Witness Name: Chung Yeung Andy Li
Statement No.: WITN3175001
Exhibits: **WITN3175002 – WITN3175017**
Dated: 18/06/2019

INFECTED BLOOD INQUIRY

WRITTEN STATEMENT OF CHUNG YEUNG ANDY LI

I provide this statement in response to a request under Rule 9 of the Inquiry Rules 2006 dated 3 May 2019.

I, Chung Yeung Andy Li, will say as follows: -

Section 1: Introduction

1. My name is Dr Andy Chung Yeung Li and my professional address is Lyndhurst Rd, Worthing, West Sussex, BN11 2DH.

2. I have been a Consultant gastroenterologist since 2005 at Worthing Hospital charged with the care of patients with general gastroenterological problems. I see inpatients, conduct outpatient clinics and perform endoscopic procedures. I completed my specialist training in 2005 at King's College Hospital in London.

3. I have never been a member of any committee or groups relevant to the Inquiry's Terms of Reference.

Section 2: Response to Criticism of Michelle Delia Baker

Background

4. Unfortunately it seems that Mrs Baker's recollection of events regarding my care of her husband vary from my contemporaneous clinical correspondence which happens to also include a medical report I drafted for Mr Baker's Health Insurance Claim on 27th May 2009 and the referral letter I sent to King's College Hospital dated 29th May 2009 [see exhibit WITN3175002] . During the preparation of my response I gained access to Mr Baker's records held by Worthing Hospital.

5. I have great sympathy for Mrs Baker and her family for their terrible loss of her husband at such a young age and the circumstances around this. It is understandable with the passage of time that details and dates may be forgotten. However, I must set the record straight regarding my role in his care, backed up with documented evidence.

Question 4: diagnosis of cirrhosis of the liver

6. At paragraph 7 of Mrs Baker's statement, she claims that I informed Mr Baker that his diagnosis of the liver was "probably caused by alcohol" and failed to inform him about his Hepatitis C (HCV) infection or to mention the HCV as a probable cause.

7. I first met Mr Baker on 10 February 2009 as a result of a referral from the admitting general physician. I was asked to give an opinion on the cause of his vomiting of blood and abdominal swelling. The clinical records available to me at the time indicated that Mr Baker had been drinking alcohol to unsafe limits. The records reported that this had occurred until that current time (February 2009). Other medical practitioners had documented this fact for prior to this. The records show the following reports in relation to alcohol consumption:

    a. On the 16th August 2007, the accident and emergency doctor was told that Mr Baker drank 3 bottles of Bacardi Rum per week [see exhibit WITN3175003].

    b. On 17 March 2008 an ENT surgeon wrote that Mr Baker drank 2 bottles of Bacardi per week [see exhibit WITN3175004].

    c. On the 5th February 2009, it is documented that Mr Baker told the accident and emergency doctor that he had been drinking >100 units per week but had not drank for 2 weeks prior [see exhibit WITN3175005].

    d. On the 10th February 2009 Mr Baker stated to me that he had been abstinent since Christmas 2008, [see exhibit WITN3175006].

    e. On 10 April 2009 Mrs Baker told treating clinicians that he was an alcoholic but stopped drinking alcohol in January, after being diagnosed with liver cirrhosis. [see exhibit WITN317007]

8. It is clear, therefore, that reports were inconsistent with regards to when he had become abstinent from alcohol. On the 29th of May 2019, when I referred him to Kings College Hospital, I took Mr Baker at his word, that he had not drank heavily since Christmas 2008. I did not feel I needed to contest this as the requisite six months of abstinence would have likely elapsed by the time he was seen in Kings, in any event.

9. At the time of my first meeting with Mr Baker there was no suggestion that he was a carrier of HCV as I and my team were not aware of his transfusion history and he did not give a history of participating in at-risk behaviours such as intravenous drug abuse. It is only by subsequent testing for HCV that it was discovered he was infected.

10. Therefore, in light of the abovementioned evidence, it was reasonable to suspect that alcohol had a significant part to play in the development of Mr Baker's chronic liver disease at the time.

Question 5: failure to provide Mr Baker with information regarding HCV

11. At paragraph 10 of her witness statement, Mrs Baker claims that I failed to provide Mr Baker with specific information regarding HCV.

12. Mr Baker was first tested for HCV in February 2009. However, I did not become aware that Mr Baker had HCV infection until receipt of a letter from Sandwell Hospital in the West Midlands on 16th April 2009 [see exhibit WITN3175008]. The reason for that delay is canvassed in my response to question 7, below.

13. Mr Baker had been involved in a road traffic accident on the 9th of April 2009 and was admitted there with jaundice and hepatic encephalopathy. The latter is a clouding of consciousness characteristic of a very dysfunctional liver. The doctors at Sandwell suggested the hepatic encephalopathy had been caused by an infection of his leg (cellulitis), on a background of his pre-existing severe liver disease. During his stay in Sandwell they did some blood tests including tests to check for HCV. The results came back positive and were communicated to the treating team at Sandwell on 14 April 2009 [see exhibit WITN3175009]. Those results were then communicated to me in the abovementioned letter.

14. Mr Baker was subsequently repatriated to Worthing Hospital for further management of his complications of severe liver disease from 17th April 2009. Subsequently all correspondence generated after that date that I have seen mentions Hepatitis C infection as being extremely important in the generation of severe liver disease in conjunction with a previous high alcohol intake. The diagnosis of Hepatitis C is documented in the Worthing Hospital junior doctor's notes from the 17th April 2009 [see exhibit WITN3175010]. I also referred Mr Baker to King's Cross Hospital hepatology department on 29 May 2009.

15. I regret that Mrs Baker believes that I failed to talk to Mr Baker regarding the implications of Hepatitis C infection. The above information was not available to me until mid-April 2009. Unfortunately I did not record my discussions around hepatitis C with the patient in the medical record. However, once I learned of the diagnosis, I would have had great

concerns around how he contracted it and I would have had to consider the transmission route prior to referring him for a liver transplant i.e. previous operations and transfusions and lifestyle choices. I believe that these facts were discussed (in accordance with my usual practice) at the very latest when he was referred for his transplant. It is inconceivable in my view that I did not. Foremost we will have discussed (as is my usual practice) how hepatitis C is transmitted and any concerns around transmission to family members and others.

Question 6: stage of HCV and treatment

16. In paragraph 10 of her statement, Mrs Baker claims that by the time Mr Baker was told of his HCV infection, the infection was too advanced for treatment to be effective.

17. Mr Baker was diagnosed with HCV in Sandwell Hospital between the 9th and 16th April 2009. Unfortunately, the severity of Mr Baker's liver disease and the fact that he had decompensation as exhibited by bleeding oesophageal varices and the need for recurrent drainage of abdominal fluid (ascites) precluded him from treatment with antiviral therapy available at the time in 2009. This would have been the drug combination (or given individually) Interferon and ribavirin, which are contraindicated in patients with decompensated liver cirrhosis, as was in this case.

18. However, Mr Baker would have been an unlikely candidate for antiviral therapy from his first presentation to me in February 2009, due to his signs of decompensated liver cirrhosis (irrespective of the cause). Antiviral therapy would only have been viable if there was significant and sustained clinical improvement. In fact when Mr Baker was seen at King's, his clinical condition had improved to a point which allowed Dr Kosh Agarwal, his hepatologist, to consider that antiviral therapy might be possible at a later date.

19. I also discussed referral for liver transplant with Mr Baker on the first day I met him, on the 10th February 2009. However, I advised that a condition for transplant would be to demonstrate abstinence from alcohol for 6 months (not 12 months as erroneously documented by the junior doctor), which at that time was (optimistically) at least 4 months away. [see exhibit WITN3175006] This would still have been the case even if I had been aware that HCV infection was present at the time.

20. Dr Agarwal at the King's liver unit in September 2009 suggested that, at the time of Mr Baker's first appointment in August 2009, antiviral medicine could possibly be offered (following further review), but only if his condition remained stable [see exhibit WITN3175011]. That clinical position was only reached, however, after treatment provided from February 2009 had improved and stabilised Mr Baker's liver disease. By the time I referred Mr Baker to King's on the 29th May 2009, his liver disease was much

improved since his first presentation in February 2009 [see exhibit WITN3175012] and Mr Baker's liver disease remained stable for his first appointment with Dr Agarwal in August 2009. Dr Agarwal was optimistic that Mr Baker had no contraindications to transplant at the time and was to review Mr Baker for antiviral therapy in four weeks' time. Unfortunately Mr Baker subsequently deteriorated, developed hepatic encephalopathy again, and was admitted to hospital with a fever in November 2009 that was subsequently found to be a result of an infection of the heart valves (infective endocarditis). He therefore was never able to receive the antiviral therapy or transplant. I provide more detail on this in relation to my response to question 8, below.

Question 7: knowledge of the HCV infection

21. In paragraph 11 of her statement, Mrs Baker claims that I knew about Mr Baker's HCV infection as it was noted in his medical records.

22. As outlined in my response to question 4, I first became aware that Mr Baker was a carrier of the Hepatitis C virus, only after he had been tested in Sandwell Hospital in the West Midlands in April 2009.

23. I became aware that Mr Baker had received such a diagnosis as a result of correspondence from Mr Baker's GP on the 30th April 2009. The GP asked why my clinic letter of the 6th April 2009 did not mention the HCV diagnosis, and yet Worthing Hospital laboratory had stated to him over the phone that the blood test done in February 2009 showed that he had HCV.

24. A blood test for viral hepatitis screen had been undertaken by the Worthing Hospital on 10th February 2009, on my instruction to the admitting team. I required this to be undertaken as a pre-requisite to exclude HCV and other infections prior to liver transplant. That test was positive for HCV. However, the printed report was sent to the admitting doctor at Worthing Hospital, but not to me after 27 February 2009. The usual turnaround for viral hepatitis tests was several weeks in my trust at the time due to paper systems and not electronic reporting being in place (report was dated 27th Feb when blood was taken on 10th Feb 2009). I saw Mr Baker in my outpatient clinic for follow up on the 6th April 2019. The usual processes of the trust was that positive results for viral hepatitis would be highlighted by phone call, despatch of original paper report or email to the responsible clinician but not negative results. There was no hepatitis virus test result available in the notes on the 6th April 2009. I wasn't aware at the time that the piece of paper had gone astray or had stayed with the emergency physician. Because I had not been notified by the aforementioned routes I wrongly assumed that the HCV test I had recommended was negative.

25. It was many months later that I discovered a paper copy that was eventually forwarded to me. When I did receive it – I made a point to annotate the document with the words-file carefully [see exhibit WITN3175013]. At that time infection screen results were only recorded on paper and were not accessible by computer. I initiated an investigation as to this lapse of communication. That incident was investigated by the Trust [see Incident Details Form exhibit WITN3175014] To summarise, I did not receive a printed copy of the HCV result till much later having not been notified by the emergency doctor nor the hospital laboratory. I was extremely confused as the to the chain of events and how I was last to know, and so I wrote to both the chief Trust microbiologist and Mr Baker's GP on 23rd September 2009 asking if there had been any testing for HCV without my knowledge and whether HCV test results had been documented elsewhere apart from Sandwell Hospital [see exhibit WITN3175015]. I subsequently received no replies.

26. My correspondence items from May 2009 [see exhibit WITN3175002 and WITN3175012] onwards demonstrate that Mr Baker's diagnosis has never been suppressed or withheld from him or to third parties when consent for release of information was granted. Furthermore all written medical notes refer to the diagnosis of HCV related cirrhosis after the diagnosis had been made in Sandwell Hospital.

27. I regret that a failure in communication within the Trust has led to the misconception that there was something to hide, albeit for a relatively short time. I can assure the Inquiry and Mrs Baker that is not the case.

Question 8: failure to diagnose and treat

28. Mrs Baker claims that I was the main obstacle for Mr Baker accessing HCV treatment. Mrs Baker claims that the failure to treat Mr Baker's HCV meant that when he required a liver transplant, he was too ill to receive the transplant. She further claims that if Mr Baker had been diagnosed sooner he would "still be alive today".

29. As explained in my response to question 6, Mr Baker was not a candidate for antiviral drugs as at February 2009. Such treatment was not a possibility until he improved enough (through treatment) to be referred to Kings College Hospital in May 2009 Mr Baker did not show sustained objective signs of improvement such as reduction of oesophageal variceal size and the reduced need for ascitic drainage until May 2019. Interferon and ribavirin are known to risk causing a deterioration in patients with liver decompensation hence the cautious approach adopted by Dr Agarwal as well. Unfortunately this improvement was not sustained.

30. I referred Mr Baker to the liver transplant service at King's College Hospital on the 29th May 2009. This was after I became aware of the diagnosis of HCV on the 17th April 2019 and Mr Baker received care on multiple occasions both as an inpatient and in

outpatients by myself and my team since my first encounter with him on 10th February 2009. He underwent multiple upper GI endoscopy procedures to deal with his oesophageal varices and he had two drainage procedures of his abdominal fluid accumulation (ascites) between April and May 2009. I do not believe there was any undue delay in making the referral to Kings because:

a. it was important that Mr Baker's complications of liver cirrhosis such as ascites and variceal haemorrhage were dealt with locally. Transplant centres look favourably on the same patients who respond well to treatments for varices and ascites as was the case with Mr Baker as it implies a favourable result post-transplant;

b. Liver transplant centres only accept inpatient urgent referrals for chronic liver patients directly if facilities dealing with these complications are not locally available, which is extremely rare in the modern era.; and

c. Furthermore, liver transplant centres such as King's look unfavourably on transplant candidates who have not been abstinent from alcohol for less than 6 months. It is documented in the written record that on the first clinical contact I had with Mr Baker that I intended to refer him to the liver transplant centre if alcohol abstinence was maintained. Given that optimistically he had been abstinent according to the medical records from August-October 2008 and pessimistically only 2 weeks before February 10th 2009 onwards, the interval to referral was appropriate.

31. In any event, the perceived delay could only have been 3 months from when I first met Mr Baker. Such a delay (from February 2009 to May 2009) would have had no impact on Mr Baker's prognosis as he had already had features of severe liver cirrhosis which had been present for several years and optimisation of his condition as explained in paragraph 29 was essential to present him as a suitable transplant candidate. Mr Baker's condition improved dramatically as a result of treatment during that time, with endoscopy procedures, done on a fortnightly and eventually monthly basis, as well as diuretic medicines that eventually reduced his need for physical drainage of his abdominal fluid accumulation (ascites). Indeed the improvement was such that Dr Argawal in King's stated in September 2009 that there were no obvious contraindications to transplantation at the time.

32. It took several months after my referral (in May 2009) for King's Liver Unit to finally see Mr Baker in September 2009. This reflects the high demand for the services of a liver transplant centre. As stated above, this did not result in impacting Mr Baker's prognosis.

33. Unfortunately Mr Baker was taken off the transplant list by the 22nd February 2010 as he had become very ill with two episodes of a very serious heart valve infection (infective endocarditis) which was the main culprit in his untimely death. Acquiring infections (especially unusual ones as in this case) is a common complication of patients with all forms of chronic liver disease, not limited to HCV infection nor alcohol related liver disease. Severe liver disease results in an impaired immune response to infection. It is also unfortunate that infection in turn makes other complications of liver disease more severe such as hepatic encephalopathy and bleeding from oesophageal varices.. The infection damaged his heart valves and unfortunately due to the severity of his liver disease Mr Baker was deemed not to be fit enough for heart valve surgery nor liver transplantation [see exhibit WITN3175016]. I therefore think it is inaccurate to assert that I was an obstacle for Mr Baker assessing HCV treatment, that the failure to treat Mr Baker's HCV meant he was too ill to receive the transplant or that earlier diagnosis would have meant Mr Baker would "still be alive today".

34. I regret that Mrs Baker had to suffer her bereavement with such concerns for such a long time and if this reflects poor information sharing at the time, I am truly sorry on behalf of my team. I recall talking to a group of Mr Baker's wider family and discovering how hepatic encephalopathy had affected Mr Baker's mental state. This had twisted his behaviour and made him very difficult towards other family members and his wife in particular. There are written descriptions of this in our records that I do not want to bring up again. It must have been a terrible situation for her and she has my every sympathy. I hope I have convinced her that at no time did I aim to conceal any material fact regarding her Husband's diagnosis to him, and that I tried my best to give him timely and appropriate care.

Section 3: Other Issues

35. I previously undertook investigations regarding the source of Mr Baker's infection for Mr Baker's claim for the Skipton fund in November 2019. It was initially difficult to trace when exactly and how he acquired the infection. We narrowed it down to a surgical procedure related to a fractured right femur he had in 1983 [see exhibit WITN3175017]. According to the medical records, he did receive a blood transfusion in Southlands Hospital on the 11th March 1983. I am not aware of any other likely source for Mr Baker's HCV infection.

36. As a result of the failure to notify me of Mr Baker's HCV infection (before he was told the news by a different hospital rather than me) I initiated a Hospital internal incident investigation (as referenced at paragraph 25 of my statement, above). Since that time, the Hospital Trust has invested heavily in modern computerised pathology reporting and there is now much less reliance on paper systems. I regret that this failure in

communication has led to the misconception that there was something to hide, albeit for a relatively short time.

Statement of Truth

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

Signed GRO-C

Dated 18 - 6 - 19