retired consultant physician, specialising in gastroenterology and hepatology and I held that position at Colchester Hospital University NHS Foundation Trust from 1981 to 2008 when I retired from the NHS.

#### **Section 2: Response to criticisms by Witness W0404**

2. To inform my responses to the criticisms made by Witness W0404 I have obtained copies of the clinical notes from East Suffolk and North Essex NHS Foundation Trust (formerly Colchester Hospital University NHS Foundation Trust) dating from 1985 to 1995.
3. Introduction: It is important to appreciate that the Witness posed a problem to me and my surgical colleagues of how to explain her intermittent and severe

abdominal pains, sometimes associated with vomiting. With these symptoms she had been first assessed under the care of one of my consultant surgical colleagues in June 1986 and no diagnosis was reached despite multiple investigations. See exhibit **WITN7734002**. She did not re-present with abdominal symptoms until September 1989 when she was admitted under the care of a different consultant surgeon with whom I worked closely as we are both specialists in gastroenterology and hepato-biliary problems. During that and subsequent admissions with abdominal pain the focus of our attention was trying to explain this and the other associated symptoms rather than whether the Witness had acquired a chronic viral infection from one of the many blood products she had received hitherto in her life. Her bleeding diathesis, Factor X deficiency, was being supervised by my colleague in haematology, as and when required. For example, when the Witness underwent surgical procedures in 1987 and 1988, she was treated prophylactically with a combination of fresh frozen plasma (FFP) and tranexamic acid under the instructions of a consultant haematologist. It is important to be aware also that serological testing for hepatitis C infection did not become available in the UK until 1991 whereas she had been tested for hepatitis B infection in 1979 and 1984, both with negative results. See exhibit **WITN7734003**. In answer to the criticisms made of me, I believe it is important to carefully review the history of the Witness' abdominal symptoms from 1989 to 1994 as I hope this will clearly show mine and my colleagues' commitment to the Witness and will show that her central health problem between those dates was unrelated to an undisclosed hepatitis C infection.

4. Details of the admissions: The Witness was admitted as an emergency to Colchester Hospital on 21 September 1989 under the care of my consultant surgical colleague with bouts of the recurrent upper abdominal pains. She underwent extensive investigation, including oesophago-gastro-duodenoscopy (OGD) and CT scan of the abdomen with particular attention to the pancreas, and no definite explanation was revealed. On admission there was extensive abdominal tenderness and 'some guarding' and initial blood tests, taken in the A&E Department, showed a high total white cell count, raising the question of

an inflammatory process or possibly stress-induced leucocytosis which rapidly resolved. See exhibit **WITN7734004**.

5. I was asked by my surgical colleague to see the Witness for my opinion on the explanation of her recurrent symptoms and I met her for the first time on 6 November. This was in her home for the sake of speed of seeing her. I was already acquainted with the Witness as she was a nurse at the local private hospital where I saw patients and her husband, a consultant radiologist, was a work colleague of mine. The letter I sent to her general practitioner clearly states I thought her attacks of abdominal pains were genuine, despite the normal investigations she had recently undergone and previously in 1986. I felt the pains were visceral and possibly of intestinal origin, probably colonic. Specifically, I suggested the Witness may be experiencing bouts of colonic spasm centred on the transverse colon. Usefully, her husband pointed out that his wife had been worried by uncertainties caused by a change of management of her place of work, the local private hospital, where she was one of the nursing staff. He suggested the degree of anxiety she expressed was enough to trigger her intestinal symptoms. I recommended a trial of antispasmodic therapy and said I would be in touch with the Witness in a couple of weeks for a progress report. See exhibit **WITN7734005**.
6. In the event the Witness was admitted as an emergency to Colchester Hospital with further episodes of the same abdominal pain on 10 December 1989, this time under my care. On this occasion there was less widespread upper abdominal tenderness but on 11 December liver blood tests showed a raised aspartate aminotransferase (AST) of 386 (normal <35) and a raised gamma glutamyl transferase (Gamma GT) of 164 (normal <55). See exhibit **WITN7734006**. When this blood test was repeated on 14 December the AST was 24 and the gamma GT was 111. See exhibit **WITN7734007**. It would appear that, as both abdominal ultrasound (USS) and abdominal CT scan were reported as normal 6 weeks earlier, they were not repeated and instead imaging focussed on whether there was evidence of intermittent intestinal obstruction, given the severity of the pain and associated vomiting and the knowledge that small bowel adhesions had been seen at laparotomy in 1988.

Another attack of upper abdominal pain occurred on 17 December when there was upper abdominal tenderness and increased bowel sounds but again erect and supine abdominal X-rays showed no evidence of small intestinal obstruction and the serum amylase was 150 (normal <390) and the AST was 33 and gamma GT 93. In the light of the distressing and recurring symptoms suggesting an obstructive cause it was agreed with the same surgical surgeon that a diagnostic laparotomy or laparoscopy should be the next step in attempting to reach a diagnosis to which on 19 December 1989 the Witness gave her verbal consent. The clinical notes record the following: "Patient happy to have a laparotomy or laparoscopy as long as problem is sorted out". See exhibit **WITN7734008**. The procedure was scheduled for 3 January 1990.

7. With the aid of hindsight now it is surprising the transient but significant raised levels of AST and gamma GT on admission were not further pursued. As previously stated, the AST level had fallen to within the normal range by the next blood test on 14 December. The admission abnormalities point to liver cell inflammation for which there are several possible explanations, including the subsequently discovered small gallstones. I can only suggest it was the transient nature of the raised AST and the nature of her symptoms at the time which distracted me from explaining these abnormalities. If this failure contributed to the continued distress and suffering experienced by the Witness I most sincerely apologise. It is of note that blood tests both before (1986) and subsequent to the result on 11 December have repeatedly shown raised levels of the MCV (enlarged red blood cells) and the gamma GT. These mild abnormalities seem never to have stimulated any interest or explanation.
8. Although the Witness went home from hospital prior to Christmas, she was readmitted on 29 December with continuing symptoms, including nausea and loss of appetite. She had vomited on 28 December. Diagnostic laparotomy via a right para-medium incision was performed on 3 January 1990 with FFP and tranexamic acid cover for her Factor X deficiency. The entire small bowel and large bowel were normal, as was the stomach, liver, gall bladder and pancreas. Surprisingly, there were no small bowel adhesions. Lymph nodes posterior to the bile ducts and along side the upper abdominal aorta were

enlarged, so a lymph node was removed for histology and microbiological culture. A bone marrow aspiration and trephine biopsy were performed at the end of the laparotomy. This showed no evidence of marrow infiltration with non-haemopoietic cells. Lymph node histology was also normal. Her recovery was uneventful and she was discharged on 12 January 1990. with the abdominal symptoms still not explained.

9. She was seen in our Joint Clinic by my surgical colleague and me on 25 January 1990 when she reported continuing symptoms. We advised her that, as in patient assessment and multiple investigations had failed to reveal an explanation for her continuing symptoms, the next step should be a psychological assessment to which she agreed. See exhibits **WITN7734009** and **WITN7734010**. My letter at that time says the Witness' attitude towards this suggestion was "resigned rather than enthusiastic". I discussed her case with the Clinical Psychologist and sent a referral letter to him on 5 February. She saw him on 12 February 1990. The outcome of this consultation was to invite her to attend group psychotherapy sessions but she was very reluctant and declined the invitation. Similarly, she declined the offer of a couple more sessions of supportive counselling with the Psychologist alone. In her Witness Statement she states she attended a "mental health institution.....a hospital for the insane" for this appointment. This comment seems out-dated and disrespectful to those patients with severe mental illness. I assume she was seen in the out patient department at Severalls Hospital although the letter from the Clinical Psychologist is headed the Community Mental Health Centre in Halstead. See exhibit **WITN7734011**.

10. The clinical records next show she had been seen for the same problem at the Middlesex Hospital in central London by a gastroenterologist colleague who requested an abdominal ultrasound. This was performed by a specialist gastrointestinal radiologist on 13 May and, for the first time, this type of imaging demonstrated "a number of small gall stones of up to 6mm in diameter" The gall bladder wall showed no significant thickening to suggest chronic inflammation secondary to the stones. See exhibit **WITN7734012**. This result was transmitted to my surgical colleague in Colchester on 5 June

and he scheduled admission of the Witness for cholecystectomy which took place on 16 July under cover of pre-operative Factors IX and X transfusion and tranexamic acid. The operation note describes a thin walled gallbladder containing stones with one small stone impacted in the cystic duct just proximal to its junction with the common bile duct. The operation was performed through a right transverse incision and full laparotomy was impeded by adhesions from previous surgery (presumably the laparotomy performed in January 1990). Histological examination of the gall bladder was described as "unremarkable". The Witness was discharged from Colchester Hospital on 22 July. She was reviewed in out patients on 19 September when she reported loose motions after meals since cholecystectomy but abdominal pain was not mentioned. Abdominal examination was described as "normal" and both blood and stool tests were negative. She was discharged from the surgical clinic on 10 October 1991.

11. I had no further contact with the Witness until June 1993 when my surgical colleague spoke to me and then wrote on 17 June to inform me that the Witness was again troubled by abdominal pain. See exhibit **WITN7734013**. She had been to see a specialist pancreatic-biliary surgeon at The Middlesex Hospital when the question of an abnormality in the pancreas on CT scan had been raised by the same specialist radiologist who demonstrated the gallstones in 1991. However, a repeat CT scan with special views was thought to be normal and, according to my surgical colleague in Colchester, the view of his colleague at The Middlesex, expressed in his letter, was that the Witness was suffering from Chronic Pain Syndrome. I saw the Witness on 12 July 1993 and gathered that the abdominal pains started in December 1992. She made the point that the pains were the same in character as they were in December 1989 when I first was involved in her care and which culminated in the open cholecystectomy for gallstones in July 1991. Despite the conclusion reached at The Middlesex Hospital I felt it was still possible the pains were pancreatic in origin and decided to review the radiological images from London before deciding on the requirement for an endoscopic retrograde cholangio-pancreatography (ERCP) to exclude chronic pancreatitis and/or a retained common bile duct stone. Review of the images by our specialist

radiologist colleague in Colchester felt there were minimal changes in the body and tail of the pancreas, sufficient to justify an ERCP. This was performed on 11 August and I concluded it showed no evidence of chronic pancreatitis or neoplasia and a normal post-cholecystectomy biliary tree. In a letter to her general practitioner on 1 September I expressed the view that the reaction of the Witness to the normal ERCP result was not one of relief but one of disappointment. See exhibit **WITN7734014**. It led me to raise again the possibility of the pains being at least in part functional rather than structural, a suggestion to which, in my opinion, her response was "non-plussed". I suggested she should have the benefit of a psychiatric assessment while recommending the addition of an anti-depressant to her treatment and trying to limit her use of pethidine for pain control for fear of dependence. I spoke subsequently to a psychiatry colleague who agreed to see her and thought it would be preferable if this was as a domiciliary consultation. I see no evidence in the clinical notes I have that this ever took place.

12. On 8 November 1993, the Witness agreed to see me again as long as I did not again raise the option of a psychiatric opinion to which I agreed. She reported the continuing problem of severe, band-like pain across the upper abdomen followed by vomiting. These pains occurred almost exclusively during the night. She reported this as the same pains for which she was extensively investigated in 1989 and for which she underwent a diagnostic laparotomy in early 1990. This revealed no diagnostic findings. When she underwent an open cholecystectomy in 1991, however, adhesions were noted, perhaps relating to this laparotomy. I noted there was "considerable puckering and depression" of the operation scar in the epigastrium, prompting me to suggest in my letter her symptoms might result from episodes of sub-acute small intestinal obstruction caused by adhesions. See exhibit **WITN7734015**. I suggested she should again have a Barium study of the small intestine, looking for tethering or fixation sufficient to cause intermittent small bowel obstruction and then to see her in the Joint Clinic with my surgical colleague.

13. I have no notes or correspondence relating to that out patient consultation but it appears to have taken place as she was next admitted electively for another diagnostic laparotomy during which a liver biopsy would be taken as, by that time, she had been found to have hepatitis C infection. The surgery was performed under cover of FFP and tranexamic acid on 29 June 1994. It showed no small bowel adhesions and only a few adhesions between the right para-median scar and the underlying omentum. The liver was noted to be partially adherent to the diaphragm and the colon and omentum was adherent to the undersurface of the liver. Particular steps were taken to assess the pancreas and it was thought to be normal. The adhesions were divided and a liver biopsy was taken. On 4 July it was recorded that the Witness was still requiring pethidine tablets. She was afebrile and thought well enough to go home. Histological assessment of the liver biopsy showed appearances falling between "mild reactive hepatitis and chronic persistent hepatitis", findings that would be consistent with chronic hepatitis C infection. See exhibit **WITN7734016**.

14. On 14 July the Witness was admitted under the same surgical team with a 5 day history of malaise, fever, nausea and an episode of vomiting on the morning of admission. She looked unwell, pale, mildly febrile with epigastric tenderness around the wound from the recent laparotomy, suggesting the possibility of wound infection, so antibiotic treatment was commenced. Abdominal USS on 15 July showed a small sub-phrenic collection for which reason the antibiotics were continued along with analgesics. See exhibit **WITN7734017**. On 18 July the wound was partially opened to allow the drainage of pus. The Witness remained febrile with poor pain control on oral pethidine, so this was supplemented with oral morphine and the wound was opened further to encourage more drainage. On 22 July microbiology results were available and these showed streptococcal milleri grown in the pus from the wound/sub-phrenic collection and cultures taken from the liver biopsy specimen also grew streptococcal milleri. Antibiotic therapy was changed to benzyl penicillin. See exhibit **WITN7734018**. By 25 July the fever was starting to settle but later that day the Witness complained of pain in the left side of her chest with clinical signs of a pleural effusion, confirmed on chest Xray and



thought to be sympathetic to the sub-phrenic collection. This was drained on 26 July when 500ml of clear fluid was removed. A repeat chest Xray on 28 July showed a persistent left sided effusion while a repeat abdominal ultrasound showed a small collection near the spleen, deemed to be too small to be successfully drained. A further pleural aspiration was performed on 30 July when 400ml straw-coloured fluid was removed and sent for both cytological assessment and microbiological culture. By 1 August the Witness was feeling better with no further discharge from the abdominal wound and no further spikes of fever. She was discharged on 4 August.

15. It was on 1 August that I saw the Witness on the ward for the first time during this admission and in response to a request to discuss the future management of her type C viral hepatitis. This was the first time I had seen her since hepatitis C infection had been diagnosed by a positive HCV antibody test and since her appointment at the Joint Clinic earlier in the year. I had not reviewed the liver biopsy histology by that time but from the pathologist's report (see exhibit **WITN7734019**), I was able to reassure the Witness that the inflammation in her liver was low grade with limited scarring consistent with mild chronic hepatitis. I discussed with her the appropriateness and timing of anti-viral therapy which would be administered by colleagues in the Liver Unit at Addenbrookes Hospital. I told her I would review the biopsy and arrange to see her in my out patient clinic. I reviewed the liver biopsy slides with my pathologist colleague on 26 August and was satisfied with his diagnosis of chronic persistent type C hepatitis with no loss of the normal liver architecture. In my letter to my surgical colleague, copied to her GP and the haematologist who had supervised the management of the Factor X deficiency treatment prior to her recent operations, I said I was not in favour, at that time, of pursuing anti-viral treatment with interferon, instead allowing her time to recover from the recent major health problems and to see me in 2 months in clinic. See exhibit **WITN7734020**. I wrote at the same time to the Witness with the findings of my review of her liver biopsy and reassured her that these were mild and, while consistent with changes seen with chronic hepatitis C infection, there was no need for immediate referral to Addenbrookes Hospital for consideration of anti-viral therapy. Instead, I

suggested she should see me in my out patient clinic and an appointment was offered for 23 November. See exhibit **WITN7734021**. It appears she did not keep that appointment which means I did not see the Witness after our meeting on the ward on 1 August 1994. Instead, she was referred by her GP to the Liver Unit at the Royal Free Hospital where she was first seen on 3 November.

16. Specific responses: I have been asked to respond specifically to the comments made in paragraphs 40-44 and 74 of the Witness Statement.

- a. Paragraph 40: it is important for the Witness to appreciate that in early 1990 she was not referred to “a mental hospital” but to a Clinical Psychologist to be seen in his out-patient clinic. There was never any suggestion that in-patient care would be required. As I hope I explained at the time, psychological assessment is often required in the management of conditions in which an organic or physical cause has not been identified. This is exemplified by the abdominal symptoms of ‘functional bowel disease’ commonly known as the Irritable Bowel Syndrome, and it was this explanation that my surgical colleague and I were pursuing at that time. While it does not inevitably influence the patient’s symptoms, to know that no serious pathology has been found to explain them, sometimes can be a source of great relief.
- b. Paragraph 41: it was certainly a disappointment to all concerned that removal of the gallbladder and with it the gallstones did not relieve the Witness of the abdominal symptoms. Similarly, the division of adhesions during the laparotomy in 1994 seemed to produce no benefit as, in paragraph 70, the Witness says that “Each day I am in constant pain” sufficiently severe to require morphine analgesia twice daily. I am very sorry to learn that the symptoms (which I and many others tried to explain and resolve) continue to the degree of requiring opiates for relief. It would be good to hear that since writing her statement in December 2021 her abdominal symptoms have resolved but I fear this is unlikely.
- c. Paragraph 42: I cannot refute or confirm the comment I was said to have made in reference to the Witness’ husband, apparently in jest. I

would agree that the comment, if it was from me, was inappropriate even if it was a reflection of the relationship we had as fellow medical professionals. If it helps I am prepared to offer the Witness an apology even though, unsurprisingly, I have no recollection of making such a comment.

- d. Paragraph 43: I suggest that generally doctors do not like to be disliked by their patients or to be regarded as “not very nice” but, even when that does apply, it is not usual for the doctor concerned to have this spelt out to him. I must hope this opinion of me is a rare among the very many other patients I have treated and I hope my comprehensive response, when read by the Witness, helps to reduce her anger and bitterness towards me. I hope also that all the details outlined above, describing the management of her case by me and other colleagues over a 5 year period, will allow her to understand that what she was experiencing and what we were trying to diagnose and treat was not due to hepatitis C infection.
- e. Paragraph 44: the Witness states that we met in 2018 when she attended a retirement party for the same surgical colleague with whom I collaborated in her care. She states that, during the party, I asked to be introduced to her when I did not recognise her. I have no recollection of this event or this encounter and I have checked with my colleague who retired in 2013 which was when his retirement party was held at the Royal Society of Medicine in London and which I attended. He does not recall the Witness attending that event but he was not responsible for the guest list. We wonder if the Witness could be mistaken in the year she has suggested.
- f. Paragraph 74: it is unreasonable for the Witness to suggest I was acting “in complete ignorance” of her condition. I had been involved in her care from November 1989, including an admission under my care in December that year and I had the additional benefit of seeing the efforts other colleagues had made to explain her abdominal symptoms earlier in 1989 and in 1986. Her Factor X deficiency was not directly related to her abdominal symptoms and, even if it had been diagnosable at that time, either acute or chronic hepatitis C infection

does not cause episodic, cramp-like upper abdominal pain over the course of several years. On the other hand, gallstones can present with these abdominal symptoms and yet, at that stage in 1990, these were not diagnosed despite efforts being made to do so. When gallstones were eventually diagnosed in 1991 it was assumed they were the cause and, appropriately, cholecystectomy was performed. The small size of the stones at that time suggests they were likely to be even smaller in 1989 and defied the diagnostic sensitivity of abdominal ultrasound and CT scanning. As she has stated, unfortunately removal of the stones and gallbladder did not stop her pains. When the Witness refers to "the truth", I believe she means the hepatitis C liver disease and, if she does, it is disappointing she might hold the belief that this condition was causing her presenting problem of abdominal pain, first investigated in 1986. Our preoccupation between 1989 and 1994 was to define the cause for her abdominal symptoms and treat it successfully. I suggest I and my colleagues were acting with her best interests in mind which meant that all possible and reasonable explanations needed to be pursued. Her hepatitis C liver disease, while being important, was a consequence of one of the various blood products she had received for the Factor X deficiency prior to 1986 and was a problem I was prepared to help her manage but presumably, by that time, she had lost faith in me and sought help from a liver specialist at the Royal Free Hospital. On 2 December 1994 she commenced interferon therapy which she continued for 6 months when on 1 June 1995 it was decided she had not made a complete response, so treatment was stopped. It was disappointing also to read in the clinic letter of 25 July 1995 she continued to have abdominal pains for which she continued to take opiate analgesia. See exhibit **WITN7734022**. I am entirely prepared to offer the Witness my sincere apologies for what she would appear to feel were inappropriate steps and suggestions taken by me in the course of her management. I refer particularly to the referral to a clinical psychologist and the suggestion of her seeing a psychiatrist. I have previously acknowledged that more attention should have been given to the transient liver enzyme

abnormalities in 1989. I totally reject, however, the assertion from the Witness that I nearly killed her.

### **Statement of Truth**

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

Signed     GRO-C    

Dated   31.01.2024  

### **Table of exhibits**

Date	Notes/ Description	Exhibit number
16/06/1989	Letter from Mr May to Dr Hall discussing multiple investigations	WITN7734002
08/11/1994	History of W0404 liver function tests	WITN7734003
21/09/1989	A&E admission record from 21 September 1989	WITN7734004
07/11/1989	Letter to Dr C Hall W0404's GP	WITN7734005
11/12/1989	Test results showing raised AST levels	WITN7734006
14/11/1989	Repeat test results with lower AST levels	WITN7734007
10/12/1989	Extract from handwritten notes from 10 December 1989 to 12 January 1990	WITN7734008
25/01/1990	Letter to Dr C Hall following combined clinic	WITN7734009
05/02/1990	Referral letter to clinical psychologist Dr F Lohman	WITN7734010
23/02/1990	Letters with heading "community mental health centre"	WITN7734011
13/05/1991	Abdominal ultrasound results showing 6mm gall stones	WITN7734012
17/06/1993	Letter from Mr Motson on W0404 reporting further abdominal pain	WITN7734013
01/09/1993	Letter to Dr C Hall with W0404's reaction to ERCP results	WITN7734014

08/11/1993	Letter to Dr C Hall noting considerable puckering and depression of the operation scar in the epigastrium	WITN7734015
28/06/1994	Colchester General Hospital admission record with histological assessment of the liver biopsy	WITN7734016
15/07/1994	Abdominal USS showing small sub-phrenic collection	WITN7734017
22/07/1994	Microbiology results showing streptococcal milleri grown	WITN7734018
13/07/1994	Pathologist report of the liver biopsy	WITN7734019
12/09/1994	Letter to Mr Roger Motson after review of liver biopsy results	WITN7734020
12/09/1994	Letter to W0404 discussing results of liver biopsy	WITN7734021
25/07/1995	Letter from Dr G M Dusheiko re W0404's ongoing abdominal pain	WITN7734022