GENERAL MEDICAL COUNCIL

-and-

DR BARTON

FFW/64/01

ROBERT WILSON

6053009 v

Field Fisher Waterhouse

GENERAL MEDICAL COUNCIL

-and-

DR BARTON

ROBERT WILSON

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GENERAL MEDICAL COUNCIL

DR BARTON

Index to Robert Wilson's File

File 1

1. Summary of Evidence. Missing.
2. Report prepared by Dr Andrew Wilcock dated 21 May 2006. ✓
3. Report prepared by Professor R Baker dated February 2006. ✓
4. Interview of Dr Jane Ann Barton dated 19 May 2005 at 09:04. ✓
5. Statement of Dr Jane Barton regarding Robert Wilson. Missing.
6. Witness List and Witness Statements given to Hampshire Constabulary. ✓
   - (a) Iain Alister Wilson dated 9 March 2004. M
   - (b) Iain Alister Wilson dated 28 February 2005. M
   - (c) Keran Lynn Edwards dated 7 April 2004. ✓
   - (d) Neil Huntingdon Wilson dated 13 April 2004. M
   - (e) Lynne Joyce Barrett dated 15 April 2005. M
   - (f) Mollie Edwards dated 20 April 2004. ✓
   - (g) Gillian Kimbley dated 26 April 2004. M
   - (h) Lesley Ann Clarke dated 9 August 2004. ✓
   - (i) Tracie Huntington dated 24 February 2005. ✓
   - (j) Tracie Huntington dated 2 June 2005. ✓
   - (k) David Andrew Huntington dated 28 February 2005. ✓

(l) Robert Logan dated 6 March 2005.

(m) Freda Vaughan Shaw dated 14 April 2005.

(n) Siobhan Marie Collins dated 24 April 2005.

(o) Siobhan Marie Collins dated 22 June 2005.

(p) Kathryn Taylor-Barnes dated 9 May 2005.

(q) Sandra Margaret Milner dated 11 May 2005.

(r) Debra Barker dated 13 May 2005.

(s) Althea Everesta Geradette Lord dated 19 May 2005.

(t) Carole Jane Arnold dated 20 May 2005.

(u) Christopher Scott Yates dated 23 May 2005.

(v) Christopher Scott Yates dated 7 September 2005.

(w) John Albert Henry Grunstein dated 2 June 2005.

(x) Jacqueline Ann Spragg dated 6 June 2005.

(y) Margaret Rose Perryman dated 7 June 2005.

(z) Gillian Elizabeth Hamblin dated 11 June 2005.

(aa) Gillian Elizabeth Hamblin dated 30 September 2005.

(bb) Jeanette Elizabeth Florio dated 16 June 2005.

(cc) Rosie Lusznat dated 17 June 2005.

(dd) Marjorie Jane Wells dated 29 June 2005.

(ee) Marjorie Jane Wells dated 19 October 2005.

(ff) Shirley Sandra Hallmann dated 11 July 2005.

(gg) Shirley Sandra Hallmann dated 27 July 2005.

(hh) Geoffrey James Quade dated 14 July 2005.

(ii) Irene Margaret Dorrington dated 19 July 2005.

(jj) Irene Margaret Dorrington dated 6 October 2005.

(kk) Irene Margaret Dorrington dated 17 October 2005.

(ll) Irene Margaret Dorrington dated 15 February 2006.

(mm) Ewenda Jay Peters dated 21 July 2005.

(nn) Cristian Birla dated 25 July 2005.

(oo) Christopher John Hand dated 27 July 2005.

(pp) Lindsey Ann Hay dated 27 July 2005.

(qq) Deidre Durrant dated 3 August 2005.

(rr) Kathryn Mary Robinson dated 14 September 2005.

(ss) Claire Louisa Dyson dated 6 October 2005.

(tt) Claire Lousia Dyson dated 25 January 2006.

(uu) Ruth Clemon dated 2 November 2005.

(vv) Ruth Clemon dated 21 January 2006.

(ww) Anthony Howard Mowbray dated 11 November 2005.

(xx) Arumugam Ravindrane dated 16 November 2005.

(yy) Arumugam Ravindrane dated 25 November 2005.

b) (zz) Nicola Jayne Haynes dated 25 November 2005.

c) (aaa) Collette Billows dated 25 November 2005.

d) (bbb) Collette Billows dated 9 January 2006.

e) (ccc) Timothy Tayler dated 8 December 2005.

(ddd) Anthony Charles Knapman dated 20 January 2006.

(eee) Jonathan Charles Marshall dated 28 April 2006.

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DRAFT REPORT

regarding

ROBERT WILSON (BJC/55)

PREPARED BY: Dr Andrew Wilcock MB ChB FRCP DM
Reader in Palliative Medicine and Medical Oncology

AT THE REQUEST OF: Hampshire Constabulary

CONTENTS

1. SUMMARY OF CONCLUSIONS

Mr Wilson was a 74 year old man who was admitted to hospital after falling over and fracturing the greater tuberosity of his left humerus. He had multiple serious medical problems; alcohol-related cirrhosis leading to liver failure and encephalopathy, heart failure and kidney failure. Other problems included early dementia, depression and a high level of dependency. Although the care he received at Queen Alexander Hospital led to Mr Wilson being mentally more alert and returned his kidney function to normal, he continued to become increasingly oedematous despite the re-introduction of his diuretic therapy which was considered due to heart failure. The pain he experienced from his fracture progressively improved as anticipated and during his time at Queen Alexander Hospital, his daily analgesic requirements reduced from the equivalent of 20mg to 3mg of oral morphine. Nevertheless, given the time it takes for a fracture to heal, it was not surprising that pain on movement was still present at the time of his transfer. There are no concerns regarding the care proffered to Mr Wilson at the Queen Alexander Hospital.

On transfer to Dryad Ward, the care proffered to Mr Wilson by Dr Barton and Dr Knapman fell short of a good standard of clinical care as defined by the GMC (Good Medical Practice, General Medical Council, July 1998 pages 2–3) with particular reference to a lack of clear note keeping, adequate assessment of the patient (Dr Barton and Dr Knapman) and providing treatment that could be excessive to the patients needs (Dr Barton). No pain assessment was carried out on Mr Wilson, but his only regular analgesic, paracetamol, was discontinued and prescribed p.r.n. (as

required). Instead of his usual codeine 15–30mg p.r.n., approximately equivalent to morphine 1.5–3mg, he was prescribed morphine 5–10mg p.r.n. for pain relief. He received two doses of 10mg (a total of 20mg/24h) and the next day commenced on regular morphine 10mg every 4h and 20mg at night. In total he received 50mg of morphine in this 24h period, representing a larger dose than that he received in the initial 24h after his fracture. This is against the general expectation that pain from a fracture would have been improving over time and, without a clearly documented pain assessment, it is difficult to justify. However, the impact of this dose of morphine on Mr Wilson is impossible to judge because he deteriorated rapidly in the early hours of the 16th October 1998. The nature of his rapid decline and subsequent death were in keeping with worsening heart failure with or without a sudden event such as a heart attack. This, combined with his liver failure, could easily have precipitated his terminal decline. His reduced level of consciousness could have been due to a hepatic coma precipitated by the morphine or by a reduced level of blood oxygen secondary to the excess fluid on the lungs (pulmonary oedema) due to the heart failure. Later that day a syringe driver was commenced containing diamorphine 20mg/24h and increased over the next 48h to 60mg/24h, equivalent to oral morphine 120–180mg/24h. This increase in dose appears difficult to justify, as Mr Wilson was not reported to be distressed by pain, breathlessness or the secretions and was likely to be excessive for his needs. However, because heart and liver failure could also have led to a reduced level of consciousness, in my opinion, it is difficult to state with

any certainty that the doses of morphine or diamorphine he received would have contributed more than minimally, negligibly or trivially to his death

2. INSTRUCTIONS

To examine the medical records and comment upon the standard of care afforded to the patient in the days leading up to his death against the acceptable standard of the day. Where appropriate, if the care is felt to be suboptimal, comment upon the extent to which it may or may not disclose criminally culpable actions on the part of individuals or groups.

3. ISSUES

3.1 Was the standard of care afforded to this patient in the days leading up to his death in keeping with the acceptable standard of the day?

3.2 If the care is found to be suboptimal what treatment should normally have been proffered in this case?

3.3 If the care is found to be suboptimal to what extent may it disclose criminally culpable actions on the part of individuals or groups?

4. BRIEF CURRICULUM VITAE

Code A

Code A

5. DOCUMENTATION

This Report is based on the following documents:

[1] Full paper set of medical records of Robert Wilson, including the medical certificate of cause of death.

[2] Full set of medical records of Robert Wilson on CD-ROM.

[3] Operation Rochester Briefing Document Criminal Investigation Summary.

[4] Hampshire Constabulary Operation Rochester Guidance for Medical Experts.

[5] Hampshire Constabulary Summary of Care of Robert Wilson.

[6] Palliative Care Handbook Guidelines on Clinical Management, Third Edition, Salisbury Palliative Care Services (1995); also referred to as the 'Wessex Protocols.'

[6] Portsmouth Health Care NHS Trust Policies:

i) Control of Administration of Medicines by Nursing Staff Policy (January 1997).

ii) Prescription Writing Policy (July 2000).

iii) Policy for Assessment and Management of Pain (May 2001).

iv) Compendium of Drug Therapy Guidelines, Adult Patients (1998).

v) Draft Protocol for Prescription Administration of Diamorphine by Subcutaneous Infusion, Medical Director (December 1999).

vi) Medicines Audit carried out by the Trust referred to as Document 54 on page 52 in the Chi Report (reference 6).

[7] General Medical Council, Good Medical Practice (July 1998).

[8] British National Formulary (BNF). Section on Prescribing in Terminal Care (March 1998).

[9] British National Formulary (BNF). Section on Prescribing in the Elderly (March 1998).

[10] Statement of Dr Jane Barton as provided to me by Hampshire

Constabulary (undated).

[11] Statement of Dr Jane Barton RE: Robert Wilson, 19th May 2005.

[12] Draft Report regarding Statement of Dr Jane Barton RE: Robert Wilson (BJC/55), Dr A Wilcock, 18th January 2006.

[13] Draft overview of Robert Wilson (BJC/55), Dr A Wilcock, 24th November 2005.

[14] Report regarding Robert Wilson, Dr J Marshall, April 2006.

6. CHRONOLOGY/CASE ABSTRACT

Events at Queen Alexander Hospital, September 21st–October 14th 1998

Mr Wilson, a 74 year old man, who lived at home with his wife, was seen in Accident and Emergency on the evening of the 21st September 1998 (page 157 of 642). He had been drinking alcohol and had fallen onto his left shoulder. An x-ray revealed a fracture of the greater tuberosity of the left humerus with 'some displacement' of the fragment (pages 141 and 157 of 642). For pain relief he received morphine 10mg IV (as cyclimorph) at 20.45h; followed by a prescription for morphine 10mg IV at 21.00h, it is unclear if this was given, as a time of administration is not stated (page 157 of 642). His fracture was managed with immobilisation using a sling and analgesia (page 159 of 642). It was necessary to admit Mr Wilson as there was no one at home (his wife was on holiday in Plymouth) and he was feeling nauseated (page 159 of 642).

On the 22nd September 1998, he received 2 kapake (co-codamol 30/500; each tablet containing codeine phosphate 30mg, paracetamol 500mg) at 07.00h. It is unclear if he was receiving this regularly or p.r.n. 'as required'. He was noted to be confused at times (page 161 of 642). Vomiting was a problem and he reported that this normally happened after he had been drinking 'so much alcohol' (page 161 of 642). Mr Wilson was reviewed in fracture clinic where it is reported that he was not keen to undergo surgical fixation of the fracture (page 141 of 642). (He subsequently changed his mind, although by the time of his orthopaedic review on the 6th October 1998, he had again decided against surgery, but was, in any case, then considered unsuitable for surgery (pages 161 and 333 of 642)). As Mr Wilson felt unwell, was vomiting and unsteady on his feet, it became clear that he would not be able to manage at home and he was transferred to Dickens Ward (page 163 of 642).

Mr Wilson was fully clerked at 02.00h on the 23rd September 1998 (page 165 of 642). He reported an increasing cough for the past 6 months, productive of white sputum; vomiting associated with dizziness/vertigo and tingling in the left hand (page 165 of 642). Mr Wilson was known to have alcohol-related liver disease; he had been admitted 18 months earlier with epigastric pain, vomiting and pitting oedema (swelling). Investigations revealed liver cirrhosis, liver failure and fluid in the abdomen (ascites). He received diuretics (water tablets) and vitamins and told to abstain from alcohol (pages 165, 253 and 465 of 642). Mr Wilson's current medication consisted of spironolactone

100mg once a day, furosemide 40mg once a day (both water tablets) thiamine 100mg once a day (a vitamin) and multivitamins (page 110 and 166 of 642). He lived in a house with his second wife and was usually independent. He smoked 40 cigarettes a day until 3 years ago and drank six double whiskies a day (page 166 of 642). Examination revealed his left arm to be painful on palpation and on movement. He was able to move his fingers, had normal sensation to light touch and pulses were present. There was dullness to percussion and crackles at the base of his left lung. Routine blood investigations, a chest x-ray (I could find no comment or report in the notes) and an ECG (electrocardiogram; with inverted T waves in leads II, III and V1 suggestive of poor blood supply to the heart) were undertaken (pages 167, 301 of 642). Mr Wilson was initially for resuscitation in the event of an unexpected cardiorespiratory arrest (page 168 of 642). It was noted that his pain relief was unsatisfactory despite the co-codamol and he was prescribed morphine 2–5mg IV every 4h p.r.n. (pages 106 and 168 of 642). His other medication now consisted of cyclizine 50mg PO/IV p.r.n. (an anti-emetic), paracetamol 1g p.r.n., codeine phosphate 30mg p.r.n., furosemide 40mg once a day, spironolactone 100mg once a day, thiamine 100mg once a day, multivitamins 1 tablet once a day, chlordiazepoxide 15mg four times a day (a benzodiazepine given as a course in a reducing dose to manage alcohol withdrawal) (pages 106, 110, 113 of 642).

Blood test results from the 23rd September 1998 revealed abnormal liver function: bilirubin 67micromol/L (normal 3–20micromol/L), alkaline

phosphatase 120IU/L (normal 30–95IU/L), aspartate aminotransferase (AST) 91IU/L (normal 12–40IU/L), gamma-glutamyl transferase (GGT) 102IU/L (normal 4–45IU/L), albumin 25g/L (normal 37–50g/L) (page 209 of 642). None of these findings were new; abnormal liver function was present 18 months earlier (page 129 of 642). Kidney function was also abnormal: urea 11mmol/L (normal 3–7.6mmol/L), creatinine 178micromol/L (normal 60–120micromol/L) (page 209 of 642). This appeared to be a new finding, not present 18 months earlier (page 195 of 642).

Mr Wilson's analgesic requirements varied over his time at Queen Alexandra Hospital. Between the 23rd and 24th of September 1998, the pain seemed severe and he received three doses of morphine (5mg, 2.5mg and 2.5mg) IV/SC, five doses of codeine 30mg and one dose of paracetamol PO (pages 25 and 106 of 642). Mr Wilson was reviewed early on the morning of 24th September 1998 because of pain in the left arm and reduced forearm sensation. He was discussed with the orthopaedic team and although the pain persisted, it appeared to improve and the left limb pulse, colour and function were monitored regularly and remained satisfactory (pages 25 and 169 of 642).

On the 25th September 1998 he commenced co-dydramol 2 tablets four times a day regularly, providing a daily dose of dihydrocodeine 80mg and paracetamol 4g; together with senna and magnesium hydroxide as laxatives (page 114 of 642). He continued on this regimen until the 30th September 1998 when he was prescribed paracetamol alone (page 114 of 642). The discontinuation of the dihydrocodeine appeared to be in

response to his drowsiness (page 171 of 642). He took the paracetamol erratically, although fairly regularly in the days leading up to his transfer to Dryad Ward on the 14th October 1998 (pages 114 and 115 of 642). Additional analgesia was intermittently required; morphine 2.5mg IM on the 3rd and 5th October 1998 (page 107 of 642) and codeine 30mg, each time as a single dose, on the 8th, 9th, 12th and 13th of October 1998 (page 107 of 642).

A full blood count dated 25th September 1998 was abnormal with a haemoglobin of 10.5g/dL (normal 13–18g/dL), white cell count of $15.1x10^9$/L (normal 4–$11x10^9$/L) due to an increase in neutrophils, platelets $133x10^9$/L (normal 150–$400x10^9$/L) and a mean corpuscular volume (MCV) of 113.4fL (normal 80–96fl) (page 239 of 642). A repeat blood test on the 25th September 1998 also revealed worsening kidney function with urea 17.8mmol/L and creatinine 246micromol/L (pages 170 and 207 of 642). This was acted upon on the 28th September 1998. It was considered due to dehydration; the water tablets furosemide and spironolactone were discontinued and he was given intravenous fluids (page 170 of 642).

On the 27th September 1998, Mr Wilson's second wife returned from holiday and made it clear that she would not be able to care for him in his present condition. The staff explained the concerns about his poor nutritional intake and improving his pain relief. His wife informed the staff that Mr Wilson frequently had nothing to eat all day (page 12 of 642). The pain remained 'bad' in the arm although at night time he was

reported to appear comfortable with regular analgesia (pages 27 and 28 of 642).

On the 29th September 1998, Mr Wilson's first wife visited and expressed concern to the staff about Mr Wilson's low mood. [redacted] personal data [redacted]

Because Mr Wilson had not passed urine all day he was catheterised and had a residual volume of 600ml (page 13 of 642). It was noted that he was able to lift his left arm quite well without any pain (page 28 of 642). His resuscitation status was changed to not for resuscitation in the event of an unexpected cardiorespiratory arrest because of his liver failure, kidney failure, poor quality of life and poor prognosis (page 171 of 642).

On the 30th September 1998, Mr Wilson's kidney function had improved with urea 14.4micromol/L and creatinine 165micromol/L (page 171 and 199 of 642). He was noted to be drowsy but did not have a flap (one sign of hepatic encephalopathy; see technical issues) and his temperature was normal. The top of his left arm was oedematous and weeping in small areas (page 14 of 642). The sedative drugs chlordiazepoxide and dihydrocodeine were discontinued (pages 113 and 114 of 642). Mr Wilson had pain in the neck and his arm, had a restless night but was unable to express his needs (pages 29 and 30 of 642).

On the 1st October 1998 it was recorded that his left arm was 'painful+++ on movement' but 'not complaining of pain at rest' (page 30 of 642).

On the 2nd October 1998, Mr Wilson was noted to be very sleepy but to be awake at night. He was noted to be oedematous (swelling of the tissues due to retained fluid) and to have crackles in his chest. These are signs of excess fluid and the IV fluids were discontinued. He was referred to the psychogeriatricians as he was very withdrawn and depressed (page 172 of 642). Mr Wilson expressed that he was desperate for sleep, but was awake at night and asleep during day (page 15 of 648). It was considered that he would require long term care (page 172 of 642). His arm remained painful on movement (page 30 of 642).

Between the 4th and 6th October 1998, Mr Wilson's level of sleepiness improved but pain was still present in his left arm on movement. He was reviewed at the fracture clinic, who advised physiotherapy (pages 31, 32, 173, 174 and 333 of 642). He was not tolerating the sling and so the arm was elevated on pillows (page 16 of 642). Blood tests revealed that Mr Wilson's urea and creatinine had returned to normal (page 201 of 642). On the 4th October 1998 at 23.10h he refused to take oral analgesia and had morphine 2.5mg IM with good effect (page 31 of 642). A further dose of morphine 2.5mg IM was given at 02.00h on the 5th October 1998, as Mr Wilson refused paracetamol, stating that it didn't help (page 32 of 642). On the 6th October 1998, he was reported

to have had a comfortable afternoon but at night that the prescribed analgesia had only a small effect on his arm pain (page 33 of 642).

On the 7th October 1998 he was reported to be brighter, more talkative and eating and drinking more. He walked a short distance with help and expressed the wish to return home (pages 17 and 174 of 642). At night he was reported as uncomplaining (page 33 of 642).

On the 8th October he was reviewed by Dr Lusznat, consultant in old age psychiatry. She noted that Mr Wilson had been sleepy, withdrawn, low in mood with disturbed nights but that he was now eating and drinking well and his mood had improved. Examination found him still to be low in mood, admitting that there was no point in living. He was fully orientated in place, partially orientated in time and had mildly impaired short-term memory, scoring 24/30 on the mini-mental state examination. She noted him to be obese with his left arm in a sling, his left hand grossly swollen and bruised and marked oedema of both legs. Dr Lusznat considered that Mr Wilson may have developed an early dementia which could be alcohol related, or alternatively, an early Alzheimer's or vascular dementia. In addition she considered him depressed and commenced him on the sedative antidepressant trazadone 50mg at night (pages 114 and 118 of 642). Because of the gross oedema, diuretics were recommenced by Dr Ravi, this time as spironolactone 50mg twice a day and bendrofluazide 2.5mg once a day (pages 114 and 176 of 642). At night, the nurses requested stronger analgesia for Mr Wilson and codeine phosphate 30mg p.r.n. was prescribed (pages 35 and 107 of 642).

On the 9th October 1998, his urinary catheter was removed (page 35 of 642). On the 10th October 1998, it was noted that Mr Wilson was a bit brighter and that the swelling in his left arm seemed better (page 177 of 642).

The pain remained variable; on the 11th October 1998 co-dydramol 2 tablets p.r.n. were also prescribed but never administered (pages 35 and 107 of 642). His pain was reported as 'quite bad' but his night was comfortable with regular analgesia (page 35 and 36 of 642). Despite the improvement in his level of alertness and nutritional intake, Mr Wilson's Barthel score (activity level) was still reduced (13 on the 23rd September, 7 on the 11th October 1998)(page 69 of 642).

On the 12th October, it was noted that Mr Wilson 'remains in a lot of pain when being cared for' and had a restless night (page 36 of 642).

On the 13th October 1998, it was noted that Mr Wilson was still very oedematous and his weight was increasing (suggesting increasing fluid retention) and the diuretic furosemide 80mg was commenced (pages 36, 114, 115, 177 and 178 of 642). His weight had progressively increased during his admission from 103.9kg on 27th September 1998 to 114.3kg on 14th October (pages 61, 63, 65 of 642). As he still needed both nursing and medical care it was noted that a 'short spell in long term NHS bed would be appropriate'. Mr Wilson's limbs were considered at high risk of breakdown (his right foot was about to breakdown) due to the oedema caused by heart failure and low protein. He was also considered at high risk of self neglect and injury if he

started to take alcohol again (page 21 of 642). There were no complaints of any pain (page 36 and 37 of 642).

On the 14th October 1998, an entry at 05.00h records that Mr Wilson had had a peaceful night, slept well with no complaints of pain. He was later moved to Dryad Ward at Gosport War Memorial Hospital (pages 37, 177 and 178 of 642). The transfer letter indicated that Mr Wilson was being transferred for continuing nursing care until his arm was healed; that he still had a lot of pain in his arm and difficulty moving it and that his oedematous legs due to heart failure and low protein were at high risk of breaking down. His regular medication was listed but not his codeine 15–30mg p.r.n. (page 81 of 642).

Events at Gosport War Memorial Hospital, Dryad Ward, 14th–18th October 1998

14th October 1998

An entry in the medical notes records that Mr Wilson was transferred to Dryad Ward Continuing Care, his fractured left humerus on the 27th August 1998 (an incorrect date, already acknowledged by Dr Barton) and his past medical history of alcohol problems, recurrent oedema and CCF (congestive cardiac failure). It was noted that he needed help with ADL (activites of daily living) required hoisting, was continent and had a Barthel score of 7. The plan was for 'gentle mobilisation' (page 179 of 642).

He was prescribed furosemide 80mg once a day, spironolactone 50mg twice a day, bendrofluazide 2.5mg once a day, trazadone 50mg once a day, thiamine 100mg once a day, multivitamins 1 tablet once a day, magnesium

hydroxide 10ml twice a day and senna 2 tablets once a day (pages 260 and 261 of 642). The regular paracetamol was discontinued and prescribed p.r.n. instead, although he never received any (page 258 of 642). On the daily review section of the drug chart, 'Regular prescription' was crossed out and replaced with 'p.r.n.' and morphine solution 5–10mg prescribed every 4h p.r.n. (page 262 of 642). He never received a 5mg dose; but 10mg at 14.45h and 23.45h on the 14th October 1998. Although undated, Dr Barton anticipates that on the 14th October 1998, she also prescribed diamorphine 20–200mg SC/24h, hyoscine hydrobromide 200–800microgram SC/24h and midazolam 20–80mg SC/24h (page 262 of 642). The nursing summary for the 14th October 1998 notes 'seen by Dr Barton. Oramorph 10mg in 5ml given (page 265 of 642). His Barthel score was 4 (page 273 of 642). The nursing care plan for 'requires assistance to settle at night' noted that morphine 10mg was given for pain relief (page 278 of 642).

15th October 1998

There was no entry in the medical notes, but Mr Wilson was prescribed morphine 10mg every 4h and 20mg at night. In total he was given 50mg of morphine over the next 24h (page 261 of 642). The nursing summary notes recorded that this was for pain in his left arm and that Mr Wilson's wife was seen by Sister Hamblin who explained that his 'condition is poor' (page 265 of 642). The nursing care plan for 'requires assistance to settle at night' reported that Mr Wilson settled and slept well with morphine 20mg given at 12 midnight but that his condition had deteriorated overnight 'very chesty

and difficulty in swallowing medications, incontinent of urine++'. Morphine 10mg was given at 06.00h (page 278 of 642).

16th October 1998

An entry in the medical notes was made by Dr Knapman, one of Dr Barton's partners. He notes 'declined overnight with shortness of breath. On examination bubbly, weak pulse, unresponsive to spoken orders. Oedema++ in arms and legs. Diagnosis ?silent MI (myocardial infarction; heart attack, silent indicating without pain), ?decreased liver function. Dr Knapman prescribed an additional dose of furosemide 80mg PO (pages 179 and 261 of 642). Mr Wilson received this extra dose once only on the 16th October 1998.

The nursing summary notes record 'seen by Dr Knapman a.m. as deteriorated overnight. Increase furosemide to 80mg daily (although he was already on furosemide 80mg daily; page 260 of 642), for all nursing care. Wife informed of visit this morning' (page 265 of 642). A later entry notes 'p.m. patient very bubbly chest this p.m., syringe driver commenced 20mg diamorphine, 400micrograms hyoscine hydrobromide. Explained to family reason for driver. Wife informed of patient's continued deterioration, has been to visit' (page 265 of 642). The syringe driver was commenced at 16.10h (page 262 of 642).

The nursing care plan for 'requires assistance to settle at night' noted 'has been on syringe driver since 16.30h. Diamorphine 20mg and hyoscine 400microgram. A little bubbly at approximately 22.30h when repositioned/pad changed. More secretions – pharyngeal - during the night

but Robert hasn't been distressed. Appears comfortable' (page 278 of 642).

17th October 1998

An entry in the medical notes was made (according to Dr Barton's statement) by Dr Peters, one of her partners 'comfortable but rapid deterioration. Nursing staff to verify death if necessary' (page 179 0f 642).

The nursing summary notes for the morning recorded '05.15h hyoscine increased to 600microgram as oropharyngeal secretions increasing overnight. Diamorphine 20mg' (unchanged) (page 265 of 642). A later entry noted 'p.m. Slow deterioration in already poor condition. Requiring suction very regularly – copious amounts suctioned. Syringe driver renewed at 15.50h with diamorphine 40mg, midazolam 20mg, hyoscine 800micrograms. Mrs Wilson visited again this evening and is aware that his condition is poorly' (pages 265 and 266 of 642). The drug chart confirms the times and changes in the medication (page 262 of 642).

The nursing care plan 'requires assistance to settle at night' notes '05.15h hysocine increased to 600microgram as secretions increased. During day diamorphine 40mg and hyoscine increased to 800microgram, midazolam 20mg added. Night: noisy secretions but not distressing Robert. Suction given as required during night. Appears comfortable, hot at times' (page 278 of 642).

18th October 1998

The nursing summary notes record 'further deterioration in already poor condition. Wife has remained overnight. Seen by Dr Peters who spoke to Mrs Wilson. Syringe driver renewed at 14.50h with diamorphine 60mg, midazolam 40mg and hyoscine 1200microgram. Continues to require regular suction. His children have also visited' (page 266 of 642). A later entry notes 'p.m. All care has been given. Oral suction has been required and performed. Condition continues to deteriorate' (page 266 of 642). The drug chart confirms the times and changes in the medication (page 262 of 642).

The nursing care plan for 'requires assistance to settle at night' records 'Suctioned at 22.30h for large amounts of sputum. Patient died peacefully at 23.40h (page 278 of 642). Confirmation of death is recorded in the nursing summary notes and in the medical notes by the nursing staff (pages 179 and 266 of 642).

The cause of death was given as '1a (*Disease or condition directly leading to death*) Congestive cardiac failure, 1b (*Other disease or condition, if any, leading to 1a*) Renal failure and 2 (*Other significant conditions contributing to the death but not related to the disease or condition causing it*) Liver failure. The approximate interval between onset and death was given as 2 years for each of the above.

7. TECHNICAL BACKGROUND / EXAMINATION OF THE FACTS IN ISSUE

i) Syringe drivers, diamorphine, midazolam and hyoscine hydrobromide

A syringe driver is a small portable battery-driven pump used to deliver medication subcutaneously (SC) via a syringe, over 24h. Indications for its use include swallowing difficulties or a comatose patient. In the United Kingdom, it is commonly used in patients with cancer in their terminal phase in order to continue to deliver analgesic medication. Other medication required for the control other symptoms, e.g. delirium, nausea and vomiting can also be added to the pump.

Diamorphine is a strong opioid that is ultimately converted to morphine in the body. In the United Kingdom, it is used in preference to morphine in syringe drivers as it is more soluble, allowing large doses to be given in very small volumes. It is indicated for the relief of pain, breathlessness and cough. The initial daily dose of diamorphine is usually determined by dividing the daily dose of oral morphine by 3 (BNF 35, March 1998). Others sometimes suggest dividing by 2 or 3 depending on circumstance (Wessex protocol). Hence, 60mg of morphine taken orally a day could equate to a daily dose of 20 or 30mg of diamorphine SC. It is usual to prescribe additional doses for use 'as required' in case symptoms such as pain breakthrough. The dose is usually 1/6th of the 24h dose. Hence for someone receiving 30mg of diamorphine in a syringe driver over 24h, a breakthrough dose would be 5mg. One would expect it to have a 2–4h duration of effect, but the dose is often prescribed to be given hourly as required. As the active metabolites of morphine are excreted by the kidneys, caution is required in patients with impaired kidney function.

Midazolam is a benzodiazepine, a diazepam like drug. It is commonly used in syringe drivers as a sedative in patients with terminal agitation. Sedation can be defined as the production of a restful state of mind. Drugs that sedate will have a calming effect, relieving anxiety and tension. Although drowsiness is a common effect of sedative drugs, a patient can be sedated without being drowsy. Most practitioners caring for patients with cancer in their terminal phase would generally aim to find a dose that improves the patients' symptoms rather than to render them unresponsive. In some patients however, symptoms will only be relieved with doses that make the patient unresponsive. A typical starting dose for an adult is 30mg a day. A smaller dose, particularly in the elderly, can suffice or sedate without drowsiness. The BNF (BNF 35, March 1998) recommends 20–100mg SC over 24h. The Wessex protocol suggests a range with the lowest dose of 5mg a day. The regular dose would then be titrated every 24h if the sedative effect is inadequate. This is generally in the region of a 33–50% increase in total dose, but would be guided by the severity of the patients symptoms and the need for additional 'as required' doses. These are generally equivalent to 1/6th of the regular dose, e.g. for midazolam 30mg in a syringe driver over 24h, the 'as required' dose would be 5mg given as a stat SC injection. The duration of effect is generally no more than 4h, and it may need to be given more frequently. As an active metabolite of midazolam is excreted by the kidneys, caution is required in patients with impaired kidney function.

Hyoscine hydrobromide is an antimuscarinic drug most commonly given to reduce excessive saliva or retained secretions ('death rattle'). It also has

anti-emetic, antispasmodic (smooth muscle colic) and sedative properties. Repeated administration can lead to cummulation and this can occasionally result paradoxically in an agitated delirium, highlighted in both in the BNF and the Wessex protocol (page 41). It is usually given in a dose of 600–2400microgram SC over 24h (BNF 35, March 1998) or 400–600microgram as a stat SC dose. The Wessex protocol gives a dose range of 400–1200microgram over 24h.

The titration of the dose of analgesic or sedative medication is guided by the patients symptom control needs. The number and total dose of p.r.n. doses needed over a 24h period are calculated and this guides the increase necessary in the regular dose of the drugs in the syringe driver in a way that is proportional to the patients needs. The ideal outcome is the relief of the symptoms all of the time with no need for additional p.r.n. doses. In practice, this can be difficult to achieve and the relief of the symptoms for the majority of the time along with the use of 1–2 'as required' doses over a 24h period is generally seen as acceptable.

ii) The principle of double effect

The principle of double effect states that:

'If measures taken to relieve physical or mental suffering cause the death of a patient, it is morally and legally acceptable provided the doctor's intention is to relieve the distress and not kill the patient.'

This is a universal principle without which the practice of medicine would be impossible, given that every kind of treatment has an inherent risk. Many discussions on the principle of double effect have however, involved the use

of morphine in the terminally ill. This gives a false impression that the use of morphine in this circumstance is a high risk strategy. When correctly used (i.e. in a dose *appropriate* to a patient's need) morphine does not appear to shorten life or hasten the dying process in patients with cancer. Although a greater risk is acceptable in more extreme circumstances, it is obvious that effective measures which carry less risk to life will normally be used. Thus, in an extreme situation, although it may occasionally be necessary (and acceptable) to render a patient unconscious, it remains unacceptable (and unnecessary) to cause death deliberately. As a universal principle, it is also obvious that the principle of double effect does not allow a doctor to relinquish their duty to provide care with a reasonable amount of skill and care.

iii) Hepatic (liver) encephalopathy

Hepatic encephalopathy is a life-threatening condition that arises when toxic substances, usually removed by the liver, cumulate in the blood (e.g. ammonia). It causes confusion, disorientation, abnormal neurological signs, loss of consciousness and death. It is common in patients with chronic liver disease/cirrhosis who binge drink or develop an acute infection. It can also be precipitated by, for example:

- gastrointestinal bleeding
- constipation (increases nitrogen-containing compounds)
- dehydration (cumulation of nitrogen-containing compounds, e.g. urea)
- electrolyte imbalances (e.g. low levels of potassium)

- drugs such as sedatives (e.g. opioid analgesics) or diuretics (via dehydration ± low potassium)
- reduced levels of oxygen (hypoxia).

Symptoms of hepatic encephalopathy range from minor changes in personality, energy levels and cognition to deep coma. There may be inappropriate behaviour, lack of interest in personal grooming, mood swings and poor judgment. The patient may be less alert than usual and develop new sleep patterns. Movement and speech may be slow and laboured. As the disease progresses, patients become confused, drowsy, and disoriented. The breath and urine acquires a sweet, musky odour. The hands shake, the outstretched arms flap ('liver flap') and the patient may lapse into unconsciousness. Agitation occasionally occurs. Seizures are uncommon.

Confusion, disorientation, and other signs of impaired brain function strongly suggest encephalopathy in patients known to have liver disease. Management consists, when possible, of treating reversible causes, removing or avoiding precipitating factors, improving liver function and decreasing the body's production of toxic substances. For example, non-essential medications are discontinued, antibiotics, enemas or laxatives are used to decrease the production of ammonia by bacteria in the intestine and dietary protein intake is reduced.

Encephalopathy may be reversible if the responsible factor is identified and removed or treated. Patients whose condition is the result of chronic liver disease may recover completely after the underlying cause is corrected. However, those with chronic liver failure often die in hepatic coma.

8. OPINION

Events at Queen Alexander Hospital, September 21st–October 14th 1998

Mr Wilson was a 74 year old man with alcohol-related cirrhosis and liver failure. He had fallen after drinking alcohol and fractured the greater tuberosity of his left humerus which showed 'some displacement' of the fragment. Other problems around the time of his initial admission were vomiting, unsteadiness on his feet, vertigo and intermittent confusion, also likely to be related directly or indirectly to alcohol. I note that Dr Marshall considers hepatic encephalopathy a likely explanation for some of Mr Wilson's problems (see technical issues).

Blood tests confirmed liver failure as noted previously. However, on this admission his kidney function was also abnormal, most likely related to dehydration. Urinary retention may also have contributed. Further, liver failure can also compromise the blood supply to the kidneys leaving them more prone to damage from insults such as dehydration. On receiving intravenous fluids and discontinuing his diuretics, Mr Wilson became increasingly oedematous. This can be a direct consequence of severe liver failure, which results in a low level of protein in the blood stream; this in turn allows fluid to be drawn out of the blood stream and into, for example, the subcutaneous tissues or abdomen, resulting in oedema or ascites respectively. Because the blood volume is reduced the kidneys retain more water, creating a vicious circle. Increasing oedema would also occur as a consequence of heart failure which was considered a problem for Mr Wilson. His heavy smoking would have increased his risk of heart problems (his ECG was suggestive of a reduced blood supply to the heart)

and alcohol can directly damage the heart. The chest X-ray film or report should be sought, as this may also provide evidence of heart failure. The fluids were stopped and although the diuretics were recommenced his weight (and hence fluid retention) continued to increase. By the time of his transfer, he was receiving a larger dose of diuretic than on admission and his weight had increased by about 10kg, equivalent to about 10L of (additional) retained fluid.

During his admission, Mr Wilson did improve with regard to his level of alertness. He was more talkative and eating and drinking more. There may be several reasons for this improvement; abstinence from alcohol, discontinuation of sedative drugs; correction of his dehydration and better nutritional intake. Nevertheless it was considered likely that he had an alcohol-related early dementia, a depression and he remained dependent on others for his care.

I note that the orthopaedic team considered surgical fixation of the displaced tuberosity, only to ultimately decide against this, based on Mr Wilson's wishes and clinical condition. I am not an expert in orthopaedics, nor have I seen the X-rays and thus I am unsure to what extent the 'some displacement' of the fragment could impact upon the anticipated clinical course of the fracture I describe below. If this aspect of the case is considered important, the opinion of an orthopaedic surgeon should be obtained. However, it is my general understanding that pain from this sort of a fracture can initially be severe enough to require strong opioids. Subsequently, the main approaches for pain relief would be immobilisation and weak opioids as proffered to Mr Wilson. Movement is likely to

aggravate the pain until the fracture begins to heal, a process that can take several weeks and not be fully complete for 12 weeks (although there is wide variation). Nevertheless, one would anticipate that Mr Wilson's pain would improve so that he was pain-free when the limb was at rest, followed by a progressive improvement in the movement-related ('incident') pain. Attempting to provide sufficient analgesia to manage incident pain can be difficult; the dose of opioid required to fully relieve the pain on movement can be excessive for the patient whom for the majority of the time is resting and pain free. Typically in this situation the patient becomes increasingly drowsy as the dose of opioid increases.

Thus, it was not unusual that Mr Wilson initially had severe pain and he received at least one and possibly two doses of morphine 10mg IV in the Accident and Emergency department. The dose of morphine the BNF recommends for acute pain varies with the route of administration: 10mg (15mg for heavier patients) SC or IM, and one quarter to one half of this dose if given IV (i.e. up to 7.5mg IV in heavier patients). Although Mr Wilson was heavy (about 100kg) he also had severe liver failure and it would have been prudent in my opinion to have used smaller doses, as he was subsequently prescribed (e.g. 2.5–5mg morphine IV/SC). Mr Wilson was treated with a sling and initially prescribed analgesia to be given as required; the most he received in one day was on the 24th September 1998 consisting of morphine (total of 5mg IV/SC), codeine (total 90mg) and paracetamol 1g. The oral morphine equivalent of this combination of morphine and codeine is approximately 20mg. Subsequently he was prescribed co-dydramol 8 tablets a day regularly (a total of dihydrocodeine

80mg; the oral morphine equivalent is approximately 8mg). This was discontinued after 6 days as Mr Wilson was drowsy, leaving him just on paracetamol. It is possible that the dihydrocodeine could have been aggravating his hepatic encephalopathy and he did subsequently improve. However, he had also been receiving chlordiazepoxide, a sedative benzodiazepeine, which was discontinued at the same time.

The reports regarding Mr Wilson's level of comfort did vary. This may relate to varying levels of activity causing movement-related pain or his depressed mood. His pain also appeared more bothersome at night. This is not unusual and thought partly due to there being less happening at night to distract the persons' attention away from the pain. Whatever the cause, there were times when the paracetamol alone appeared ineffective or inadequate for Mr Wilson's analgesic requirements and a small number of additional doses of morphine and codeine were administered. However, this never exceeded morphine 2.5mg IM (last dose on the 5th October 1998) or codeine 30mg in one day, an oral morphine equivalent of 3–5mg. Given this infrequent use of additional analgesia, in my opinion, the approach to Mr Wilson's analgesia was reasonable. Although the transfer letter noted 'still has a lot of pain in his arm and difficulty moving' overall his analgesic requirements had reduced over the course of his admission; over the 48h prior to his transfer his only analgesia was paracetamol 1g four times a day along with only one additional dose of codeine 30mg each day. Further, the nursing daily summary notes for the 13th October 1998 reported no complaints of pain from Mr Wilson, and the entry dated the 14th October 1998, the day of his transfer, noted that Mr Wilson had had a

peaceful night, slept well with no complaints of pain. I have no concerns regarding the care proffered to Mr Wilson at the Queen Alexander Hospital. Although Dr Marshall suggests that high dose vitamins IV and lactulose should also have been considered, I note that he also concludes that Mr Wilson's care at Queen Alexander Hospital was 'not perfect but very reasonable'.

Events at Dryad Ward, 14th October–18th October 1998.

Infrequent entries in the medical notes during Mr Wilson's stay on Dryad Ward make it difficult to closely follow his progress over the last four days of his life. There are three entries prior to the confirmation of death taking up less than one page in length. In summary and in approximate chronological order, Mr Wilson was admitted to Dryad Ward for 'gentle mobilisation'. There was a brief history but no pain assessment or examination documented in the medical notes. The transfer letter listed his regular medication, but omitted to note that he was also prescribed codeine phosphate 15–30mg p.r.n. Mr Wilson's regular medication was continued largely unchanged on Dryad Ward, but his regular paracetamol was discontinued and made p.r.n. If pain was considered such a problem for Mr Wilson, it unclear why his only regular analgesic was discontinued. He was prescribed morphine solution 5–10mg p.r.n. for pain relief. As required analgesics are sometimes written as a choice of two doses that cover a small dose range, but the effect of the smaller dose is generally evaluated first and it is unclear why this did not happen; Mr Wilson received two doses of 10mg on the day of his arrival on Dryad Ward.

Although Mr Wilson was transferred for 'gentle mobilisation' it is of concern that on the day of his transfer he was also prescribed diamorphine 20–200mg SC/24h, hyoscine hydrobromide 200–800microgram/24h and midazolam 20–80mg SC/24h. There appeared to be no immediate indication for the prescription of these drugs in these dose ranges. In particular, the dose range of diamorphine, equivalent to 40–600mg of oral morphine/24h, in my opinion, contains doses that would likely be excessive to Mr Wilson's needs.

On the day following his admission Mr Wilson was commenced on regular oral morphine 10mg every 4h and 20mg at night. The nursing summary notes recorded that this was for pain in his left arm. In total he received 50mg of morphine in this 24h period, representing a larger dose than he received in the initial 24h after his fracture. This is against the general expectation that pain from a fracture would have been improving over time and, without a clearly documented pain assessment, it is difficult to justify. He had required two p.r.n.s of morphine in the previous 24h and this generally suggests regular analgesia is required. However, as the total dose he received was 20mg/24h, in my opinion an equivalent dose, i.e. morphine 2.5mg every 4h and 5mg at night (20mg/day) would have been most prudent.

However, the impact of this dose of morphine on Mr Wilson is impossible to judge. He deteriorated rapidly in the early hours of the 16th October 1998 becoming 'very chesty, difficulty in swallowing medications and incontinent of urine'. When reviewed later that day by Dr Knapman it was noted that he had declined overnight with shortness of breath, he was 'bubbly' (retained

secretions causing noisy breathing), had a weak pulse and was not able to respond. The doctor made a clinical diagnosis of a silent (i.e. without chest pain) myocardial infarction and decreased liver function. As an additional dose of furosemide 80mg was given, this suggests that the doctor considered pulmonary oedema (fluid in the lungs) was responsible for his shortness of breath. The nature of his rapid decline and subsequent death could be in keeping with worsening heart failure precipitated by a sudden event such as a myocardial infarction. His reduced level of consciousness could have been due to hepatic encephalopathy precipitated by the morphine or by a reduced level of blood oxygen secondary to the pulmonary oedema. A respiratory rate and oxygen saturation level were not recorded in Mr Wilson and it is difficult to comment further regarding respiratory depression. Very rarely, pulmonary oedema has been reported following an opioid overdose, mainly in IV drug users; to my knowledge, there has been only one published case of possible opioid-induced pulmonary oedema in a patient with cancer following a rapid escalation in the dose of morphine given IV (200mg → 2,000mg/24h increased over 6 days for unrelieved pain). It is generally associated with the rapid administration of a dose large enough to cause sudden onset respiratory depression and hypoxia. There may also be release of the chemical histamine in the lungs. Both hypoxia and histamine cause the blood vessels in the lung to become leaky, resulting in pulmonary oedema. However, in my opinion, this is unlikely to have been a contributing factor to Mr Wilson's pulmonary oedema, partly because there was no such problem when he received the largest and most rapidly administered dose of

morphine (10mg IV) at the time of his fracture. Further, Mr Wilson had documented increasing fluid retention and heart failure which would put him at risk of a sudden deterioration leading to pulmonary oedema. This, combined with his liver failure, could easily have precipitated his terminal decline. It would have been appropriate to have excluded an abnormal heart rate or rhythm as a cause of his heart failure as this may have been reversible; there was no record of his pulse rate at the time of his deterioration and it is difficult to comment further. However, as Mr Wilson had most likely entered a terminal decline, providing symptom relief 'comfort' measures only was appropriate. If he was distressed by the breathlessness, this could still have included giving oxygen and trying to reduce the pulmonary oedema with diuretics IV, nitrates sublingual/IV and opioids IV. Mr Wilson was described as unresponsive to commands and only given an increased dose of diuretic PO rather than IV, suggesting that he may not have been that distressed. However, the fact he took the diuretic PO does suggest he was at that time conscious enough to swallow tablets.

At 16.10h on the 16th October 1998, a syringe driver was commenced containing diamorphine 20mg/24h, equivalent to oral morphine 40–60mg/24h and hyoscine hydrobromide 400microgram/24h. Although the hyoscine was most likely to be for the secretions, there is no entry relating to the syringe driver in the medical notes and the indication for the use of the diamorphine is not documented in the nursing notes. It is unclear if the nursing staff contacted Dr Barton or the duty doctor before the syringe driver was commenced as was 'the usual way' indicated by Dr Barton in her

statement. It may have been simply to replace the dose of oral morphine he had been prescribed and if the comfort of a patient is in doubt in the terminal stage, this could be seen as reasonable. However, it is subject to the same comments as the oral dose and, thus, in my opinion, diamorphine 10mg/24h CSCI would have been a more reasonable dose. It is of note that despite the pharyngeal secretions, Mr Wilson was not distressed by them and appeared comfortable. This suggests that he was unconscious.

On the 17th October 1998, because of the secretions, the hysocine was increased to 600microgram/24h at 05.15h. Despite this, copious amounts of secretions were suctioned. This further suggests that the secretions were due to pulmonary oedema and as such, if Mr Wilson was distressed by the secretions, diuretics IV/SC should have been considered because hyoscine hydrobromide would have little chance of improving the pulmonary oedema. The syringe driver was changed at 15.50h with an increased dose of hyoscine hydrobromide 800microgram/24h and diamorphine 40mg/24h, equivalent to oral morphine 80–120mg/24h and midazolam 20mg/24h added. It was reported that the secretions were noisy but not apparently distressing Mr Wilson. Thus, although diamorphine and midazolam are used to relieve the sensation of breathlessness in the terminal stage, it is unclear from the medical or nursing notes why it was necessary in Mr Wilson's case to increase the diamorphine or add the midazolam.

Mr Wilson continued to require regular suctioning and at 14.50h on the 18th October 1998, the hysocine hydrobromide was increased to 1200microgram/24h. There were no reports that Mr Wilson was intolerant of this regular suctioning, which can be an unpleasant stimulus as it entails

the insertion of a catheter into the back of the throat; this again suggests that Mr Wilson was likely to be unconsciousness and unaware. It is thus unclear from the medical or nursing notes why it was considered necessary, and by whom, to further increase the diamorphine to 60mg/24h, equivalent to oral morphine 120–180mg/24h and the midazolam to 40mg/24h. There were no reports of Mr Wilson being distressed because of the secretions or pain and as such it is unclear why his dose of diamorphine was trebled over a 48h period.

The cause of death was given as 1a. congestive cardiac failure which is in keeping with his terminal decline. 1b. was given as renal failure, present for a period of 2 years, this is inaccurate; his renal impairment at Queen Alexander Hospital resolved completely with appropriate therapy. 1c. was given as liver failure, which was an important contributing factor to his death.

Was the standard of care afforded to this patient in the days leading up to his death in keeping with the acceptable standard of the day?

The medical provided by Dr Barton and Dr Knapman to Mr Wilson following his transfer to Dryad Ward, Gosport War Memorial Hospital is suboptimal when compared to the good standard of practice and care expected of a doctor outlined by the General Medical Council (General Medical Practice, General Medical Council, July 1998, page 2–3) with particular reference to:

- good clinical care must include an adequate assessment of the patient's condition, based on the history and clinical signs and, if necessary, an appropriate examination

- in providing care you must keep clear, accurate, and contemporaneous patient records which report the relevant clinical findings, the decisions made, the information given to patients and any drugs or other treatment prescribed
- in providing care you must prescribe only the treatment, drugs, or appliances that serve patients' needs.

Specifically:

i) There was insufficient assessment and documentation of Mr Wilson's physical state and pain on his transfer to Dryad Ward on the 14th October 1998.

ii) Mr Wilson was prescribed doses of oral morphine initially p.r.n. and subsequently regularly, likely to be excessive to his needs. On the day of his transfer he was also prescribed doses of diamorphine to be given by syringe driver p.r.n. in a range that would likely be excessive to his needs.

iii) There was insufficient assessment and documentation of Mr Wilson's clinical condition when he deteriorated on the 16th October 1998.

iv) Mr Wilson subsequently received doses of diamorphine over the last 48h of his life that were likely to be excessive to his needs.

If the care is found to be suboptimal what treatment should normally have been proffered in this case?

Issue i (lack of clear documentation that an adequate assessment has taken place; lack of clear, accurate and contemporaneous patient records).

Mr Wilson's admission to Dryad Ward was accompanied by the minimum of medical notes. A medical assessment usually consists of information

obtain from the patient ± others, the existing medical records (the history), and the findings of a relevant physical examination documented in a structured fashion. Although the history can be restricted to the most salient points, it is unusual to omit relevant sections, e.g. past medical history, drug history, etc. When a new medical team takes over the day-to-day care of a patient with serious medical problems, a physical examination is warranted to inform the ongoing management of those medical problems and to also provide a base line for future comparison. This allows monitoring of changes for the better or worse. A clear assessment and documentation of medical care is also particularly useful for on-call doctors who may have to see a patient, whom they have never met, for a problem serious enough to require immediate attention.

There was no pain assessment that would help to justify why his only regular analgesic was discontinued and why morphine rather than his usual codeine was prescribed p.r.n.

Issue ii (in providing care you must prescribe only the treatment, drugs or appliances that serve patients needs).

Mr Wilson was prescribed doses of oral morphine p.r.n. and subsequently regularly that were likely to have been excessive to his needs. In general, if regular paracetamol is considered insufficient, then a weak opioid such as codeine would be considered appropriate. It is known from the Queen Alexander Hospital that Mr Wilson had recently required, at most, only one dose of codeine 30mg a day, thus maintaining its use p.r.n. rather than giving it regularly would have been most appropriate in my opinion. If it

were considered necessary to give it regularly, a reasonable dose would be codeine 30mg 4 times a day (120mg/day). Some doctors do prescribe small doses of morphine instead of a weak opioid when paracetamol is inadequate. In this case, a comparable dose would be morphine 2.5mg p.r.n. or 2.5mg every 4h (15mg/24h).

Generally, if ≥2 p.r.n. doses are consistently required in a 24h period, this suggests that regular analgesia is indicated. The total amount of p.r.n. given also guides the amount of analgesia likely to be required on a regular basis. The patient's age, kidney and liver function (as in Mr Wilson's case) may also need to be taken into account. Thus, because Mr Wilson received 10mg x 2 p.r.n. doses (20mg/24h), if it was considered necessary to give him regular analgesia, a reasonable starting dose would have been morphine 2.5mg every 4h (15mg/day). Because of his liver failure, the effect of this dose would need to have been evaluated over the next 24–48h.

The prescription of a syringe driver containing diamorphine, midazolam and hyoscine hydrobromide p.r.n. for a patient transferred for 'general mobilisation' is not usual in my experience, particularly with such a wide dose range. This is because of the inherent risk that would arise from a lack of clear prescribing instructions on why, when and by how much the dose can be altered within this range and by whom. For these reasons, prescribing a drug as a range, particularly a wide range, is generally discouraged. Doctors, based upon an assessment of the clinical condition and needs of the patient usually decide on and prescribe any change in medication. It is not usual in my experience for such decisions to be left for

nurses to make alone. If there were concerns that a patient may experience, for example, episodes of pain, anxiety or agitation, it would be much more usual, and indeed seen as good practice, to prescribe appropriate doses of morphine/diamorphine or diazepam/midazolam respectively, which could be given p.r.n. PO or SC. This allows a patient to receive what they need, when they need it, and guides the doctor in deciding if a regular dose is required, the appropriate starting dose and subsequent dose titration. The wide dose range of diamorphine 20–200mg/24h is not justified at all in the notes. As already indicated, even the lower end of this dose range may have been excessive for Mr Wilson's needs. Doses of opioids excessive to a patients needs are associated with an increase risk of drowsiness, delirium, nausea and vomiting and respiratory depression.

Issue iii (lack of clear documentation that an adequate assessment has taken place; lack of clear, accurate and contemporaneous patient records). Generally, when a patient's clinical condition changes for the worse, a thorough medical assessment should be carried out to ascertain the possible cause(s) in order to identify if they are reversible with appropriate treatment. The assessment will consist of the history, examination and appropriate investigation. With regards to the entry made by Dr Knapman on the 16th October 1998, following the rapid deterioration in Mr Wilson's condition, even basic observations have not been recorded including, for example, temperature, pulse rate/rhythm, blood pressure and auscultation of heart and breath sounds (although noisy secretions can impede useful

auscultation). These observations should have been undertaken, particularly as Dr Knapman considered that Mr Wilson had possibly experienced a serious event such as a myocardial infarction. It should be clarified on what basis Dr Knapman satisfied himself that Mr Wilson's condition was terminal rather than due to a potentially reversible complication, e.g. cardiac arrhythmia, chest infection. That said, in my opinion, given Mr Wilson's combination of severe liver failure and heart failure this rapid deterioration was most likely to be a terminal event and, as such, it was appropriate to focus his care on comfort measures.

From the description, it was likely that Mr Wilson had developed an acute worsening of his pulmonary oedema. As such, oxygen, intravenous diuretics, nitrates and opioids could all have been appropriate therapies, particularly if Mr Wilson was experiencing difficulty in breathing. The only treatment proffered to Mr Wilson was an additional dose of oral furosemide and the reason for this should be clarified. For example, IV furosemide may not have been available but IV diamorphine would have been. Did this less optimal approach to treating pulmonary oedema reflect that Mr Wilson was not particularly aware/distressed by his situation, because of being semiconscious or unconscious? When diamorphine is used for acute pulmonary oedema, it is usually given IV. It works by dilating the veins, reducing the amount of blood returning to the heart, reducing the heart's workload. Other drugs are more effective at this, e.g. nitrates, and some would use these in preference to opioids. However, I am not a cardiologist and if this aspect of the case is considered important then the opinion of a cardiologist should be sought.

Issue iv (providing treatment that serves the patients needs).

Mr Wilson received doses of diamorphine over the last 48h of his life that were likely to be excessive to his needs. It is not clear who decided to start the syringe driver later on the day of his deterioration, the drugs it should contain and the doses to use. There was no entry relating to the syringe driver in the medical notes and the indication for the use of the diamorphine is not documented in the nursing notes and this should be clarified. It may have been simply to replace the dose of regular oral morphine and, if the comfort of a patient is in doubt in their terminal stage, this could be seen as reasonable. However, given the comments in issue ii relating to an appropriate dose of oral morphine, in my opinion, diamorphine 10mg SC/24h would have been an appropriate dose.

Over the next two days the hyoscine was increased in an attempt to improve the secretions, and this would not be unusual. However, given that his situation was suggestive of pulmonary oedema, other measures would have been more likely to help, e.g. furosemide IV, IM, SC. Despite the secretions being noisy and requiring frequent suctioning, Mr Wilson did not appear distressed and this suggests that he was unconscious. Given the apparent lack of distress, it is unclear why it was considered necessary to increase the diamorphine to 40mg then 60mg SC/24h. This is equivalent to oral morphine 120–180mg/24h and, in my opinion, would have been likely to be excessive to his needs. The combination of diamorphine and midazolam are used to relieve the sensation of breathlessness in the terminal stage, but I can find no reports of Mr Wilson being distressed

because of breathlessness (or pain) and thus it is difficult to justify why his dose of diamorphine was trebled over a 48h period.

If the care is found to be suboptimal to what extent may it disclose criminally culpable actions on the part of individuals or groups?

Dr Barton and her partners had a duty to provide a good standard of practice and care that would include good palliative and terminal care. In this regard Dr Barton and Dr Knapman fell short of a good standard of clinical care as defined by the GMC (Good Medical Practice, General Medical Council, July 1998 pages 2–3) with particular reference to a lack of clear note keeping, adequate assessment of the patient (Dr Barton and Dr Knapman) and providing treatment that could be excessive to the patients needs (Dr Barton).

The dose of oral morphine prescribed for Mr Wilson's arm pain both p.r.n. and regularly were likely to be excessive for his needs. As a result, the initial dose of diamorphine 20mg/24h would also likely to be excessive to his needs. The subsequent increase in the dose of diamorphine to 60mg/24h over the following 48h was not obviously justified. Mr Wilson was likely to be unconscious; he was not reported to be distressed by pain, the secretions or his breathing and he appeared to tolerate regular suctioning. A dose of diamorphine excessive to Mr Wilson's needs would be associated with an increased risk of drowsiness, confusion, agitation, nausea and vomiting and respiratory depression.

In patients with cancer, the use of diamorphine and other sedative medications (e.g. midazolam) when appropriate for the patient's needs, do

not appear to hasten the dying process. This has not been examined in patients dying from other illnesses to my knowledge, but one would have no reason to suppose it would be any different. The key issue is whether the use and the dose of diamorphine and other sedatives were *appropriate* to the patient's needs. Although the principle of double effect could be invoked here (see technical issues), it remains that a doctor has a duty to employ effective measures that carry the least risk to life. Further, the principle of double effect does not allow a doctor to relinquish their duty to provide care with a reasonable amount of skill and care. This, in my view, would include the use of a dose opioid that was *appropriate* and not excessive for a patients needs.

Dr Barton could be seen as a doctor who, whilst failing to keep clear, accurate and contemporaneous patient records, had been attempting to allow Mr Wilson a peaceful death, albeit with what appears to be an apparent lack of sufficient knowledge, illustrated, for example, by the reliance on large dose range of diamorphine by a syringe driver rather than a fixed dose along with the provision of smaller p.r.n. doses that would allow Mr Wilson's needs to guide the dose titration. Dr Barton could also be seen as a doctor who breached the duty of care she owed to Mr Wilson by failing to provide treatment with a reasonable amount of skill and care. This was to a degree that disregarded the safety of Mr Wilson by unnecessarily exposing him to receiving excessive doses of diamorphine.

However, Mr Wilson had significant medical problems. His clinical condition was not stable in that his oedema and thus heart failure were worsening over his time in Queen Alexander Hospital, despite the

reintroduction of diuretic therapy. In this regard an acute deterioration in Mr Wilson's heart failure would not have been that unusual, whether or not precipitated by a myocardial infarction, and his death was in keeping with severe heart failure and liver failure which combined to cause a rapid irreversible physical decline. Although the dose of morphine may well have contributed to his reduced level of consciousness, either directly or by precipitating a hepatic coma, it is difficult to say with any certainty that the dose of morphine he received would have contributed more than minimally, negligibly or trivially to his death because the heart and liver failure could also have done this. Similarly, although the doses of diamorphine used were likely to have been excessive to his needs, it is difficult to say with any certainty that the dose of diamorphine he received would have contributed more than minimally, negligibly or trivially to his death, because drowsiness/unconsciousness, the one feature of excess opioid seen in this case, is also a feature of the terminal stage of heart failure and liver failure.

9. LITERATURE/REFERENCES

British National Formulary 35 (March 1998):

- Prescribing in terminal care, pages 12–15
- Prescribing for the elderly, pages 16–17

Good Medical Practice, General Medical Council July 1998, pages 2–3

Palliative Care Handbook, Guidelines on Clinical Management, Third Edition 'Wessex Protocol' Salisbury Palliative Care Services May 1995.

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: _____

3

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

Code A

Code A

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. Sensitive personal data [redacted] 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: _____

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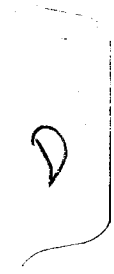

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