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When calling please ask for: Mr. Hazlehurst

Our ref: ADH/JMG/M313

Your ref:

Date 15 July 1997

Mrs. M. Murphy,
GRO-C
Liverpool GRO-C

Dear Mrs. Murphy,

Re: Your Claim

We have now received the report from Dr. Davies and enclose a copy for your consideration. Please telephone to arrange an appointment to discuss the report.

Yours sincerely,

GRO-C

IRVINGS

Enc.

Report of Dr Mervyn Davies on behalf of WILLIAM AUGUSTINE MURPHY (Deceased):

Synopsis

9/11/81 Admission
GI bleed, endoscoped, DU diagnosed, plus oesophageal ulcer (known DU from Barium meal in 1968)
Discharged on cimetidine

20/11/81 Admission
Ongoing abdominal pain and melaena
24/11/81: Laparotomy, vagotomy and pyloroplasty – relatively uncomplicated recovery

9/12/81 Admitted: melaena and jaundice

Endoscopy: bleeding duodenal ulcer

Initial hepatitis B serology negative, subsequently positive

28/7/88
Out patients Dr Hay
Hepatosplenomegaly, no cutaneous signs of chronic liver disease

31/7/89
Out patients, thought to have AIDS related complex, in view of splenomegaly

22/5/90
Pain in groin

22/10/90
Painful left inguinal hernia
Hepatosplenomegaly on examination

23/10/90
Admission, incarcerated inguinal hernia.
Repaired surgically

14/1/91: Increasingly painful left knee, referred to orthopaedics. Impression needs replacement, but pre operative anaesthetic assessment arranged.

6/12/91 Admission for knee replacement

Examination revealed splenomegaly, no ascites or peripheral oedema. The liver was not palpable.

10/12/91 Left knee replacement

30/12/91 post operative bleed into prosthetic joint, despite factor VIII replacement. Moderate thrombocytopenia thought to be contributory

4/1/92: Haematemesis, managed conservatively

13/1/92 Considered to have had further bleed into joint

13/1/92 Swollen testicle, thought to be haematocoele

13/1/92 Noted to have generalised dependent oedema, with no evidence clinically of cardiac failure. Prolonged PT noted, so thought to be due to chronic liver disease.

14/1/92
Haematology review, Dr Hay
Attributes developments to liver disease, 'which is more advanced than we had appreciated. Transaminases and physical signs are unreliable indicators of the severity of liver disease. Had we appreciated the severity of his liver disease we would not have proposed surgery in the first place.
I think his recurrent bleeding probably reflects haemophilia, reduced platelets mildly disordered coagulation secondary to liver disease'.

Dr Hay has explained to the family that he thinks Mr Murphy has cirrhosis.

Ongoing problems with oedema. Increasing bilirubin, to 35μmol/l, with troublesome ascites, despite diuretics. Albumin 27. Planned transfer to ward 7Y.

Ultrasound, ascites, splenomegaly, normal liver texture, but suggests cirrhosis should be considered.

17/1/92 Degree of disseminated intravascular coagulation – treated with cryoprecipitate.

20/1/92
Low grade pyrexia, considering infection of prosthetic knee and return to theatre.

Dr Hay considers that the knee is replaced, but this may settle with antibiotics.
20/1/92: Upper GI endoscopy: 3 columns of oesophageal varices from 32cm. No evidence of recent bleed.

23/1/92 Left knee aspiration – culture negative

23/1/92 Open exploration of knee. Clot aspirated. No infection and no need for removal of prosthesis.

Knee subsequently improved over next 2 – 3 weeks. Discharged at end of February

18/4/92 Rectal bleeding

18/4/92: Upper GI endoscopy. Bleed from oesophageal varices at endoscopy. Rx octreotide and settled.

18/4/92: Elective upper GI endoscopy, with injection sclerotherapy with factor VIII cover. Gastric varices noted, not thought to be the source of bleeding. Suggested admission for liver work up.

21/4/92: Upper GI endoscopy with injection sclerotherapy

30/4/92 Admission with melaena

Rx octreotide, then 6/5/92 Gastroscopy and sclerotherapy

14/5/92: Readmission with melaena

Emergency upper GI endoscopy:showed: bleed from oesophageal varices – treated with octreotide

19/5/92 Upper GI endoscopy with injection sclerotherapy

7/6/92: Planned admission for full formal liver work up under care of Prof Shields and Dr Hay.

8/6/92, Dr Hay advised against this and was not at all happy for this man to have full liver work up, considering it essential to restrict investigations to endoscopy and sclerotherapy and anything else vital. "Clearly prognostic indicator work up not vital, because of the risks versus the likely benefits". Discharged with plan for elective sclerotherapy 29/6/92.

30/6/92 Elective upper GI endoscopy with sclerotherapy. Fundal varices noted. Oesophageal varices thrombosed. PT 15 seconds

Appearance much improved. Extensive para variceal injection sclerotherapy.

Planned further elective examination in 6/12.

10/8/92: Review by Dr Hay, small amount of ascites controlled by small doses of diuretics

19/8/92 Surgical clinic Mr Sutton: plan endoscopy in 6/12.

8/9/92: dysphagia.

PT 19 seconds.

Endoscopy: 8/9/92: obliterated oesophageal varices. No stricture or stenosis, with easy passage of scope to stomach.

28/9/92 ascites and left inguinal hernia

12/1/93: elective uper GI endoscopy

Appearances generally good. No patent varices seen. No therapy required.

Plan for repeat endoscopy in 6/12.

1/2/93 Out patients Dr Hay, complaining of pain from hernia.

10/3/93 Prof Shields clinic, well from liver point of view – noted to have requested AFP. I was unable to find the result.

23/2/94 Arranged TCI for repair of left inguinal hernia and orchidectomy.

22/3/93 Out patients Dr Hay

Venous ulceration of right external malleolus.

21/6/93 Out patient review Dr Hay, improvement of venous ulceration following bandaging.

19/7/93 Review Dr Hay

31/8/93: Elective admission for endoscopy. Ascites and left inguinal hernia noted.

Endoscopy revealed obliterated varices. PT 19 seconds.

24/11/93 Out patient review. Recurrence of hernia.

15/10/93 Discharged from Dr King's clinic, since leg ulcer has completely resolved.

14/11/93 Admitted with recurrent left inguinal hernia

22/11/93
Review in Dr Hay's out patient clinic

12/1/94 Out patients Dr Hay. Peripheral oedema and ascites under control with amiloride 10mg daily.

18/2/94 Chalaion of right lower lid, treated by ophthalmic team. PT 19.5 seconds.

22/3/94 Elective admission for repair of left inguinal hernia and left orchidectomy – a difficult procedure.

Post operative increase of ascites, treated with increased dosage of diuretics, with effect.

27/4/94

Swelling of scrotum more difficult

1/6/94 Reviewed by surgical team. Not for further surgery. Plan to refer to Dr Gilmore for management of ascites.

3/7/94 Dr Gilmore

Severe haemophilia
Chronic HCV with presumed cirrhosis
Varices
Intermittent ascites over 2 years, increased over past 6 months

Poor concentration, but no confusion.

Previous hepatitis B

On examination: tense ascites.

Discussed with Dr Gilmore, with planned admission for investigation of tense ascites and consideration of liver transplantation.

5/7/94 Admission of assessment of liver disease, Dr Gilmore

19/7/94 Persistence of ascites. Patient unkeen on paracentesis, because of bruising which followed the last episode, therefore diuretics increased.

Consideration of referral to Newcastle for consideration of transplantation.

August 1994: repeat elective endoscopy and injection sclerotherapy of oesophageal varices

7/8/94: Admission with encephalopathy, presumed triggered by sepsis. Increasing diuretic requirements.

29/8/94 Elective admission.

30/8/94: Upper GI endoscopy and sclerotherapy

3/9/94 Admission with abdominal pain – awaiting chemotherapy. Possible intraperitoneal haemorrhage from tumour.

Condition deteriorated and Mr Murphy died. The family apparently declined a post mortem examination.

Summary of Investigations:

Ultrasound examinations liver and abdomen:

16/1/92: gross ascites, splenomegaly, homogeneous liver texture. Consider cirrhosis. No focal lesion.

20/7/94: 6.5cm well defined mass noted on ultrasound examination. It is impossible to differentiate between regenerative nodule and tumour. Patent portal vein. Large ascites and splenomegaly. Examination by Senior Registrar, Dr Walters.

The most prolonged prothrombin time I found in the notes was 22 seconds.

November 1981, full blood count, prothrombin time and liver profile were normal.

December 1981, elevated bilirubin and transaminases, consistent with hepatitis, HbsAg negative 12/81 and 2/82 and 2/86

7/87 platelets 126 and deranged LFT consistent with post transfusion hepatitis

10/88 platelets 107, ALT 134, bilirubin normal

11/88 anti HBc positive, anti HBs positive
6/89 platelets 100 ALT 105
10/89 Bilirubin 11μmol/l, ALT 127
1/90 ALT 298, platelets 111
9/90 LFT requested, ALT 292 platelets 99
3/91 ALT 278 platelets 52 albumin 32, bilirubin 16μmol/l prothrombin time 18 seconds
7/91 ALT 183
12/91 ALT 179, bilirubin 21μmol/l, albumin 33

Following the operation on his knee and the prolonged recovery, Mr Murphy's bilirubin rose to a peak of 59μmol/l, the albumin to a nadir of 27 and the prothrombin time to a maximum of 21seconds.

Following discharge, in March 92, the prothrombin time measured 16, bilirubin 20μmol/l and albumin 25 and Mr Murphy was shown to be HCV antibody positive

Following the first variceal bleed in April, the liver tests deteriorated, with bilirubin 62μmol/l, but albumin reasonably maintained at 32 prothrombin time 18

27/4/92 bilirubin 34μmol/l, albumin 33

1/5/92 bilirubin 55μmol/l, albumin 33 and prothrombin time 20 seconds

5/5/92 bilirubin 49μmol/l, albumin38, prothrombin time 22

15/5/92 bilirubin 31μmol/l, albumin 36

19/5/92 bilirubin 30μmol/l, albumin 40

8/6/92 bilirubin 17μmol/l, albumin 36

8/9/92 bilirubin 34μmol/l, prothrombin time 19

23/11/92 bilirubin 27μmol/l, albumin 32, prothrombin time 18

18/1/93 bilirubin 23μmol/l, albumin 30

22/3/93 bilirubin 26μmol/l, albumin 31

21/6/93 bilirubin 27μmol/l, albumin 32

31/8/93 bilirubin 32μmol/l, albumin 28

27/9/93 bilirubin 35μmol/l, albumin 31

18/2/94 prothrombin time 16.5

23/3/94 bilirubin 26μmol/l, albumin 31 prothrombin time 21 seconds

3/4/94 bilirubin 43μmol/l, albumin 28

4/5/94 bilirubin 38μmol/l, albumin 29

23/5/94 bilirubin 38μmol/l albumin 29

15/7/94 AFP 9280

Management of the case:

Mrs Murphy's Stated Key Areas

1) Suitability For Knee Replacement
2) Referral For Liver Transplant Assessment
3) Failure To Diagnose Hepatitis C
4) Delay In Chemotherapy
5) Final Correspondence Of Dr Gilmore And Dr Hay

Additional points to consider:

a) General Standard Of Care
b) Management of Haemophilia
c) Management of Orthopaedic Complications
d) General Surgical care – peptic ulcer disease and inguinal herniae
e) Management of Liver Disease:
   - I Oesophageal Varices
   - II Transplant Assessment
   - III Screening For Hepatoma
   - IV Management Of Ascites
   - V Hepatitis C

f) Management of Mr Murphy's Venous Ulcer

Mrs Murphy's key areas:

1) **Knee replacement operation December 1991.**

The decision to operate on Mr Murphy's knee was based upon a perceived need because of Mr Murphy's severe symptoms.
Mr Murphy suffered haemophilia and as a consequence had suffered haemorrhages into his joints, with severe osteoarthrosis as a consequence of this. Dr Hay recommended referred Mr Murphy to the orthopaedic surgeons for evaluation of his joint symptoms and functional capacity. Professor Klenerman believed that Mr Murphy would benefit from knee replacement surgery. Because of the severe haemophilia, Professor Klenerman specifically arranged for a pre operative assessment by the anaesthetists. This included assessment of physical status, in addition to review of investigations and a chest X-ray.

Mr Murphy developed complications of surgery, including recurrent haemorrhage into the prosthetic joint and possible infection. The latter responded to antibiotics, but the overall stress of the operation and its complications temporarily altered the status of Mr Murphy's liver disease, from being in a well compensated state to a decompensated state.

Dr Hay commented in the notes that if he had known the severity of the liver disease, he would not have requested surgery in the first place.

There are several issues.

Was the joint disease assessed expertly?

I propose that Mr Murphy's arthrosis was carefully assessed, since Mr Murphy was reviewed by Dr Hay who is experienced in the management of joint disease consequent upon haemophilia and he was then referred to and assessed by a consultant orthopaedic surgeon. The notes refer to severe symptoms and disability. Thus, the merits of surgery apparently outweighed the inevitable risks of such surgery.

Was Mr Murphy's physical status assessed?.

With the benefit of hindsight Mr Murphy's did not withstand the acute trauma of the post operative complications well and his previously unrecognised cirrhosis became manifest. Mr Murphy was assessed pre operatively by Dr Hay, Prof Klenerman and Dr Cohen. They were unable to diagnose cirrhosis. This is not particularly surprising, since he was not jaundiced, the liver was not enlarged and there was no ascites when examined at the time of his pre operative admission, although the spleen was enlarged. With the benefit of hindsight, it is clear that Mr Murphy had well compensated liver disease at the time of surgery. His liver function tests were near normal. Values recorded include serum bilirubin of 21μmol/l and serum albumin of 33g/l from December 1991. Cirrhosis per se is not a contraindication to surgery. Mr Murphy's's joint symptoms were severe and if cirrhosis had been diagnosed pre operatively, I expect the recommendation would have been for surgery to proceed. I do not conclude that it was negligent for the surgery to have taken place. It is likely that, but for the unexpected complications, Mr Murphy would have tolerated the surgery well. The complications were not a predictable result of surgery. It appears that the long term results from Mr Murphy's joint replacement were actually excellent. The episode of decompensation of liver disease was not permanent, although liver function tests deteriorated acutely and Mr Murphy developed ascites. Following recovery from the acute effects of surgery and the variceal haemorrhage, the liver function improved and Mr Murphy again entered a prolonged period of relative stability with well compensated liver disease.

2) **The timing of referral for liver transplantation and the reasons for not referring at an earlier stage in Mr Murphy's disease.**

During the entire duration of his illness, Mr Murphy was kept under review by Dr Hay. Following the variceal haemorrhage, Mr Murphy was also kept under review from Professor Shields. Cirrhosis of the liver was presumed to be present from the time that liver function tests deteriorated, when Mr Murphy developed ascites following the joint replacement surgery. I have recorded many of the liver function tests carried out during the period from December 1991 to the time of referral for liver transplantation, which provide an objective assessment of the status of Mr Murphy's liver disease.

Indications for liver transplantation

Objective indications for liver transplantation include persistent hypoalbuminaemia, with serum albumin <25g/l, if there is no recent acute and reversible precipitant. Persistent elevation of bilirubin >150μmol/l, intractable diuretic resistant ascites or episodes of spontaneous bacterial peritonitis. None of these clear indications was present in the case of Mr Murphy, until the time of referral to Dr Gilmore and then on to The Freeman Hospital, at which time ascites had become diuretic resistant and serum albumin was in the mid 20's.

Delay in obtaining a specialist liver opinion.

There are no clear or absolute guidelines as to when patients should be referred for a specialist liver opinion. In my practise an opinion is always offered when it is requested, but there are very many patients with disease similar to that of Mr Murphy who are well managed by their local team. In this particular case, Professor Shields was involved in the management and follow up of Mr Murphy. Professor Shields has an international reputation in the management of patients with liver disease, cirrhosis and oesophageal varices.

Indications for liver transplantation

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On each of Mr Murphy's emergency admissions with variceal haemorrhage, under the care of Dr Hay and Professor Shields, he was always managed expertly. Even in non haemophiliacs, a variceal haemorrhage is a medical emergency associated with a high mortality. At the time of each variceal bleed, an urgent upper GI endoscopy was performed.

Investigations were carried out to determine the cause of the liver disease when the liver disease had become manifest. The disease was assessed clinically and deemed to be initially in a well compensated stage (or early) from the point of view of hepatic function. As previously described, Mr Murphy acutely suffered decompensation of his disease in response to the complications of knee surgery. At this stage, even if a referral had been made to a transplant team, the fear of sepsis in a prosthetic joint would have precluded a liver transplant. Therefore there was no need to refer for a specialist liver opinion. It was rightly anticipated that the liver function would improve following the complications of surgery and the variceal bleeding.

Examination of the liver tests between April 1992 and March 1994 show that Mr Murphy's liver had maintained good synthetic function, since the serum albumin was above 30g/l and the bilirubin only slightly elevated. The albumin level dipped slightly further during April and May following the surgery for his hernia. Mr Murphy was referred to Dr Gilmore in July 1994, when it was apparent that his disease had progressed, with resistant ascites. We know, with the benefit of hindsight that this final deterioration was due to the complication of the development of a large tumour.

The only specific reason for a prior referral to a specialist liver unit would have been if Professor Shields did not feel competent to treat the oesophageal varices. The course of events showed that he made a very favourable effect upon the varices by means of endoscopic variceal sclerotherapy. Professor Shields and the haematology team combined to obliterate safely the varices. This was carried out efficiently and to good effect.

The presence of varices are not an indication for liver transplantation. Intractable variceal haemorrhage or recurrent bleeding unresponsive to endoscopic sclerotherapy or band ligation is an indication for transplantation, but this was not the case. It is my view that a transplant would not have been considered at this stage because of the well preserved liver function, unless symptoms such as poor quality of life would be added to the equation. Such subjective symptoms

cannot be deduced from reading a set of notes in retrospect. The role of liver transplantation for patients with subjective symptoms is also modified by diagnosis. Patients with hepatitis C infection invariably suffer re-infection of the liver graft with the hepatitis C virus following a transplant. Recurrent disease due to hepatitis C post transplant is an increasingly recognised problem. The longer the duration of post transplant hepatitis C, the more likely is recurrent cirrhosis. Therefore patients with hepatitis C are usually counselled against early transplantation for subjective symptoms.

Mrs Murphy's Summary

There are some error's in Mrs Murphy's summary. For example, varices are not an indication of advanced liver failure and can even occur in the setting of a non cirrhotic or completely normal liver. Varices per se are not an indication for liver transplantation unless unresponsive to intervention. I think Mrs Murphy fails to appreciate that although liver transplantation has developed a long way in recent years, it remains a huge undertaking on behalf of the patient and family, because of its associated morbidity and mortality. The absolute indications, stated above, are based upon an expected life expectancy of <12 months, or on the basis of quality of life. As already discussed, quality of life cannot be assessed in retrospect from notes. Mrs Murphy states quality of life was nil. This is a subjective indication. It is not possible to prove negligence for non-referral in a patient who otherwise has relatively well preserved hepatic function, not fulfilling transplant criteria.

The explanation for the timing of referral to Dr Gilmore and then his immediate referral on to the transplant team was that the liver disease had then declared itself to be advancing and to be reaching the point where a transplant was indicated. This was not due to natural deterioration due to cirrhosis of the liver, but to the unexpected complication of primary liver cancer. The development of this tumour caused a rapid decline in hepatic function, since by this stage Mr Murphy was dying of hepatocellular carcinoma. Median survival from the time of presentation of primary liver cancer is between 2 and 5 months.

3) Failure in July 1994 to diagnose cancer prior to transfer to The Freeman Hospital.

I will discuss the relative merits of screening for hepatoma elsewhere. In this case and to answer Mrs Murphy's question, the hepatoma was, in effect, diagnosed shortly before transfer to Newcastle. The information was over looked and therefore not acted upon. The finding of an AFP in excess of 9,000 in the setting of cirrhosis is always due to hepatoma. Furthermore, the ultrasound scan of 20/7/94 clearly showed a focal lesion which warranted further investigation.

It was very unfortunate that this information was not acted upon and this was a mistake of the team responsible for the care of Mr Murphy at that stage. This resulted in unnecessary stress for the patient and family. The distress of a transfer to Newcastle might have been avoided, or the transfer could have been made in the light of the knowledge that liver cancer had developed and that the transplant assessment would be based upon this information. However, the delay in diagnosis between July 1994 when Mr Murphy was looked after in Liverpool and the actual diagnosis of the tumour a month later in Newcastle would not have altered the final outcome.

4) The delay in initiation of chemotherapy

Any delay in treatment for cancer is difficult for a family to come to terms with. Treatment for hepatoma is, however, complicated and inevitably requires some necessary arrangements. Although a 19 day delay seems a long time to the patient and his family, in the context of Mr

Murphy's type and size of tumour I do not think it was unreasonable or particularly unusual. It is likely that the tumour had been present for a great deal of time. The benefit of chemotherapy for primary liver cancer is frequently only marginal. It is not a 'wonder' treatment and is usually considered as being only at best palliative. It is not standard practise in the UK to transplant for large hepatomas, especially if there is evidence of capsular invasion. The role of pre operative chemotherapy is not well established. I do not believe that the planned delay was unreasonable nor do I believe that the initiation of chemotherapy would have altered the outcome.

5) Comments from the final letters of Dr Hay and Dr Gilmore

I agree with Mrs Murphy that neither of these letters appeared particularly helpful. Both seemed to suggest delay in the referral of Mr Murphy. In the first case, the letter of Dr Hay refers to the hepatologists as dragging their feet a bit, with respect to the referral for transplantation.

Secondly the letter of Dr Gilmore refers to the influence of the decision making and timing of referral on the lack of a transplant centre in Liverpool. I agree with Mrs Murphy, that this issue has nothing to do with this particular or indeed any other case. It would only be relevant if a referral had been made to a transplant centre elsewhere and the transplant centre had been responsible for an unreasonable delay. This was not the case.

General standard of Care

Inevitably patients with severe haemophilia have long and complex medical cases. Mr Murphy's condition was complicated further by the complication of hepatitis C infection and cirrhosis.

I consider that generally the standard of care of Mr Murphy was high. A great deal of attention was made to optimally managing his haemophilia and the complications as they developed. For the most part, expert input was obtained from the Consultant grade, including from the haematologists, the orthopaedic surgeons, the anaesthetic input and from Professor Shields. From the correspondence it is clear that Dr Hay played a close part in the management of this case. Some of the individual aspects will be addressed.

a) Haemophilia

I am not qualified in this area, but I would judge from the notes that great attention to the provision of adequate clotting factors and close haematological input was ensured.

b) Orthopaedic

The decision to operate on Mr Murphy's knee was based upon a referral from Dr Hay, who is clearly experienced in the management of joint complications from haemophilia. Mr Murphy was then looked after by an orthopaedic consultant with close attention from the anaesthetists. I would not criticise this aspect of care. This is discussed in greater detail previously.

c) General surgical

Mr Murphy was recognised as first having a duodenal ulcer in 1968, following a barium meal examination. The duodenal ulceration manifested again during November 1981. The disease was appropriately investigated when Mr Murphy presented with a gastro intestinal bleed. The duodenal ulcer was diagnosed endoscopically and treated initially with cimetidine. This was the treatment of choice at that time. Unfortunately, the ulcer recurred, with further bleeding. The

most appropriate course of management was undertaken, namely surgical treatment of the ulcer and cure of his duodenal ulcer disease.

The other surgical aspects of Mr Murphy's care relate to the management of his inguinal hernia. The management of such herniae is always rather more complicated in patients with cirrhosis, but if a patient has reasonably well preserved liver function, as Mr Murphy did, I consider it was reasonable to attempt surgical correction, since the hernia was causing him great discomfort.

d) Hepatic

The management of Mr Murphy's liver disease has been discussed previously in greater detail, in response to Mrs Murphy's points.

In brief, I do not consider that Mr Murphy was negligently managed from the hepatology point of view. His liver tests were monitored closely throughout, both prior to the diagnosis of cirrhosis and subsequently. Complications of cirrhosis, including variceal haemorrhage and ascites was managed expertly. He did not suffer from the complications of sclerotherapy or from inappropriate or over zealous use of diuretics. Unfortunately, Mr Murphy's liver disease followed a late unpredictable complication, since he developed primary liver cancer. This complication is almost always fatal and this proved to be the case.

I) Oesophageal varices

Mr Murphy suffered 3 definite bleeds associated with oesophageal varices. On each occasion an emergency endoscopy was performed, a diagnosis made and treatment initiated. Thereafter the varices were electively treated and eradicated. Regular check endoscopies were carried out. The management of Mr Murphy's varices could not be faulted and was expert.

II) Liver transplant assessment

Liver transplantation is an established procedure for the management of end stage liver disease. This can be divided into acute or chronic liver disease. The former will not be discussed. Indications for transplantation in chronic liver disease can be divided into objective or absolute and subjective. Failure to act upon objective and absolute indicators of the need for transplantation may be considered negligent. In the case of Mr Murphy, he did not reach a stage of absolute need for liver transplantation prior to his referral to Newcastle. I do not consider the timing of referral for transplantation was negligent. In the event referral was too late, but this was through nobody's fault, but due to a complication of his liver disease accelerating due to malignant transformation.

III) Screening for hepatocellular carcinoma (hepatoma)

It is likely that Mr Murphy's management would have been different had the hepatoma been diagnosed at an earlier stage. Hepatoma is a recognised complication of hepatitis C infection, when complicated by cirrhosis, but the role of screening for this complication is far from accepted. Many physicians do not routinely screen for the development of hepatoma.

Two recent reviews from an international medical journal debate this issue: ('Screening for hepatocellular carcinoma in patients with chronic viral hepatitis: Can the results justify the effort?' By Richard Sallie in the journal Viral Hepatitis, 1995, 1: 77 – 95 and 'Should patients

with chronic viral hepatitis be screened for hepatocellular carcinoma?' by Massimo Colombo, also in the journal Viral Hepatitis, 1995, 1: 67 – 75).

Both of these articles were written at around the time of Mr Murphy's illness and draw upon expertise from publications written at aroun' this time. The abstract from Dr Sallie is concise and I will quote this:

'Screening patients with chronic viral hepatitis for hepatocellular carcinoma is widely practised, but is of unproved benefit ……..
While sophisticated mathematical models of cancer screening programmes exist and have been validated for cancer of the breast, the data required by such models to reach a logical answer about the value of screening for hepatocellular carcinoma in the setting of chronic viral hepatitis are incomplete and often poorly and inconsistently reported….published data suggest hepatocellular carcinoma screening programmes have the potential to cause real harm – psychological, physical and economic in a proportion of the majority of patients who would not ultimately develop HCC and who could not benefit from screening, in addition to whatever uncertain benefits may accrue to the relatively small numbers of patients found to have HCC by screening.'

In conclusion, although some individual physicians choose to screen for hepatocellular carcinoma, screening for hepatoma is not accepted medical practise and is unproven. It cannot be considered negligent not to have carried out routine screening during this time.

IV) Management of ascites

Mr Murphy developed ascites at a variety of times during his illness. Usually it coincided with acute decompensation following surgery. Diuretic therapy was appropriately prescribed and monitored. Generally the ascites was controlled. At the time that Mr Murphy's ascites became diuretic resistant, he was referred to Dr Gilmore. He was unable to improve upon the situation and therefore referred Mr Murphy on for transplant assessment, since the ascites had become intractable.

V) Hepatitis C

The hepatitis C virus was first detected serologically in 1989. Thereafter the development of tests to diagnose this condition progressed fairly rapidly, such that by 1991, a reasonably sensitive and specific test had been developed and the blood transfusion service were screening blood products for this virus. A diagnosis of hepatitis C was confirmed in March 1992 in Mr Murphy's case. I do not think this represents a particularly long duration of delay. I do not think that earlier diagnosis would have influenced management. His blood test etc were already being considered high risk, because of his haemophilia. If the hepatitis C had been diagnosed prior to the knee surgery I do not think that this would have altered the decision to proceed with this operation, since his disease was well compensated. I think that the benefits of surgery for such a debilitating knee condition would reasonably outweigh the risks of surgery, which he seemed to recover from well ultimately, despite the protracted course.

Mr Murphy would not have been a candidate for interferon in 1991. Firstly the trials at that stage were excluding patients with haemophilia and most were excluding patients with cirrhosis. Furthermore, the data sheet for interferon alpha lists a low platelet count as a contraindication to therapy. I do not think that earlier diagnosis of hepatitis C would have altered Mr Murphy's management. As discussed previously, the diagnosis of hepatitis C does have a bearing on the

timing of transplantation. Because of recurrent disease post transplant, the tendency is towards less transplantation for symptoms in hepatitis C disease and more specifically transplantation in this condition for objective evidence of synthetic liver failure.

e) venous ulcer

The management of Mr Murphy's venous ulcer was successful, although it took a good deal of time and attention to detail

In summary:

Mr Murphy suffered hamophilia and he had a history of duodenal ulcer disease, inguinal herniae and haemarthroses due to his coagulopathy, consequent upon haemophilia. As a consequence of infusion of blood products he was infected with the hepatitis B virus, but recovered from this. He was also infected with the hepatitis C virus. This led to the complication of cirrhosis, portal hypertension and hepatocellular carcinoma.

Mr Murphy's liver disease was relatively well compensated for most of the time between diagnosis and 1994. Intermittently acute episodes of decompensation coincided with complications following knee and hernia surgery and the complications of portal hypertension and variceal bleeds. In general terms, Mr Murphy's liver condition was managed jointly by Dr Hay and Professor Shields, who frequently monitored liver function tests. Screening for primary liver cancer was not carried out. These tests showed that generally liver function was well preserved until mid 1994 and that Mr Murphy had not reached a point where liver transplantation would definitely be indicated on objective grounds. In mid 1994 his liver function deteriorated as a consequence of the manifestation of a large primary liver cancer. It was this development that precluded liver transplantation and caused his death.

Conclusion

I conclude that the standard of care of Mr Murphy was generally excellent. The family's distress is understandable, since the infection was contracted as a consequence of infected blood products. The various facets of Mr Murphy's disease were all managed from doctors of a high level of experience and expertise in the appropriate fields. There is not a proven case for screening for hepatoma in the setting of hepatitis C infection and failure to do so was not negligent. I do not believe there was negligence in failing to refer Mr Murphy earlier for a liver transplant and it was only thanks to the local expertise in the management of his variceal haemorrhage that the earlier need for transplantation was avoided.

The failure to act upon the results of investigations in July 1994, prior to Mr Murphy's transfer to Newcastle was most unfortunate, but did not alter Mr Murphy's outcome. The oversight did result in unreasonable family expectation at the time of transfer to Newcastle.

The delay in inititiating chemotherapy was not protracted and it is most unlikely that earlier chemotherapy could have prevented Mr Murphy's sudden death due to a presumed massive intra peritoneal haemorrhage.

Signed.. GRO-C Dated.. 9/X/97

Dr Mervyn H Davies MD MRCP