DRAFT REPORT

regarding

Patient Name Robert Wilson (Ref No. BJC/55)

PREPARED BY: Professor R Baker……..

AT THE REQUEST OF: Hampshire Constabulary

CONTENTS

1. SUMMARY OF CONCLUSIONS

I have studied the copies of the records provided to me by Hampshire Constabulary in order to consider three issues – the certified cause of death, the prescription of opiates and sedatives, and whether Mr Wilson fell into the category of patients who might have left hospital alive.

With respect to death certification, I have concluded that the certificate was inaccurate in that Mr Wilson did not have renal failure, and had liver dysfunction but not failure. He probably did have heart failure, although I believe the initiation of opiate medication was an important factor in leading to death.

With respect to the prescription of opiate drugs, I have concluded, on the evidence available to me, that the initiation of opiate medication on transfer to Dryad ward was inappropriate; I have also concluded that the starting dose was too high. The prescription of hyoscine and midazolam was justified by the use of opiates.

With respect to leaving hospital alive, I have concluded that Mr Wilson was in the category of patients who might have left hospital alive if he had not been commenced on opiate medicate on transfer to Dryad ward.

1. INSTRUCTIONS

I have been asked to provide a statement of evidential use that could be used in the event of criminal proceedings arising from the case of Mr Robert Wilson.

2. ISSUES

I was asked to address three questions:

1. Certified cause of death. In this case, was the certified cause of death supported by the medical history of the patient?
2. Prescription of opiates and sedatives. In the case of Mr Wilson was his prescribing in accordance with his clinical need?
3. Leaving hospital alive. In my statement (080904) I had referred to patients who were administered opiates and eventually died who may have recovered and left hospital had they not received this medication. The issue to be addressed was whether, in my opinion, Mr Wilson fell into this category.

3. BRIEF CURRICULUM VITAE

Academic Qualifications

1975: MBBS (Royal Free Hospital School of Medicine, University of London)

1996: MD (University of London)

1980: MRCGP

1992: FRCGP (by assessment)

Current Posts

Head of Department of Health Sciences, University of Leicester.
Director, Clinical Governance Research and Development Unit
Division of General Practice and Primary Health Care,
University of Leicester

Director, Leicester provider unit of National Collaborating Centre – Primary Care (NCC-PC) of NICE.
Non-principal in General Practice.

Research

My principal research interest is quality of care, including methods of improving professional performance, patient experience of care, and patient safety.
I have published around 130 peer reviewed articles.

4. DOCUMENTATION

This Report is based on the following documents:

[1] Full paper set of medical records of Mr Robert Wilson, provided to me by Hampshire Constabulary.

[2] A copy of my report dated 08 September 2004.

[3] The Palliative Care Handbook Guidelines on clinical management fourth edition, of the Portsmouth Healthcare NHS Trust, Portsmouth Hospitals NHS Trust, and the Rowans (Portsmouth Area Hospice), 1998.

5. CHRONOLOGY/CASE ABSTRACT (prepared by Hampshire Constabulary) *The numbers in square brackets[ ] refer to the page of evidence.*

1.1. 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 humerus and tuberosity [169].

1.2. 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.

1.3. When he attends A&E in September 1998 with a fracture of his left humerus 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 1998 to the Care of the Elderly team at the Queen Alexandra Hospital [163].

1.4. 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].

1.5. 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].

1.6. His liver function is significantly abnormal on admission and on 29th his albumin is 22, his bilirubin 82 (he would have been clinically jaundiced) 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].

1.7. 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.

1.8. 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 reported as vomiting, having variable communication problems, and being 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].

1.9. There is ongoing communication with his family which is complicated 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 notes that he is starting to improve [32], although he remains catheterised and has been faecally incontinent on occasion.

1.10. 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 is also depressed. 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.

1.11. 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 home and he should now be considered for further rehabilitation [21]. The nursing cardex notes that his catheter is out [35] and 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].

1.12. On 14th October he is transferred to Dryad Ward and the notes [179] say "for continuing care". The notes document the history of fractured humerus, his alcohol problem, recurrent 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.

1.13. 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 [265] state commenced Oramorphine 10 mgs 4 hourly for pain in left arm, poor condition is explained to wife. According to the cardex on 16th he is "seen by Dr Knapman am as deteriorated overnight, increased Frusemide".

1.14. (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 06.00 hours. Condition deteriorated overnight, very chesty and difficulty in swallowing medications. Then on 16th it states has been on syringe driver since 16.30 hours. As will be seen from the analysis of the drug chart, Mr Wilson received the Oramorph at midnight on 15th and then 06.00 hours Oramorph on 16th. The first clinical deterioration is on the night of 15th – 16th October not the night of the 14th – 15th October.)

1.15. 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 23.40 hours and certified by Staff Nurse Collins. 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].

1.16. Two Drug Charts: The first is the Queen Alexandra drug chart [106-116]. This records the regular laxatives, vitamins and diuretics given for his liver disease. The reducing dose of Chlordiazepoxide stops on 30th September for his alcohol withdrawal and the Trazodone started for his mild depression and night sedation. In terms of pain management Morphine, slow IV or subcutaneous 2.5 – 5 mgs written up on the prn side and 5 mgs given on 23rd September and 2.5 mgs twice on 24th September. Morphine is also written up IM 2 – 5 mgs on 3rd October and he receives 2.5 mgs on 3rd and 2.5 mgs on 5th. He is also written up for prn Codeine Phosphate and receives single doses often at night up until 13th October but never needing more than 1 dose a day after 25th September. Regular Co-dydramol starts on 25th September until 30th September when it is replaced by 4 times a day regular Paracetamol which continues until his transfer.

In summary, his pain relief for the last week in the Queen Alexandra is 4 times a day Paracetamol and occasional night time dose of Codeine Phosphate.

1.17. The second drug chart is the drug chart of the Gosport War Memorial Hospital [258-263]. His diuretics, anti-depressant, vitamins and laxatives are all prescribed regularly. The regular Paracetamol is not prescribed but is written up on the as required (prn) after the drug chart. This is never given. Regular prescriptions also contains Oramorphine 10 mgs in 5 mls to be given 10 mgs 4 hourly, starting on 15th October [261]. 10 mgs is given at 10 am, 2pm and 6 pm on 15th, 6am, 10 am and 2 pm on 16th. A further dose of 20 mgs at night given at 10 pm is given at 10 pm on 15th October. Although these prescriptions are dated 15th October it is not clear if they were written up on the 14th or 15th.

1.18. On a further sheet of this drug chart [262] regular prescription has been crossed out and prn written instead. Oramorphine, 10 mgs in 5 mls, 2.5 – 5 mls 4 hourly is then prescribed on this sheet. It is not dated but it would appear 10 mgs is given at 2.45 on 14th October and 10 mgs at midnight on 14th October. Further down this page Diamorphine 20 – 200 mgs subcut in 24 hours from Hyoscine 200 – 800 micrograms subcut in 24 hours, Midazolam 20 – 80 mgs subcut in 24 hours are all prescribed. It is not clear what date these were written up. The first prescription is 16th October and the 20mls of Diamorphine with 400 micrograms of Hyoscine are started at 16.10. On 17th October, 20 mgs of Diamorphine, 600 micrograms of Hyoscine are started at 5.15 and the notes suggest that what was left in the syringe driver at that stage was destroyed [262]. At 15.50 hours on 17th October, 40 mgs, 800 mgs of Hyoscine and 20 mgs of Midazolam are started and on 18th 60 mgs of Diamorphine, 1200 micrograms of Hyoscine (a new prescription has been written for the Hyoscine) and 40 mgs of Midazolam are started in the syringe driver at 14.50 and again the notes suggest the remainder that was previously in the syringe driver is destroyed.

6. TECHNICAL BACKGROUND / EXAMINATION OF THE FACTS IN ISSUE

Figures in square brackets [ ] refer to page numbers of the notes.

1. Certified cause of death. In this case, was the certified cause of death supported by the medical history of the patient?

The certified cause of death was Ia congestive cardiac failure, Ib renal failure, II liver failure. The certifying doctor was Dr E.J. Peters.

Liver failure

Mr Wilson was known to have a poorly functioning liver. The primary diagnosis relating to his admission between 17/02/97 and 12/03/97 was alcoholic liver disease [129], and at that time he had abnormal liver function tests including low albumin level, and an ultrasound had shown a small liver, possibly cirrhotic, with marked ascites.

His liver function was also impaired at the time of admission in September 1998 [207, 199]. Jaundice does not seem to have been remarked upon in the notes relating to this admission. The working diagnosis during the admission in Queen Alexandra Hospital was active alcoholic hepatitis [171]. A hand written entry in the records dated 13/10/98 records results of blood tests taken 12/10/98 [178]. At that time, the bilirubin had fallen to 48 umol/L and the AST to 37 IU/L, although the alkaline phosphatase was 181 IU/L. I would tend to interpret these results as indicating some improvement. The notes do not record a diagnosis of liver failure although this diagnosis is mentioned on blood test forms [199, 213, 217]. The liver function tests, whilst abnormal, are not sufficiently abnormal to suggest fulminant liver failure. Diuretics can precipitate hepatic encephalopathy in patients with cirrhosis (Jones, 2003), but the hepatic encephalopathy was not diagnosed and the records do not include mention of the signs of encephalopathy. Mr Wilson was noted to have some depression and mildly impaired short term memory when assessed by Dr Luznat, the consultant in old age psychiatry on 08/10/98 [118, 119], and the nursing records indicate he was sleepy

and had poor speech on 29/09/98 [29], but these features were not sufficiently consistent, progressive or severe to suggest hepatic encephalopathy. The course of Mr Wilson's final illness was one of gradual if limited progress until transfer to Dryad ward, which tends to rule out the progressive development of encephalopathy due to liver failure.

Renal failure

Mr Wilson also had renal dysfunction. His creatinine reached 246 umol/l and his urea 17.8 mmol/l on 25/09/98 [213], but there was some improvement over the following days. On 30/09/98 his creatinine was 165 umol/l and his urea 14.4 mmol/l [203], and by the 05/10/98 his creatinine had fallen to 97 umol/l and his urea to 7.5 mmol/l [201]. The results on the 05/10/98 were within the normal range, and remained so on 07/10/98 and 13/10/98 [178]. The improvement in renal function appears to have occurred following the temporary withdrawal of diuretics and the institution of intravenous fluids [170, 89] on 28/09/98.

Congestive cardiac failure

The note on admission to Dryad ward records the problems of 'alcohol problems', recurrent oedema, and CCF (congestive cardiac failure). Heart failure is a syndrome rather than a specific disease, that is, it is a collection of symptoms and signs that can be caused by several different diseases. Congestive cardiac failure is a term that is less commonly used today. It can mean different things to different doctors (Fry and Sandler, 1993), and may indicate right ventricular failure to some doctors, left ventricular failure to others, or failure of both ventricles to others. Mr Wilson had ankle, leg and sacral oedema which may have been explained by right heart failure (the low albumin level secondary to the alcoholic liver disease and poor nutrition would also have played a role in causing the oedema), although he did not have a raised jugular venous pressure [166] when admitted to Queen Alexandra Hospital. He did have 'crackles' in the lung bases especially the left, and this might have been a

feature of left heart failure [166]. Diagnosis of cardiac failure on clinical grounds alone is difficult (Khunti et al, 2000).

The notes indicate that Mr Wilson suffered from retention of fluid leading to swelling of his arm [174] and legs [81, 129, 118, 265]. Potential explanations for heart failure in Mr Wilson's case include ischaemic heart disease and alcohol induced cardiomyopathy. He was treated with high doses of diuretics at his admission in 1997, specifically spironolactone 100mgs daily and frusemide 80 mgs daily [129]. During the admission in 1997, his weight declined from around 103kgm to around 93 kgm, suggesting that the diuretics had produced a satisfactory diuresis [367, 369]. In contrast, in 1998, his weight rose from 103 kgms on 27/09/98 [65] to 114 kgm on 14/10/98 [61], despite continued treatment with diuretics. This suggests that his cardiovascular status may have declined between the admissions in 1997 and 1998.

The medical notes on transfer to Dryad on 14/09/98 do not mention the need for additional treatment of the congestive cardiac failure [179]. Diuretics were continued, and Oramorph 10mg was prescribed, doses being given that day at 14.45 pm and 23.45 pm [262, 265]. However, there was no mention of pain at all in the medical records [179] and therefore the indications for Oramorph are unclear. Oramorph 10mg 4 hourly was commenced on 15/10/98, the first dose being given at 10.00 am, six doses being given up to 14.00 on 16/10/98. Mr Wilson was seen the next morning by Dr Knapman as he had declined overnight with shortness of breath. On examination he was reported as bubbling, had a weak pulse, unresponsive to spoken orders, and had oedema ++ in the arms and legs. The possibility of a silent myocardial infarct was raised (although not investigated), and the history of reduced liver function noted. The dose of frusemide was doubled. These notes indicate that Dr Knapman thought that congestive failure was an important factor in explaining Mr Wilson's condition. However, the fact that the deterioration coincided with the regular administration of Oramorph points to an alternative explanation, namely the side effects of opiate

medication. The side effects would include sedation leading to lack of responsiveness, and reduced ability to expectorate which could explain the 'bubbling' respiration.

In the afternoon of 16/10/98, the nursing staff noted that Mr Wilson was 'very bubbly', and that diamorphine by syringe driver had been commenced [265]. The dose began at 16.10 pm, and the prescription was written by Dr Barton [262]. The bubbly chest may have been explained by morphine. Hyoscine was also prescribed by syringe driver, midazolam being added on 17/10/98, the dose of diamorphine being increased to 40 mgs on 17/10/98 [278], and on the 18/10/98 to 60mgs [262].

2. Prescription of opiates and sedatives. In the case of Mr Wilson was his prescribing in accordance with his clinical need?

Mr Wilson was receiving soluable paracetamol four times daily from 30/09/98 until the morning of 14/10/98, prior to his transfer to Dryad ward [114, 115]. He had received 2.5-5mg morphine on 23-24/09/98 and 2.5mg on 3/10/98 and 5/10/98 [106.107], and he had also received codydramol until the paracetamol had been started. Although he did have pain throughout his stay in Queen Alexandra Hospital, it appears to have been reasonably well controlled by 13/10/98. The nursing record indicates that he had no complaints about pain on 13/10/98. nor on the morning of 14/10/98 [37]. Neither the medical or nursing records from Dryad ward mention an increase in pain later on the 14/10/98 [179, 265], although the nursing notes on 15/10/98 state that the Oramorph was for pain in the arm. On the information contained in the records, therefore, the commencement of Oramorph was not adequately justified.

The commencement of subcutaneous diamorphine on 16/10/98 followed a decline in Mr Wilson's condition, the cause of which was not clear [179]. The nursing records mention that the reason for commencing diamorphine by syringe driver was explained to the family, but the reason itself is not recorded in the records. An alternative approach to the decline on 16/10/98 would have been to stop the Oramorph and

observe whether Mr Wilson improved. For some reason which cannot be found in the records, it had been concluded that Mr Wilson was not going to recover and that terminal care was the appropriate course of action. Hyoscine was also prescribed, and I assume the intention was to control secretions. The dose of hyoscine was increased in accordance with the problems caused by the secretions (which were recorded as 'copious' on 17/10/98 [265]). The dose of diamorphine was increased, and midazolam was added, although the records do not explain the reasons for these prescribing decisions.

2. Leaving hospital alive. In my statement (080904) I had referred to patients who were administered opiates and eventually died who may have recovered and left hospital had they not received this medication. The issue to be addressed was whether, in my opinion, Mr Wilson fell into this category.

   The comment referred to from my statement (080904) is:

   As made clear in the report, I became concerned about aspects of care at Gosport War Memorial Hospital, including aspects of the care provided by Dr Barton. I concluded that it was probable that a small number of patients who had been given opiates and had died might, if they had not been given opiates, have sufficiently recovered to be discharged from hospital eventually. An attitude or culture of limited hope and expectations of recovery appeared to have existed at the hospital. I was unable to identify when this culture had first gained hold at the hospital and it may have existed before Dr Barton's appointment in 1988. In addition, I have not identified the underlying motivations responsible for this culture.

   When Mr Wilson was transferred from Queen Alexandra Hospital to Dryad ward, he was in need of nursing and medical care and at risk of falling until fully mobilised. A short spell in a long term NHS bed was regarded as appropriate when he was reviewed on the ward round on 13/10/98 [177,178]. He appeared to be making some progress,

with improved renal function, less pain, and improvement in some of the measures of liver function [178]. He still had significant problems, however, including difficulty in moving and oedema [81]. Nevertheless, the Queen Alexandra Hospital records do not indicate that death was expected in the near future – with appropriate care, gradual mobilisation was anticipated. Yet shortly after admission to Dryad ward, he was commenced on regular Oramorph.

8. OPINION

1. Certified cause of death. In this case, was the certified cause of death supported by the medical history of the patient?

In my opinion, Mr Wilson had liver dysfunction but not full blown failure. His liver dysfunction did not cause death. In the presence of other life-threatening conditions, the liver dysfunction may impair the ability to recover, and it would have been reasonable to mention on the death certificate that Mr Wilson had chronic liver disease. The cause of his liver disease – alcohol – was not mentioned on the ceritificate.

Mr Wilson did not have renal failure. He did have abnormal blood test results after his admission to hospital, but these improved with rehydration. Mr Wilson probably did have cardiac failure. There may have been other conditions as well. Haemoglobin estimations during his admission to Queen Alexandra Hospital had indicated mild anaemia. If this condition had deteriorated, the heart failure would also have become worse. However, I think this is rather unlikely since he was being closely observed in Queen Alexandra Hospital and signs of increasing anaemia would almost certainly have been recognised. Evidence of bleeding would have been noted if it had occurred. There is no convincing evidence in the records to

confirm a diagnosis of myocardial infarction such as history of chest pain, raised cardiac enzymes or ECG evidence. One could also speculate about possible occurrence of some unsuspected condition. However, despite all these speculations, it has to be acknowledged that his decline was associated with the regular administration of morphine, and was responded to by administration of diamorphine by syringe driver. The reason for commencing Oramorph is not recorded in the medical notes [179]; in particular, the reasons for not using a non-opiate drug for pain relief are not given. Even if Mr Wilson did have pain from the fracture that was not controlled by paracetamol, regular does of 10mg of oral morphine would not have been the appropriate treatment. Other non-opiate or weak opiate medication should have been used first. If these medications had failed to adequately reduce the pain, a low dose of morphine (2.5-5mg) as had been used in the early days of his admission might have been reasonable. Although Mr Wilson did have congestive cardiac failure, therefore, his death would have been hastened by opiate administration and the path to death may well have been initiated by the commencement of Oramorph on 14/10/98.

It is important to note that the general standard of completion of death certificates is unsatisfactory. For example, in a review of 1000 counterfoils of certificates in one teaching hospital in 1999-2000, only 55% of certificates had been completed to a minimally accepted standard (Swift and West, 2002). Of the remaining certificates, 25% had incomplete data, in 11% the part II section had been used inappropriately, and 9% were illogical or inappropriate. In her third report from the Shipman Inquiry, Dame Janet Smith observed: *A further problem with the current system is that the quality of certification is poor. Doctors receive little training in death certification.* (paragraph 17, page 4, Shipman Inquiry). The standard of completion

of the death certificate in Mr Wilson's case should therefore be regarded as fairly typical. Although Mr Wilson did not have renal failure, the history of recent abnormal renal function tests prompted use of this diagnosis; the mention of liver failure was probably a convenient way of describing the impaired liver function.

2. Prescription of opiates and sedatives. In the case of Mr Wilson was his prescribing in accordance with his clinical need?

The records do not contain information to explain why opiates were commenced. On the basis of the records alone, therefore, the prescribing of opiates was not indicated. The sedative midazolam was prescribed to accompany the diamorphine in the syringe driver, although the reason for the addition of midazolam is not given in the medical or nursing records.

The Palliative Care Handbook, fourth edition, published by the Portsmouth Healthcare NHS Trust, Portsmouth Hospitals NHS Trust and the Rowans (Portsmouth Area Hospice) in 1998 reproduces the WHO analgesic ladder in which step 1 (mild pain) involves the use of non opioids such as paracetamol, step 2 (moderate pain) weak opioids such as cocodamol [codeine and paracetamol], and step 3 (severe pain) strong opioids such as morphine. In Mr Wilson's case, medication for pain moved from step 1 to step 3 without any explanation. Hyoscine hydrobromide 0.4-2.4 mg over 24 hours by syringe driver is recommended in the Handbook for reducing secretions and is noted to be an excellent sedative. Midazolam 5-60mg over 24 hours is described as a sedative, higher doses to be used only for terminal sedation. The Handbook also indicates that a total daily dose of 30mg of morphine would be equivalent to 10mg of diamorphine by syringe driver in 24 hours.

The Handbook recommends starting morphine at a low dose and increase gradually according to need. This policy was applied in Queen Alexandra Hospital when occasional low (2.5-5mg) doses of morphine were needed early in Mr Wilson's admission. On Dryad ward, however, the starting dose was 10mg; on the 15/10/98 he had three doses of 10mg, and one at 10 pm of 20mgs (the time of this dose appears to be 22.00 hrs in the prescription record but is given as 24.00 hrs in the nursing record). This is a significant amount of opiate, more than would have been indicated even if step 2 of the WHO analgesic ladder had been tried first, and I would have expected sedation and drowsiness to occur.

My September 1998 copy of the British National Formulary (BNF; issue 36) notes that morphine 'may precipitate coma in hepatic impairment (reduce dose or avoid but many such patients tolerate morphine well); reduce dose or avoid in renal impairment' (page 201). It also states that in palliative care these cautions should not necessarily be a deterrent to the use of opioids.

The use of hyoscine to reduce secretions is common practice. Opiates can suppress the cough reflex, which reduces the ability to clear secretions (Schug and Cardwell, 2003). It also occurs in people who are too weak to expectorate effectively (Twycross and Lack, 1990). Midazolam, a benzodiazepine sedative, can be added to hyoscine if repeated administration of hyoscine leads to an agitated or confused state.

3. Leaving hospital alive. In my statement (080904) I had referred to patients who were administered opiates and eventually died who may have recovered and left hospital had they

not received this medication. The issue to be addressed was whether, in my opinion, Mr Wilson fell into this category.

In judging whether Mr Wilson might, if Oramorph had not been initiated on transfer to Dryad ward, eventually left Gosport War Memorial Hospital, several qualifications must be made. I am reliant on the hospital records only; records are often incomplete and I have not sought or obtained any information directly from the doctors, nurses, other staff or relatives who were involved in caring for Mr Wilson in the last days of his life. It is also difficult to predict with certainty the course of recovery that a patient will follow, especially when the patient is elderly and has a complex mix of several serious clinical problems, as did Mr Wilson. In addition to deterioration of existing conditions, new and unexpected problems can arise, including for example myocardial infarction [179]. It is also impossible to be certain about the degree of recovery, and whether the patient would have been fit for discharge to their own home or whether residential or nursing accommodation would be required. Bearing these qualifications in mind, in my opinion, Mr Wilson did fall into the category of patients who might have left hospital alive if the Oramorph had not been commenced on transfer to Dryad ward.

9. LITERATURE/REFERENCES

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10. 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 are 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.

11. 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.

Signature:_____ Date:_____