MR VICTOR TAMBURRINI

(1) Where and when the death occurred

Mr Tamburrini was born on 27 April 1957 and died on 17 November 2004 at the Edinburgh Royal Infirmary.

(2) The cause of death

The cause of Mr Tabmurrini's death, accurately recorded on the death certificate, was:

"I (a) Liver Transplant Graph Failure

(b) Recurrent Hepatitis C…"

(3) Reasonable precautions, if any, whereby the death might have been avoided

The evidence led during the Inquiry did not show how Mr Tamburrini became infected with Hepatitis C. It is not possible to say whether there were reasonable precautions whereby his death might have been avoided.

(4) Facts relevant to the circumstances of the death

In September 1984 Mr Tamburrini sustained burns in a road traffic accident. He was admitted to the Glasgow Royal Infirmary where he received a transfusion intravenously of plasma protein solution as part of his treatment. In July 1998 Mr Tamburrini was referred by his GP for the treatment of gynaecomastia and it was noted at that time that he had abnormal liver function tests. He underwent a mastectomy at the Glasgow Royal Infirmary in December 1998. During this procedure he required a blood transfusion. In March 1999 Mr Tamburrini was admitted to Glasgow Royal Infirmary with abdominal pain and a diagnosis of possible pancreatitis was made. In 2001 Mr Tamburrini was referred to the Glasgow Royal Infirmary by his GP because he had developed swelling in his legs, and by his dentist because he had developed an oral ulcer. Blood tests were carried out which suggested that he had severe liver damage and in September 2001 he tested positive for Hepatitis C.

Mr Tamburrini was assessed for a liver transplant in February 2002 and on 26 October 2002 he underwent a liver transplantation. There were subsequently difficulties with biliary strictures and an endoscopic stent had to be inserted. A liver biopsy performed in December 2003 showed that the transplanted liver already had established cirrhosis, with evidence of recurrent and active Hepatitis C infection. Mr Tamburrini consequently underwent a second liver transplantation on 4 February 2004. Although a liver biopsy performed towards the end of March 2004 showed nothing untoward it was felt that there was likely to be Hepatitis C recurring in the liver. Therefore on 29 March 2004 Mr Tamburrini was started on anti-retroviral treatment with a combination of Interferon and Ribavirin. The results of a further liver biopsy carried out on 1 June 2004 were indicative of severe recurrence with Hepatitis C and antiviral therapy was continued. Mr Tamburrini underwent a further liver biopsy in September 2004 and although the histological appearances appeared improved from those in June 2004, his condition continued to deteriorate significantly. On 7 October 2004 Mr Tamburrini was admitted as an in-patient to Glasgow Royal Infirmary where he suffered from fever, ascites and encephalopathy before his death on 17 November 2004.

Evidence was led regarding how Mr Tamburrini could have become infected with Hepatitis C, in particular whether he could have become infected via either of the transfusions that he received in 1984 and 1998. The Inquiry heard evidence to the effect that the plasma that Mr Tamburrini received in 1984 had been heat treated at 60^{O}C for 10 hours by a method that is known to inactivate the Hepatitis C virus, and that Mr Tamburrini could therefore not have been infected via that transfusion [see evidence of Dr. Cuthbertson Day 1 page 134 et seq.]. In relation to the blood that Mr Tamburrini received in 1998, the Inquiry heard evidence to the effect that all the archived samples of donations from the relevant donors were tested, including those that were given to Mr Tamburrini, and that all of them tested negative for Hepatitis C. Furthermore, by 1998 Mr Tamburrini already had abnormal function tests so his liver disease preceded that transfusion.

The only other known surgical procedure during which Mr Tamburrini could possibly have received blood or blood products was an appendectomy that he underwent in

December 1968. However there are no hospital records relating to this procedure and consequently no record of any transfusion having taken place. Dr Mutimer's opinion was that it was very unlikely that Mr Tamburrini would have had a transfusion during that procedure.

In the circumstances the evidence that was heard by the Inquiry did not demonstrate even on the balance of probabilities how Mr Tamburrini came to be infected with Hepatitis C. Dr Mutimer indicated that patients who have had hospital care, or medical or dental care at any stage in their life might be exposed to Hepatitis C infection [see Day 1 page 114 (7) and (14) to (19)] but there is no positive evidence to show how he came to be infected.

(5) Defects in any system of working which contributed to the death and/or systemic issues arising in relation to the death

No defects in the management, treatment and care of Mr. Tamburrini were demonstrated on the evidence.

Systemic issues are not usefully raised by this case since it is impossible to link the infection with any particular cause. The evidence as to the course of the Hepatitis C in Mr Tamburrini is not useful more generally given the uncertainty as to (1) the date of infection and (2) the level of alcohol consumption. In the words of Dr Mutimer "we don't have a very good estimate of the amount of alcohol that the patient consumed" [see Day 1 8/03/11 page 112(7)]. Mr Tamburrini appears on the affidavit evidence to have been a moderate user of alcohol. The medical records are incomplete and potentially misleading. The Inquiry has heard that moderate use of alcohol can accelerate the progression of the disease[1].

[1] See Management of Hepatitis C - A National Clinical Guideline - Paragraph 8.4 "Even moderate amounts of alcohol (within government recommended guidelines) have been associated with increased liver fibrosis compared to those who abstain" PEN.018.0298 [see also the literature cited at footnotes 88 and 98]