Statement number: S426

DOCUMENT RECORD PRINT

STATEMENT PRINT

Surname: MARSHALL

Forenames: JONATHAN CHARLES

Age: OVER 18 Date of Birth:

Number of Pages:

REPORT

regarding

Robert WILSON (BJC/55)

PREPARED BY: Dr J. MARSHALL

AT THE REQUEST OF: Hampshire Constabulary

CONTENTS

Statement number: S426

DOCUMENT RECORD PRINT

1. INSTRUCTIONS

I received instructions from D/Insp David GROCOTT for and on behalf of Hampshire Constabulary, to provide a medical report in relation to Mr Robert WILSON. To this end I was asked to review Mr WILSON's medical records which I have now done. I was asked to address the following issues:

1. Can you review the papers and establish whether or not Mr WILSON was suffering with alcohol related liver failure, if he was, does this mean that the prognosis is poor?

2. Can you establish at what point Mr WILSON entered what could be described as his "terminal Phase"

3. What treatment should have been considered in Mr WILSON's case? Especially in light of his increased weight (possibly due to fluids)

4. What would have been the impact on a patient with Mr WILSON's ailments when he is started on morphine from the 14th October. "*.....the effects of hepatitis or cirrhosis on drug deposition range from impaired to increased drug clearance in an unpredictable fashion..... the oral availability for high first class drugs such as Morphine.....is almost double in patients with cirrhosis compared to those with normal liver function. Therefore the size of the oral dose of such drugs should be reduced in this setting" (Harrison)* Harrisons Principles of Internal Medicine. Kesper, Braunwald, Fauci, Hauser, Longo, Jameson. 16th Edition New York 2005 (page 19).

5. In your opinion was the deterioration in Mr WILSON's condition in keeping with a progression in his liver disease or was it due to the increasing medication that he was receiving.

6. Could you comment on the fact that the cause of death that was recorded on the death certificate was Bronchopneumonia?

2. BRIEF CURRICULUM VITAE

Dr. Jonathan Charles MARSHALL MMBBSc MRCP MC

WORK: The Oxford Radcliffe NHS Trust
Department of Gastroenterology
Horton Hospital
Oxford Road
Banbury
Oxfordshire
OX16 9AL
Tel: 01295-229728
Fax: 01295-229603

Statement number: S426

DOCUMENT RECORD PRINT

GMC REGISTRATION NO. 3356395

CSST: Dual Accreditation General Medicine and Gastroenterology 1st June 2000

EDUCATION & QUALIFICATIONS:

MEDICAL SCHOOL:University College and The Middlesex, 1982 - 1988

HIGHER QUALIFICATIONS:

MD: December 2001 University of London

CSST: Medicine and Gastroenterology June 2000

MRCP: 1993

BSc: Physiology with Basic Medical Sciences: Upper Second Class, University College, London 1985

MBBS: University of London 1988

PROFESSIONAL TRAINING:

General Medical Training

Current:

Dual accreditation General Internal Medicine and Gastroenterology 1st June 2000.

Currently perform general medical duties at consultant level.

Medical on-call shared with Senior colleagues on alternative basis.

In-patient general medical commitment 1-2 ward-round per week.

Specialist Gastroenterology Training

Gastroenterology Career History:

Statement number: S426

DOCUMENT RECORD PRINT

Student elective: Professor COTTON, Duke University, North Carolina.

Basic endoscopic skills learnt as an SHO with Dr. BARRISON.

Clinical and endoscopic skills further developed at Welwyn Garden City.

The North Middlesex Hospital, presented a wide range of gastroenterological problems due to the ethnically diverse and mobile nature of the local population.

The Royal Free Hospital provided specialist hepatology, liver transplantation and inflammatory bowel disease training.

Research for MD thesis into *H.pylori* in alcoholic liver disease enabled development of a special interest in this area.

The Whittington allowed further development of general and therapeutic endoscopic skills.

King George Hospital, Ilford, enabled further development of therapeutic endoscopy including ERCP.

Currently perform two out-patient clinics and 2-3 endoscopy lists per week.

Endoscopic Training:

Trained to BSG guidelines for Upper Endoscopy (diagnostic and therapeutic), flexible sigmoidoscopy and colonoscopy (diagnostic and therapeutic).

Clinical lead for endoscopy on the Horton Hospital site for the Oxford Radcliffe NHS Trust.

PUBLICATIONS:

Published Papers and Abstracts

Marshall JC, Sharp E, BARRISON I.G.

'Once bitten, twice shy'. Multiple abscesses in an 18 year old female.

BMJ (1994) 309: 1694-1695.

Lagnado L, Marshall JC, Lodge L.

S-methyl-3-propanolamine (S-MDP) but neither papaverine nor noscapine is an N-methyl aspartate antagonist.

Neuroscience letters (1985) 21: 56A

Marshall JC, Gordon HM, Madden AM, Morgan MY
Alcohol consumption and Severity of Liver Disease Influences: *Helicobacter pylori* infection in cirrhotic liver disease.
Alcohol Clin and Exp (1998) 22: 172A

Marshall JC, Gordon HM, Madden AM, Morgan MY
Seroprevalence of *Helicobacter pylori* in Chronic Liver disease and its relation to Alcohol Misuse.
Hepatology (1998) 28: 199A.

Marshall JC, Morgan MY, Walker MM
Upper Gastrointestinal Pathology in relation to *Helicobacter pylori* Status in Alcohol Misusers Gut (1999) 44 A118

Marshall JC, Karim QN, Worku M, Morgan MY, Walker MM
Motility and Survival of *Helicobacter pylori* in Alcoholic Beverages.
Cut (1999) 45 A15

Wallace DF, Gordon HM, Marshall JC, Walker AP, Dooley JD, Morgan MY
The Role of HFE Mutation in Determining predisposition to Alcohol Related Cirrhosis in a Celtic Population.
Gut (1999) 45 A36.

Marshall JC, Karim QN, Worku M, Morgan MY, Walker MM
Motility and Survival of *Helicobacter pylori* in Organic and Non-Organic Alcohol Beverages.
Gut (2000) 46 A87.

Marshall JC, Lample F, Gordon, HM, Morgan MY
Seroprevalence of *Helicobacter pylori* is Influenced by Alcohol Consumption and Severity of Liver Injury Gastroenterology (2000) 118 A1270

Statement number: S426

DOCUMENT RECORD PRINT

Marshall JC, Karim QN, Worku M, Morgan MY, Walker MM
Motility and Survival of *Helicobacter pylori* in Alcoholic Beverages
Gastroenterology (2000) 118 A1356.

Chapters

Marshall JC, Mettler F. Management of accidentally radioactively contaminated patients. In Radiation Accidents ed Mettler.

Poster Presentations

Alcohol Consumption and Severity of Liver Disease Influences *Helicobacter pylori* infection in cirrhotic liver disease.

Poster Presentation, Ninth Congress of the International Society for Biomedical Research on Alcoholism (ISBRA) Copenhagen (1998).

Seroprevalence of *Helicobacter pylori* in Chronic Liver Disease and its Relation to Alcohol Misuse.

Poster presentation at the International Association for the Study of the Liver (IASL) Biennial Meeting Chicago (1998).

Upper Gastrointestinal Pathology in relation to *Helicobacter pylori* Status in Alcohol Misusers.

Poster presentation, British Society of Gastroenterology (BSG) Glasgow (1999)
Motility and Survival of *Helicobacter pylori* in Alcoholic Beverages
Poster Presentation, The European *Helicobacter pylori* Society Helsinki (1999)
Motibility and Survival of *Helicobacter pylori* in Organic and Non-Organic Alcohol Beverages.
Poster Presentation, British Society of Gastroenterology (BSG) Birmingham (2000)

Accepted Papers

Walker MM, Marshall JC

Helicobacter pylori and Gastric Pathology-Ask your Semmelier

Accepted Z. Gastroenterology December 2000

Marshall JC, Lample F, Morgan MY

Helicobacter pylori Infection and Hepatic Encephalopathy: The Problem of Confounding Variables

Accepted as poster International Meeting on Hepatic Encephalopathy Strasbourg November 2001

Papers Submitted or in Preparation

Marshall JC, Lample F, Madden M, Gordan H.M, Morgan MY

Seroprevalence of *Helicobacter pylori* in liver disease: Influence of liver disease and alcohol consumption in preparation for Gastroenterology

Marshall JC,Morgan MY, Walker MM

Chemical Gastritis is Not Influenced by Alcohol Consumption in preparation for J. of Clinical Pathology.

3. DOCUMENTATION

This Report is based on the following documents:

Full paper set of medical records of Robert WILSON. BJC/55

Summary of events as provided by the police.

Copy of death certificate.

4. CHRONOLOGY/CASE ABSTRACT

The numbers in brackets refer to the page of evidence.

Robert WILSON a 74 year old gentleman in 1998 attended Queen Alexandra Hospital, Portsmouth A&E Department on the 21st September 1998 (125-127) with a fracture of the left femoral head and tuberosity (169).

Mr WILSON had suffered many years before with Malaria and Diphtheria (143) but was first noticed to be abusing alcohol at the time of an endoscopy in 1994 (313). In 1997 he was admitted to hospital with a fall, epigastric pain and was found to have evidence of severe alcoholic liver disease (129). During the 1997 admission, an ultra sound showed a small bright liver compatible with cirrhosis and moderate ascites (129). His Albumin was very low at 19 (150) and a bilirubin was 48 (129). All these are markers of serious alcoholic liver disease with a poor long term prognosis. His weight was 100 kgs (152). There is no record of follow up attendance.

When he attends A&E it is originally intended to offer him an operation on his arm, which he refuses. However, he is kept in A&E overnight for observation (161-2). It becomes apparent by the next day that he is not well, is vomiting (163) and he is needing Morphine for pain (11). His wife is on holiday (11) and it is not thought possible for him to go home so he is transferred on 22nd September to the Care of the Elderly team at the Queen Alexandra Hospital (163).

The day after admission he is no longer thought fit enough to have an operation on his arm, although he would now be prepared to. He is recognised to have been an extremely heavy drinker with considerable oedema and abdominal distension on admission (167). He has abnormal blood tests on admission including a mild anaemia of 10.5 with a very raised mean cell volume of 113 and his platelet count is reduced at 133 (239). Five days later his haemoglobin has fallen to 9.7 and the platelet count has fallen to 123 (237). There are no further full blood counts in the notes, although his haemoglobin was normal with haemoglobin of 13 in 1997 (241).

He is noted to have impaired renal function with a Urea of 6.7 and a Creatinine of 185 on admission (209) and on 25th September Urea of 17.8 and a Creatinine of 246 (203). He is started on intravenous fluids on 27th September (12) and his renal function then continues to improve so that by the 7th October both his Urea and Creatinine are normal at 6.1 and 101 (199).

His liver function is significantly abnormal on admission and on 29th his albumin is 22, his bilirubin 82 (he would have been clinically jaundice) there is then little change over his admission. On the 7th October is albumin is 23 and his bilirubin also 82 (199). His AST is 66 (171).

His vomiting within 24 hours of admission may have been due to alcohol withdrawal but he had also been given Morphine for pain (11). He is started on a Chlordiazepoxide regime (11) as standard management plan to try and prevent significant symptoms of alcohol withdrawal. This has some sedative effects as well.

His physical condition in hospital deteriorates at first. He is noted to have considerable pain for the first 2 - 3 days, he is found to have extremely poor nutritional intake and has eaten little at home (12). His renal function deteriorates as documented above. He is communicating poorly with the nursing staff (28) and is restless at night on 30th September (30). His Barthel deteriorates from 13 on 23rd September to 3 on the 2nd October (69), his continued nutritional problems are documented by the dietician on 2nd October (16). In the nursing cardex he is vomiting, he has variable communication problems, he is irritable and cross on 1st October (30). On 4th October (16) his arm is noted to be markedly swollen and very painful and it is suggested he needs Morphine for pain (31). The following day he knocks his arm and gets a laceration (16).

There is ongoing communication with his family which iscomplicated by inter-family relationships between his first wife's family and his current wife. The plan by 6th October is that he will need nursing home care when he leaves hospital and his Barthel at this stage is 5 (16) (69). However on the 5th the nursing cardex note that he is starting to improve (32) although, he remains catheterised and has been faecally incontinent on occasion.

On 7th October is now more alert and is now telling the staff that he wishes to return home (17). The nursing staff notes that he is now much more adamant in his opinions (33). However on 8th he had refused to wash for 2 days (18). He is then reviewed at the request of the medical staff by a psycho-geriatrician. The opinion is that he has early dementia, which may be alcohol related and depression. He is noted to be difficult to understand with a dysarthria (117-118). He is started on Trazodone as an antidepressant and as a night sedative, he is still asking for stronger analgesics on 8th October (35). The letter also mentions (429) rather sleepy and withdrawn..... his nights had been disturbed.

On the 9th October an occupational therapy assessment is difficult because he is reluctant to comply and a debate occurs about whether he is capable of going home (19). By the 12th October (21) his Barthel has improved to 7 (69) so Social Services say that he no longer fits their criteria for a nursing

homeand he should now be considered for further rehabilitation (21). The nursing cardex notes that his catheter is out (35) he is eating better but he still gets bad pain in his left arm (36). His arms, hands and feet are noted to be significantly more swollen on 12th October (36). His weight has now increased from 103 kgs on 27th September to 114 kgs by 14th October (61,63). However his Waterlow score remains at "high risk" for all his admission (71). A decision is made to transfer him for possible further rehabilitation, although the medical review on 13th October states in view of the medical staff and because of his oedematous limbs, he is at high risk of tissue breakdown. He is also noted to be in cardiac failure with low protein and at very high risk of self neglect and injury if he starts to take alcohol again. He currently needs 24 hour hospital care (21).

On 14th October he is transferred to Draed Ward and the notes (179) say "for continuing care". The notes document the history of fractured humerus, his alcohol problem, current oedema and heart failure. No examination is documented. The notes state that he needs help with ADL, he is incontinent, Barthel 7, he lives with his wife and is for gentle rehabilitation. I am unable to read four words. The single word on the line above incontinence, two words after lives with wife (this may be a street address) and the word in front of gentle mobilisation.

The next medical notes (179) are on 16th October and state that he had declined overnight with shortness of breath. On examination he is reported to have a weak pulse, unresponsive to spoken orders, oedema plus plus in arms and legs. The diagnosis is "? silent MI, ? liver function" and the treatment is to increase the Frusemide. The nursing cardex for 14th October confirms he was seen by Dr BARTON , that Oramorphine 10 mgs was given and he was continent of urine. On 15th October the nursing notes 9265) state commenced Oramorphine 10 mgs 4 hourly for pain in left arm, poor condition is explained to wife. On 16th on the nursing cardex he is "seen by Dr KNAPMAN am as deteriorated overnight, increased Frusemide". However I find some possible confusion with the nursing care plan (278), this states for 15th October, settled and slept well, Oramorphine 20 mgs given 12 midnight with good effect, Oramorphine 10 mgs given 0600 hours. Condition deteriorated overnight, very chesty and difficulty in swallowing medications. Then on 16th it states has been on syringe driver since 1630 hours.

As will be seen from the analysis of the drug chart, Mr WILSON received the Oramorph at midnight on 15th and then 0600 hours Oramorph on 16th. The first clinical deterioration is on the night of 15th -

16th October not the night of the 14th - 15th October. The next medical note is on 19th October which notes that he had been comfortable at night with rapid deterioration (179) and death is later recorded at 2340 hours and certified by Staff Nurse Code A The nursing cardex mentions a bubbly chest late pm on 16th October (265). On the 17th Hyoscine is increased because of the increasing oropharyngeal secretions (265). Copious amounts of fluid are being suctioned on 17th.

He further deteriorates on 18th and he continues to require regular suction (266). The higher dose of Diamorphine on the 18th and Midazolam is recorded in the nursing cardex (266).

5. EXAMINATION OF THE FACTS IN ISSUE

1. Can you review the papers and establish whether or not Mr WILSON was suffering with alcohol related liver failure, if he was, does this mean that the prognosis is poor?

Response to question 1

Mr WILSON was suffering with chronic liver failure due to alcoholic cirrhosis in 1997. It is likely that he had cirrhosis of the liver as early as 1994. With cirrhosis and ongoing alcohol misuse survival is typically 6 months to one year. A case can therefore be made that by the time Mr WILSON was admitted in 1998, because he was still a "heavy drinker" [165], he had already exceeded his expected survival time with this condition. However, abstinence from alcohol can increase survival in cirrhosis by years or even decades. In addition patients generally younger than Mr WILSON who remain abstinent can be assessed for liver transplantation. It is therefore essential to try and promote abstinence from alcohol where at all practical.

2. Can you establish at what point Mr WILSON entered what could be described as his "terminal Phase"

Response to question 2

Mr WILSON entered a terminal phase at or around the 16th October 1998. There is an entry in the notes that he had declined overnight with SOB (shortness of breath). On examination at that time he had a weak pulse, was unresponsive to spoken commands and had oedema ++ in arms and legs. A differential diagnosis of a silent myocardial infarct (MI) or reducing renal function was proposed and his frusemide increased [179]. Three days prior to this event and prior to transfer to the continuing care ward (Drylands) there is an entry about a ward round [177]. It is commented that Mr WILSON still needed nursing and medical care and the concern on the ward round was that he was at risk of falling. While the handwriting is difficult to read, my interpretation of the comments was that Mr WILSON was unsteady on his feet, at risk of further falls but was mobilising at that time. While it was recognised that he had both ongoing medical and nursing needs he was clearly well enough for that assessment to take place [177]. There was clearly a marked deterioration from the 12/13th October through to the 16th October. Discussions moved from placement and mobilisation issues for Mr WILSON through to pre-terminal and ultimately terminal events from the 16th October onwards [179].

3. What treatment should have been considered in Mr WILSON's case? Especially in light of his increased weight (possibly due to fluids)

Response to question 3

Treatments that might have been considered in Mr WILSON's case were the administration of Pabrinex on admission (high dose B vitamins). This is to prevent Wernicke's encephalopathy. This condition causes *permanent* mental deterioration and is a result of alcohol misusers becoming very low in B vitamins. We know from a psychogeriatric assessment [175] that a working diagnosis of an early dementia, possibly alcohol related and/or depression was being considered on the 8th October 1998. It is impossible to say that a Wernicke's psychosis didn't play a small part in Mr WILSON's symptomatology but my view is that it is unlikely. A recurring theme throughout the record is of *variable* mental state and *varying* co-operation. It is likely that Mr WILSON had an ongoing *hepatic* encephalopathy to account for this. Hepatic encephalopathy refers to *temporary* and *reversible* mental clouding due to the injured liver being unable to clean the blood of toxic ammonia based compounds produced by gut bacteria. The level of these toxins in turn depends on frequency of bowel actions and the presence or absence of other factors such as drugs including alcohol.

In the ideal world more attention could have been paid to giving lactulose to help treat hepatic encephalopathy. In real life however, it is difficult to maintain patients on a rigorous lactulose regime as they often refuse to take this as it is unpalatable gives them frequent bowel actions. I would therefore conclude that Mr WILSON's management prior to transfer to Drylands ward was not perfect but very reasonable given the constraints of Mr WILSON's general behaviour and the challenging management issues that he posed.

Oedema had been noted on several occasions when Mr WILSON was an inpatient. Diuretics in the form of frusemide had been administered to help deal with this. My view is that the oedema was secondary to the liver disease and while the diuretics may improve the oedema its use is more for symptomatic benefit than any tangible therapeutic benefit. In addition use of diuretics in this clinical context risks putting the patient into renal failure-the so called 'hepatorenal syndrome.' [178].

4. What would have been the impact on a patient with Mr WILSON's ailments when he is started on morphine from the 14th October. ".....the effects of hepatitis or cirrhosis on drug deposition range from impaired to increased drug clearance in an unpredictable fashion..... the oral availability for high first class drugs such as Morphine.....is almost double in patients with cirrhosis compared to those with normal liver function. Therefore the size of the oral dose of such drugs should be reduced in this setting" (Harrison) Harrisons Principles of Internal Medicine. Kesper, Braunwald, Fauci, Hauser, Longo, Jameson. 16th Edition New York 2005 (page 19).

Response to question 4

The impact of regular morphine administration to Mr WILSON is likely to have hastened his decline. It's sedative effects would worsen hepatic encephalopathy which he undoubtedly had throughout his hospital stay and would cause rapid deterioration as indeed happened between the 14th and the 18th October.

While no specific mention is made in the clinical record of hepatic encephalopathy itself there were numerous mentions of liver disease and cirrhosis that should alert staff to the need for caution when prescribing opiate analgesia. In addition there is a nursing note from 14-10-98 [279] stating 'Bob may have a problem with constipation.' It was speculated that this was a side effect of opiate

analgesia. Constipation linked to opiate usage would be expected to worsen hepatic encephalopathy. Progression from 'confusion' through to coma would be a likely outcome in this clinical context.

The quote from Harrison's textbook summarise the pathophysiology of morphine in liver disease well. A more practical 'day-to-day' prescribing guide, available on any drug trolley or pharmacy, is the BNF (British National Formulary Number 43 March 2002). This states on p13 "Morphine is given by mouth as an oral solution or as standard (immediate release) tablets every 4 hours, the initial dose depending largely on the patient's previous treatment. A dose of 5-10mg is enough to replace a week of analgesic (such as Paracetamol .....) but 10-20mg or more is required to replace a strong one (comparable to Morphine itself)".

Under 'Morphine Salts' in the 'caution section' the BNF states that *Morphine Salts "may precipitate coma in hepatic impairment" (reduced dose or avoid but many such patients tolerate Morphine well).* Even if the full state of Mr WILSON's liver disease was not fully appreciated in terms of his hepatic encephalopathy there is ample warning in the day to day prescribing literature that morphine should be used sparingly and carefully monitored in this patient group. There does not appear to be any appropriate dose reduction steps taking in Mr WILSON's case or indeed monitoring for additional side effects.

Mr WILSON's received 10mg of morphine on 14th October and then was commenced on 10mg morphine 4 hourly from then on. No dose reductions appear to have been made as recommended by standard prescribing guides. Doses escalated upwards until syringe drivers containing diamorphine 40mg with hyocine and midazolam 20mg were administered [278]. These are 'terminal care' doses from which recovery could not be expected with advanced liver disease and hepatic encephalopathy.

5. In your opinion was the deterioration in Mr WILSON's condition in keeping with a progression in his liver disease or was it due to the increasing medication that he was receiving.

Response to question 5

The impact of regular morphine administration to Mr WILSON is likely to have hastened his decline. It's sedative effects would worsen hepatic encephalopathy which he undoubtedly had throughout his

hospital stay and would cause rapid deterioration as indeed happened between the 14th and the 18th October.

6. Could you comment on the fact that the cause of death that was recorded on the death certificate was Bronchopneumonia?

Response to question 6

The post mortem diagnosis of bronchopneumonia was not surprising. Bronchopneumonia is usually the terminal event in many conditions. It is a frustrating post mortem diagnosis for practising clinicians who having struggled and failed to make a definite diagnosis in life don't receive any useful information after death to improve knowledge. It is not however an uncommon situation. Morphine would have contributed to the development of pneumonia as part of it's respiratory depression side-effects. Similarly, hepatic encephalopathy leading to coma would increase the risk of aspiration which might in turn lead to and/or exacerbate bronchopneumonia. It would however be essential to get the pathologists opinion on this sequence of events.

6. OPINION

The management of Mr WILSON's liver condition following the time of initial admission was not perfect but reasonable. He should have received Pabrenex to prevent *Wernickes'* encephalopathy in addition to lactulose to treat *hepatic* encephalopathy.

Mr WILSON was assessed by a psychogeriatrician who did not detect any of the classical signs of Wernickes' encephalopathy. During most of his admission as well Mr WILSON was generally alert and so the omission of lactulose or other anti-encephalopathy treatment cannot be cited as a major omission. In real-life I suspect Mr WILSON would have refused to take lactulose for presumed encephalopathy because of its taste and laxative effects.

Mr WILSON was clearly an unwell man whose life expectancy was short. His previous record demonstrates that he would have been likely to return to drinking on discharge from hospital. The administration of high doses of morphine while an in-patient on Drylands however must be

considered reckless. Warnings about morphine usage in the context of liver disease are readily available in standard prescribing guides such as those cited from the BNF. No attempt appears to have been made to justify the use of opiates in this at risk patient group. There also does not appear to have been any attention paid to appropriate dose reduction and/or monitoring in Mr WILSON's case. The outcome was predictable in the clinical context of cirrhosis and escalating opiate dosage that Mr WILSON could not have survived.

7. LITERATURE/REFERENCES

British National Formulary (BNF) March 2002 number 43. [Any version of BNF will contain similar information under 'morphine'

8. EXPERTS' DECLARATION

1. I understand that my overriding duty is to the court, both in preparing reports and in giving oral evidence. I have complied and will continue to comply with that duty.

2. I have set out in my report what I understand from those instructing me to be the questions in respect of which my opinion as an expert is required.

3. I have done my best, in preparing this report, to be accurate and complete. I have mentioned all matters which I regard as relevant to the opinions I have expressed. All of the matters on which I have expressed an opinion lie within my field of expertise.

4. I have drawn to the attention of the court all matters, of which I am aware, which might adversely affect my opinion.

5. Wherever I have no personal knowledge, I have indicated the source of factual information.

6. I have not included anything in this report which has been suggested to me by anyone, including the lawyers instructing me, without forming my own independent view of the matter.

7. Where, in my view, there is a range of reasonable opinion, I have indicated the extent of that range in the report.

8. At the time of signing the report I consider it to be complete and accurate. I will notify those instructing me if, for any reason, I subsequently consider that the report requires any correction or qualification.

9. I understand that this report will be the evidence that I will give under oath, subject to any correction or qualification I may make before swearing to its veracity.

10. I have attached to this report a statement setting out the substance of all facts and instructions given to me which are material to the opinions expressed in this report or upon which those opinions are based.

9. STATEMENT OF TRUTH

I confirm that insofar as the facts stated in my report are within my own knowledge I have made clear which they are and I believe them to be true, and the opinions I have expressed represent my true and complete professional opinion.

Signed: J MARSHALL

Signature witnessed by: