References

1. Gillman, M.A. & Lichtigfeld, F.J. Analgesic nitrous oxide for alcohol withdrawal: a critical review after 10 years' use. *Postgrad Med J* 1990, **66**: 543–546.
2. Gillman, M.A. & Lichtigfeld, F.J. The drug management of severe alcohol withdrawal syndrome. *Postgrad Med J* 1990, **66**: 1005–1009.

Resident recognition and treatment of hypercholesterolaemic patients

Sir,

Effective treatment of hypercholesterolaemia reduces the risk of coronary heart disease (CHD). Even after the National Institutes of Health (NIH) Consensus Conference of 1985, studies reported that hypercholesterolaemia was recognized in only 47% to 66% of in-patients.^{1–4} In 1988, a nationwide physician education program was initiated by the release of the National Cholesterol Education Program Committee (NCEP) guidelines.⁵ One study suggests that family practitioners' recognition of hypercholesterolaemia has increased since then.⁶ However, the effect of the guidelines on resident physician recognition and treatment of hypercholesterolaemia is unknown. This study was performed to assess the frequency of recognition and treatment of hypercholesterolaemia by resident physicians in the in-patient setting after the publication of the NCEP guidelines.

Records of all admissions to the medical service at the Medical College of Georgia (MCG) from August 1988 to February 1989 were screened. Patients whose cholesterol was not measured, who died during the admission, or whose chart was unavailable for review were excluded. Other exclusion criteria were admission to the intensive care unit or from the emergency room, dialysis, and diagnosis of malignancy or HIV disease. Charts of the 320 patients not excluded were reviewed. Laboratory printouts were examined to determine if serum cholesterol was greater than 240 mg/dl (6.24 mmol/l). Twenty-five per cent (50) of 203 general medicine patients and 40% (47) cardiac catheterization patients had an elevated serum cholesterol. These 97 patients comprised the study group. Their charts were reviewed to determine if elevated cholesterol was recognized and if a diagnostic or therapeutic action was undertaken.

Patients were then classified as general medicine or cardiac catheterization admissions. The age, sex, serum cholesterol level, and prior history of hyperlipidaemia or coronary heart disease were recorded. The number of risk factors, including male sex, diabetes mellitus, hypertension, family history of CHD, tobacco use, and obesity was tallied. Chi-square analysis between the groups was computed for frequency of recognition, diagnostic and therapeutic actions, sex and history of hyperlipidaemia. Student's *t*-tests were computed between the groups for mean differences in age, cholesterol level, and number of risk factors.

Sixty per cent of the hypercholesterolaemic patients were recognized and 57% underwent a diagnostic or therapeutic action. Comparisons between general medicine and cardiac catheterization patients for mean age,

cholesterol level, male sex, and history of hyperlipidaemia or CHD were nonsignificant. General medicine patients had significantly fewer risk factors than cardiac catheterization patients (2.2 vs 2.7, $P < 0.05$). Hypercholesterolaemia was recognized in 38% of patients admitted to general medicine services and 83% of those admitted for cardiac catheterization ($P < 0.01$). No action was undertaken in 66% of general medicine and 19% of cardiac catheterization patients ($P < 0.01$). Cholesterol lowering diet was ordered significantly more often for cardiac catheterization patients.

We conclude that many residents often do not recognize or treat hypercholesterolaemia in the in-patient setting at the Medical College of Georgia despite publication and dissemination of the NCEP guidelines. Hypercholesterolaemia is recognized and treated significantly more often if the patient is admitted for cardiac catheterization than to a general medicine service.

Ruth-Marie E. Fincher,¹

Laura Q. Rogers,⁴

Lori M. Herring,²

Lloyd A. Lewis,³

Leonard D. Nance, III,³

¹Department of Medicine,

²Department of Surgery,

³The Curriculum Office,

Medical College of Georgia,

Augusta, Georgia,

⁴Department of Medicine,

University of Tennessee

School of Medicine, Memphis

Tennessee, USA

References

1. Consensus Conference: Lowering blood cholesterol to prevent heart disease. *JAMA* 1985, **253**: 2080–2086.
2. Madlon-Kay, D.J. Family physician recognition and treatment of severe hypercholesterolemia. *J Fam Pract* 1987, **24**: 54–56.
3. Bell, M.M. & Dippe, S.E. Recognition and treatment of hypercholesterolemia in a family practice center. *J Fam Pract* 1988, **26**: 507–513.
4. Landzberg, J.S. & Heim, C.R. Physician recognition and treatment of hypercholesterolemia. *Arch Intern Med* 1989, **149**: 933–935.
5. The Expert Panel: Report of the National Cholesterol Education Program Expert Panel on detection, evaluation and treatment of high blood cholesterol in adults. *Arch Intern Med* 1988, **148**: 36–61.
6. Madlon-Kay, D.J. Improvement in family physician recognition and treatment of hypercholesterolemia. *Arch Intern Med* 1989, **149**: 1754–1755.

Spontaneous hepatic rupture due to metastatic malignant melanoma

Sir,

The liver is a common site for the growth of metastatic deposits of malignant melanoma. The following case report demonstrates a rare complication of this condition.

A 42 year old woman presented with a 5-hour history of acute severe generalized abdominal pain, with no associated symptoms. During the preceding 4 days she had complained of dull intermittent right upper quadrant pain. On examination she was pale, clammy and hypotensive, with generalized abdominal tenderness, rebound tenderness and guarding. Resuscitation was instituted with intravenous fluids, blood sent for full blood count, urea and electrolytes, blood sugar, amylase and cross-matching, urinary catheterization performed, and radiographs taken of the chest and abdomen. Haemoglobin estimation was 8.6 g/dl, and a provisional diagnosis of spontaneous intraperitoneal haemorrhage was made. The patient was immediately transferred to the operating theatre where a long midline laparotomy was performed. The peritoneal cavity was full of fresh blood and clot, confirming the suspected diagnosis. The haemorrhaging lesion was a suspicious-looking 4 × 3 cm nodule on the dome of the right lobe of the liver, which had ruptured and stripped the capsule off a large area. Numerous such lesions were seen in both lobes of the liver; they looked like tumour nodules, but there was no evidence of a primary intra-abdominal neoplasm.

After unsuccessful attempts to control the haemorrhage by ligation, deep hepatic sutures and hepatic arterial clamping, the liver was packed off with large swabs, the abdomen closed, and arrangements made for subsequent transfer of the patient to a nearby centre with a specialist hepatic surgeon. However, the patient became hypotensive despite rapid infusion of large volumes of intravenous fluids and blood. A second laparotomy revealed gross haemoperitoneum, and both hepatic lobes were literally disintegrating, precluding any possibility of resection.

At post-mortem, in addition to the lesions described in the liver, two similar tumour nodules 1 cm in diameter were identified in the apex of the left lung. The absence of an obvious primary tumour was confirmed. Histological examination showed the nodules to be deposits of melanocarcinoma. On closer questioning of relatives, it was disclosed that the patient had undergone excision of a malignant melanoma, 0.9 mm thick, from the skin of the back, 5 years previously.

In the absence of a known hepatic lesion the diagnosis of spontaneous rupture may be difficult. A thorough history may provide important clues. The combination of peritoneal irritation and hypovolaemic shock, with a short duration of symptoms, may suggest intraperitoneal haemorrhage. Successful surgical management has been claimed for resection of the affected lobe,¹⁻³ hepatic artery ligation,⁴ or direct injection of ethanol into the bleeding lesion.⁵ However, despite early diagnosis and prompt surgery, the mortality of this condition is considerable.

M. Welch,
Trafford General Hospital,
Moorside Road,
Davyhulme,
Manchester M31 3SL, UK.

Correspondence address:
Department of Vascular Surgery,
Manchester Royal Infirmary,
Oxford Road,
Manchester M13 9WL, UK.

References

1. Hermann, R.E. & David, T.E. Spontaneous rupture of the liver caused by hepatomas. *Surgery* 1973, **74**: 715–719.
2. Mokka, R., Seppala, A., Huttunen, R., Kairaluoma, M., Sutinen, S. & Larmi, T.K.I. Spontaneous rupture of liver tumours. *Br J Surg* 1976, **63**: 715–717.
3. Nagasue, N. & Inokuchi, K. Spontaneous and traumatic rupture of hepatoma. *Br J Surg* 1979, **66**: 248–250.
4. Nilsson, L.A.V., Rudenstam, C.M. & Zettergren, L. Vascularisation of liver tumours and the effect of hepatic artery ligation. *Bibl Anat* 1967, **9**: 425–431.
5. Chung, S.C.S., Lee, T.W., Kwok, S.P.Y. & Li, A.K.C. Injection of alcohol to control bleeding from ruptured hepatomas. *Br Med J* 1990, **301**: 421.

Retroperitoneal haemorrhage from a renal carcinoma in association with streptokinase therapy

Sir,

We wish to report an unusual complication of treatment with intravenous streptokinase for suspected myocardial infarction, which we believe has not previously been described.

A 75 year old woman was admitted to another hospital following a collapse in church. On admission, she felt well and no abnormality was found on clinical examination. She had previously undergone a right mastectomy with adjuvant radiotherapy for breast carcinoma 10 years before, and was receiving treatment with captopril and hydrochlorothiazide and amiloride for hypertension. The electrocardiogram was interpreted as showing an acute myocardial infarction and treatment with 1.5 million IU of streptokinase intravenously was initiated. This was not followed by intravenous heparin. Six hours later, left upper abdominal pain developed and the patient became hypotensive to the extent that inotropic support was commenced. An aortic dissection was suspected and she was transferred to this unit.

On arrival the patient was clinically hypovolaemic with rebound tenderness in the left upper abdomen. The haemoglobin had dropped to 7.5 g/dl from 14.7 g/dl on admission to the referring hospital. Abdominal ultrasound revealed a large probable subcapsular splenic haematoma with an abnormal echogenic area within the left kidney. In the face of continuing haemodynamic deterioration despite aggressive intravenous resuscitation, and a normal clotting screen, a laparotomy was performed. Intraperitoneal blood was found associated with a large retroperitoneal haematoma extending into the splenic hilum and apparently arising from the apex of the left kidney. Further exploration revealed a 2 cm tumour arising from the upper pole of the left kidney, and subsequent histology from the nephrectomy specimen confirmed a renal adenocarcinoma. The patient made an uneventful recovery. The cause of this patient's collapse is unclear; her electrocardiogram showed a right bundle branch block pattern which, at her initial presentation, had been misinterpreted as changes of myocardial infarction.

Spontaneous retroperitoneal haemorrhage from renal causes is rare, but well recognized.¹ Series with renal adenocarcinomas as the cause of such haemorrhage vary between incidences of 1 in 309 cases, and 1 in 166.² Intravenous streptokinase when used in suspected myo-