REPORT

regarding

Geoffrey PACKMAN (Ref No. BJC/34)

PREPARED BY: Dr Jonathan Marshall

AT THE REQUEST OF: Hampshire Constabulary

CONTENTS

1. SUMMARY OF CONCLUSIONS

Mr Packman did not experience a significant (life threatening) gastrointestinal (GI) bleed while an in-patient at Portsmouth Hospital. He developed a mild anemia of chronic disease secondary to his underlying medical problems during that part of his admission.

Mr Packman is likely to have suffered a significant GI bleed while an in-patient at GWMH. Medical assessment at that time was limited and he was managed with escalating doses of opiate analgesia before he died on 3-9-99

2. INSTRUCTIONS

I was asked to prepare this report on the instructions of Detective Sergeant Code A of Hampshire Constabulary based at Fareham Police Station, Quay Street, Fareham, Hampshire PO16 0NA.

3. ISSUES

I was asked to consider the following issues.

3.1 Can you review the papers and establish beyond all reasonable doubt whether or not the gastrointestinal bleed was treatable? If it was, at what point should it have been offered?

3.2 What treatment should have been considered in Mr Packman's case?

3.3 Should non-invasive exploration have been considered by doctors whilst Mr Packman was a patient at Haslar Hospital?

3.4 Was Mr Packman morbidly obese? If so was he unfit therefore for surgery?

4. BRIEF CURRICULUM VITAE

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

EDUCATION and QUALIFICATIONS

Medical School: University College and The Middlesex, 1982–1988

Higher Qualifications: MD: December 2001 University of London
CSST: Medicine and Gastroenterology June 2000
MRCP: 1993
BSc: Physiology with Basic Medical Sciences: Upper Second Class, University College, London 1985
MBBS: University of London 1988

PROFESSIONAL TRAINING

General Medical Training

Current:

- Dual accreditation General Internal Medicine and Gastroenterology 1st June 2000.
- Currently perform general medical duties at consultant level.
- Medical on-call shared with senior colleagues on alternate basis.
- In-patient general medical commitment of 1-2 ward-rounds per week.

Specialist Gastroenterology Training

Gastroenterology Career History:

- Student elective: Prof Cotton, Duke University, North Carolina.
- Basic endoscopic skills learnt as an SHO with Dr Barrison.
- Clinical and endoscopic skills further developed at Welwyn Garden City.
- The North Middlesex Hospital, presented a wide range of gastroenterological problems due to the ethnically diverse and mobile nature of the local population.
- The Royal Free Hospital provided specialist hepatology, liver transplantation and inflammatory bowel disease training.
- Research for MD thesis into *H.pylori* in alcoholic liver disease enabled development of a special interest in this area.
- The Whittington allowed further development of general and therapeutic endoscopic skills.
- King George Hospital, Ilford, enabled further development of therapeutic endoscopy including ERCP.
- Currently perform two out-patient clinics and 2-3 endoscopy lists per week

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

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

PUBLICATIONS

Published Papers and Abstracts.

Marshall JC, Sharp E, Barrison I.G.
"Once bitten, twice shy": Multiple abscesses in an 18 year-old female.
BMJ (1994) 309: 1694-1695.

Lagnado L, Marshall JC, Lodge L.
S-methyl-3-propanolamine (S-MDP) but neither papaverine nor noscapine is an N-methyl aspartate antagonist.
Neuroscience letters (1985) 21: 56A

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

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

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

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

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

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

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

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

Chapters

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

Poster Presentations

Alcohol Consumption and Severity of Liver Disease Influences *Helicobacter pylori* infection in cirrhotic liver disease.
Poster Presentation, Ninth Congress of the International Society for Biomedical Research on Alcoholism (ISBRA) Copenhagen, (1998)

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

Poster Presentation at the International Association for the Study of the Liver (IASL) Biennial Meeting Chicago, (1998)
Upper Gastrointestinal Pathology In relation to *Helicobacter pylori* Status in Alcohol Misusers
Poster Presentation, British Society of Gastroenterology (BSG) Glasgow (1999)
Motility and Survival of *Helicobacter pylori* in Alcoholic Beverages.
Poster Presentation, The European *H.pylori* Society Helsinki (1999)
Motility and Survival of *Helicobacter pylori* in Organic and Non-Organic Alcoholic Beverages.
Poster Presentation, British Society of Gastroenterology (BSG) Birmingham (2000)

Accepted Papers

Walker MM, Marshall JC
H.pylori and Gastric Pathology-Ask your Sommelier
Accepted Z. Gastroenterology December 2000
Marshall JC, Lample F, Morgan MY
H.pylori Infection and Hepatic Encephalopathy: The Problem of Confounding Variables
Accepted as poster International Meeting on Hepatic Encephalopathy Strasbourg November 2001

Papers Submitted or in Preparation

Marshall JC, Lample F, Madden M, Gordan H.M, Morgan M.Y.
Seroprevalence of *H.pylori* in liver disease: Influence of liver disease and alcohol consumption
In preparation for Gastroenterology
Marshall JC, Morgan MY, Walker MM
Chemical Gastritis is Not Influenced by Alcohol Consumption
In preparation for J. of Clinical Pathology

5. DOCUMENTATION

This Report is based on the following documents:

[1] Full paper set of medical records of Geoffrey Packman

6. OPINION

Question 1

Can you review the papers and establish beyond all reasonable doubt whether or not the gastrointestinal bleed was treatable? If it was, at what point should it have been offered?

Opinion: Mr Packman did not experience a *significant* (life threatening) gastrointestinal (GI) bleed while an in-patient at Portsmouth hospital between 6-8-99 and transfer to GWMH on, or around 23-8-99. There would therefore be no basis to investigate him during this part of his admission with any invasive or non-invasive procedures. His medical state was stable and there were no medical reasons to delay transfer to a 'step-down' care facility from an acute hospital.

Mr Packman was likely to have experienced a *significant* GI bleed approximately 3 days after transfer to GWMH. He was assessed as being unwell and was managed with escalating doses of opiate analgesia until he died on 3-9-99.

Basis for opinion: Mr Packman was admitted because his GP and district nurse were 'unable to cope at home' despite '3x visits/day' (by district nurse) [p40]. His main problems, recorded throughout his stay, were obesity [p40], leg oedema, cellulitis and poor mobility [p45] At the time of admission his haemoglobin was 15.7 and platelets 237 [p43]. NB: This clinical record page is un-labelled but a lab print out confirming this result is on p213. He was treated with intravenous flucloxacillin and benzyl penicillin for groin and leg cellulitis [p46]. Overall he 'doesn't look ill' and was 'mainly a nursing problem' [p47]

On 13-8-99 there is a comment about 'black stool overnight' [p52]. But, clinical examination at that time showed a soft abdomen, normal bowel sounds and normal brown stool presumably on rectal examination. Although a differential of bleeding or antibiotic related diarrhoea was proposed [p52], the presence of brown (normal) stool on examination is against significant upper or lower GI bleeding.

On 20-8-99 [p53] 'no further black motion' was recorded. No symptoms of peptic ulceration were elicited on questioning ('no nausea, no epigastric pain'). The blood pressure was stable at 140/80. Further proof to support an *absence* of significant GI bleeding is provided by the stable haemoglobin (Hb) written as 12.9. A laboratory report dated the day before (19-8-99) [p215] confirms this result.

Mr Packman's haemoglobin was 15.7 on admission (6-8-99) and as stated above was 12.9 on 19-8-99. This is a decline of 2-3 units. However, this is likely to be a trend towards the 'anemia of chronic disease' rather than a significant bleeding related fall in haemoglobin because:

- The lab report dated 19-8-99 with the Hb of 12.9 also confirms a normal platelet count of 366 [p215]. This *normal* platelet count is *against* significant bleeding as the platelet count *may* rise as a response to bleeding, especially if this has been occurring over a few days.
- A normal urea at 5.4 [p53] is also *against* a bleed as this typically *rises* in the presence of significant upper (stomach or duodenum) GI bleeding.
- Mr Packmans ESR was typically raised-a marker of inflammation-due to his cellulitis/leg ulceration (ESR 31 on 7-8-99 [p213] and ESR 68 on 19-8-99 [p215]). Chronic infection is associated with raised inflammatory markers (ESR) and 'anemia of chronic disease'- as in this case at this time.
- On the same dates [p213, p215] the MCV was 87.8 on both occasions. This is a *normal* 'mid range' result; A *normal* MCV helps distinguish developing borderline anemia of chronic disease (secondary to infection/cellulitis-as in this case) from iron deficiency anemia typically due to bleeding, albeit slowly, where the MCV is below normal ('microcytic anemia' MCV <80)

On 23-8-99 Mr Packman was transferred to GWMH where his problems were listed as obesity, arthritis, immobility and pressure sores. His mental state was 'very good' and he had 'no pain' [p54]. His lack of significant pain is also supported by an undated assessment on p243 ticking the 'No' box in relation to pain. While his pressure sores could be expected to give him a degree of discomfort, during the admission period at the previous hospital from7/8/99 to 23/8/99 the only analgesia he received was paracetamol 1g 6 hourly [p177 and 179]

On 25-8-99 Nursing staff report Mr Packman 'passing fresh blood PR'. A verbal message from Dr Reasley was received to stop Clexane at that time [p62]. Use of Clexane (low molecular weight heparin) was reasonable in view of Mr Packman's immobility to prevent deep vein thrombosis and pulmonary embolism. With signs of bleeding stopping heparin would be initial management. Mr Packman also complained of vomiting and was given metaclopramide 'with good effect' [p62].

Fresh blood PR is *usually* a sign of *lower* bowel GI bleeding. The commonest cause is haemorrhoids. Under normal circumstances a *non-urgent* sigmoidoscopy examination would be

desirable to confirm this and exclude bowel cancer. This could entirely reasonably be performed as an out-patient following hospital discharge. However, in brisk significant *upper* GI bleeding there is no time for the blood to be digested from stomach to rectum and produce the characteristic black and offensive smelling melena. It is therefore observed as 'passing fresh (bright red blood) PR'. This latter possibility is most likely to have occurred in Mr Packman's case because:

- He vomited [p62]-usually associated with upper GI bleeding.
- Was 'unwell' at lunchtime [p62] and Dr Barton was called. Haemorrhoidal bleeding *rarely* makes the patient unwell but significant upper GI bleeding *invariably* does.
- Experienced a further deterioration in the afternoon complaining of 'indigestion'[p62]. A symptom suggestive of upper GI pathology.
- Clearly was more unwell and so Mrs packman was called in [p62]
- A lab report dated the same day as Mr Packman became unwell (26/8/99) showed an Hb of 7.7 [p205] Yet a result from 2 days earlier (24/8/99) showed an Hb of 12.0 [p207] He had therefore lost at least 4 units of blood in that time. Both lab reports are monogrammed by NAB. There is however no documentation in terms of action taken.
- During these 2 dates the platelet count had *fallen* from 309 to 257. The *fall* in platelet count observed as evidence of bleeding superficially *contradicts* previous comments about bleeding being associated with a *rise* in platelet count. However, while in *slow* bleeding there is an opportunity for the bone marrow to try and correct the loss of platelets needed for coagulation by producing more and tending to cause a rise above normal levels, in *uncontrollable* haemorrhage there is no time for the bone marrow to respond. The net result is that platelets are consumed by the body's attempts to arrest haemorrhage faster than they can be produced by the bone marrow and the platelet count falls as a consequence. This may have occurred in Mr Packman's case.

At around this time a verbal order was received to give 10mg diamorphine from Dr Barton [p62]

On the same date (26-8-05) an assessment in the medical notes by 'NAB' states Mr Packman 'clammy and unwell'. A differential of MI (myocardial infarct) or GI bleed was put forward [p55]. It was stated that he was 'not well enough to transfer' and so diamorphine was commenced [p55]. No attempt is apparently made to ascertain why Mr Packman had become so acutely unwell. There are no clinical observations either in terms of direct questioning of the patient or of examination findings being recorded. 'Simple' treatment for an MI would be aspirin by mouth. Diamorphine would be appropriate if the patient was experiencing severe chest pain, and is standard practice, but typically as a single dose. There is no record of Mr Packman complaining of chest pain at this time and we know that in general terms he did not have severe, opiate requiring, pain (see above). No ecg was performed to look at the possibility of an MI further. Poor copy quality ecgs are in the record on p183,185,186,187 and 188; These appear normal and are undated. The top right corner marks them as 'AandE.' It is likely therefore that they were performed in AandE at admission rather than at this time.

The alternative diagnosis considered was 'GI bleed' [p55]. On the evidence available this appears more likely than MI. It was also considered more likely by the assessing doctor as the clinical details stated on the laboratory request form of 26/8/99 were 'bleeding pr.' This report was monogrammed by NAB [p205].

No documentation in terms of attempting to examine for signs of bleeding or to offer any form of resuscitation is available. '*Resuscitation*' means supporting the patient with intravenous fluids, oxygen and other measures to stabilize a clinical situation. On none of the drug charts reviewed are intravenous 'fluids' that might be used in resuscitation prescribed. 'DNR' or '*Do not resuscitate*' orders refer specifically to not commencing cardiopulmonary resuscitation if the heart stops. Mr

packman was in this 'DNR' category reasonably (high chance of technical futility) [p46] but not in a group in whom no resuscitation is attempted if they simply becomes *unwell.*

Question 2 and 3

What treatment should have been considered in Mr Packman's case? Should non-invasive exploration have been considered by doctors whilst Mr Packman was a patient at Haslar Hospital?

Opinion : Transfer for endoscopic therapy should have been considered in Mr Packman's case when the possibility of a GI bleed was first seriously considered when he deteriorated (26-8-99,[p55]). Endoscopy can only occur after resuscitative measures have been taken such as intravenous fluids, oxygen etc. Endoscopic therapy allows accurate diagnosis of the site and cause of bleeding. It also allows further procedures to try and stop the bleeding and is 'bread and butter' emergency gastroenterology available in any endoscopic unit. In the majority of patients the procedure can be performed on an 'early elective basis (ideally the morning after admission [or event])' This British Society of Gastroenterology (BSG) guideline is followed in our unit and will be followed closely by other UK centres as it is from the National Body [see references]

The critical determinant would be how fit Mr Packman was after resuscitative measures for the ambulance transfer to endoscopy.

Question 4

Was Mr Packman morbidly obese? If so was he unfit therefore for surgery?

Mr Packman was obese and it is stated throughout his record. The definition of 'morbidly obese' depends on knowing height and weight to calculate Body Mass Index (BMI). This information is not however available in this record. The balance of evidence is that he was obese and likely to meet the BMI definition of 'morbidly obese' if calculated.

Mr Packman would represent a high risk for surgery. It would be difficult to justify the potential mortality of *elective* surgery in a morbidly obese patient. However each situation is judged on its merits. A failure of endoscopic therapy to stop bleeding is an indication for emergency surgery. In these situations it has to be put to the patient and family that death during or soon after surgery is a high probability but it is essential to proceed with this high-risk option as the only possible way to save life. Rarely, limits are 'pre-set' if the patient is seriously unwell such as 'for endoscopic therapy only' or 'limit to 10 unit transfusion.' These are however technical discussions between endoscopist, surgeon and anaesthetist.

9. LITERATURE/REFERENCES

British Society of Gastroenterology (BSG) Endoscopy Committee: Management of non-variceal upper gastrointestinal haemorrhage: guidelines Published in Gut October 2002 supplement no iv vol 51

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: Code A Date: 26/4/05