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Editorial

Laparoscopic cholecystectomy

G W Johnston

page 1

Laparoscopic cholecystectomy: experience in a district general hospital

S J Kirk, S B Kelly, S A A F Aly, V K Sharma, P G Bateson, K J S Panesar

page 3

Initial experience of laparoscopic cholecystectomy in a district general hospital

L D McKie, I Samuel, J W R Peyton, R Campbell, M Lutton, D Strehorn, H McNeill

page 8

Deliberate self-poisoning presenting at Craigavon Area Hospital: 1976 and 1986

C Kelly, R Galloway

page 12

The changing pattern of cervical cancer in Northern Ireland 1965-1989

J H Robertson, Bertha Woodend

page 19

Renal transplantation in Northern Ireland 1968-1990

D Middleton, C Cullen

page 24

Educational supervision of pre-registration house officers

C C Doherty, Gillian Stott, J R McCluggage, R G Shanks

page 29

Junior medical posts in the NHSSB: what the doctors think

D R Gorman, J D Watson, P Ramsay-Baggs

page 35

Acoustic neurinoma surgery in Belfast 1986-1989

J R Cullen, D P Byrnes, A G Kerr, A P Walby

page 39

Surgical palliation of proximal malignant biliary obstruction

R H Wilson, G W Johnston, R J Moorehead

page 45

Shrinkage of uterine fibroids by preoperative LHRH analogue injection

H R McClelland, A J Quinn

page 51

Communication of discharge information for elderly patients in hospital

P Curran, D H Gilmore, T R O Beringer

page 56

Commentary:

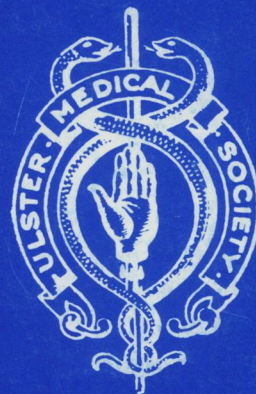
A personal view of the hospital service

J Maguire

page 59

[continued on back cover]

THE ULSTER MEDICAL JOURNAL



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HISTORICAL REVIEWS

"Who fears to speak of ninety-eight"?

Hume Logan

page 63

Cinderella had a champion

J M G Harley

page 75

The treatment of myxoedema with raw sheep thyroid gland and its introduction into practice in County Londonderry in 1892

Mary S T Logan, J S Logan

page 86

The Royal Medical Benevolent Fund Society of Ireland

W A Eakins

page 94

CASE REPORTS

Central pontine myelinolysis without hyponatraemia

A G Droogan, M Mirakhur, I V Allen, J Kirk, D P Nicholls

page 98

Prenatal diagnosis of cystic adenomatoid malformation of the lung in a twin pregnancy

Ann Harper

page 102

An aluminium foreign body in the oesophagus

G Hewitt

page 106

The air-fluidised bed in the management of chronic varicose leg ulceration

C Berry, H Taggart

page 108

Breast lymphoma: fine needle aspiration biopsy

K H McCune, M Varma, R A J Spence

page 110

Eosinophilic fasciitis presenting as a psoriatic arthropathy

G D Wright, Claire Thornton, D A J Keegan, A L Bell

page 112

Brain abscess ten years after penetrating glass injury to the skull

S B Gordon, B S Sharma, C S McKinstry, T F Fannin

page 116

Pseudogout, chondrocalcinosis and the early recognition of haemochromatosis

J S Elborn, J Kelly, S D Roberts

page 119

Book reviews

page 124

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Editorial

Laparoscopic cholecystectomy

Perhaps the most exciting advance in biliary tract surgery in the last decade has been the development of laparoscopic cholecystectomy. Keyhole surgery, often labelled the “curse of surgery” is now the “challenge of surgery”. The brief flirtation with non-operative management of gallstones is over except for a few very selected patients; only cholecystectomy cures the disease. Currently about fifty thousand cholecystectomies are carried out annually in the British Isles. Of course, laparoscopy is not a new technique. Professor Harold Rogers was a pioneer in this field in Belfast more than thirty years ago.¹ Then peritoneoscopy, as it was called, was used in diagnosis and no procedure greater than guided liver biopsy or tumour biopsy was carried out. The gynaecologists widened the technique to include therapeutic procedures, most notably sterilisation. They favoured the term laparoscopy rather than peritoneoscopy. Lapara comes from the Greek language and means flank or loin and so laparoscopy or laparotomy are really incorrect terms. Celioscopy, which means to view the abdominal cavity, would be more the most correct terminology.

In any case, in this age of minimally invasive surgery, peritoneoscopic, celioscopic or laparoscopic cholecystectomy is here to stay. Since its introduction by the French surgeons in 1988, and the subsequent media publicity, there has been a patient-driven demand for the “laser operation”. The “providers” have been goaded into action, sometimes without adequate training in the technique. This has resulted in an incidence of serious bile duct injuries in the region of one per hundred operations compared to one per five hundred conventional cholecystectomies. Good training programmes are available and simulators can be used to give the trainee an accurate “feel” for laparoscopic manipulation of the instruments.² The two following papers emphasize the need for careful selection of patients during the initial learning period together with the willingness to convert to an open operation in spite of the obvious psychological pressure not to do so once embarked on the procedure. Obviously patients have to be warned of the five to ten per cent chance of waking up with one large wound rather than four small ones!

Controversy exists regarding the pre- or peri-operative diagnosis of common ductal stones and their management in patients undergoing laparoscopic cholecystectomy. Although ultrasonography can demonstrate dilatation of ducts, it is rather poor at picking up ductal stones. Joyce and colleagues have demonstrated the accuracy of pre-operative intravenous cholangiography but many centres are reluctant to do this routinely because of the risk of hypersensitivity.³ Since only ten to fifteen per cent of patients with cholelithiasis can be expected to have choledocholithiasis, selective cholangiography makes sense. Patients with raised serum alkaline phosphatase, a history of obstructive jaundice, recent pancreatitis, or ultrasonic evidence of ductal dilatation should have either pre- or intra-operative cholangiography. When a stone is demonstrated in the common bile duct, should one proceed with laparoscopic cholecystectomy followed by ERCP,

sphincterotomy and stone retrieval or convert to open operation? At present I would advise the latter option, except in the elderly and unfit patient. Destruction of the biliary sphincter with resultant reflux chymobilia is not a good option long-term. A few centres are now doing laparoscopic choledochotomy and stone retrieval and doubtless this technique will become widespread in the near future, thereby solving the dilemma.

Laparoscopic cholecystectomy is one of the pioneering operations in the field of minimally invasive abdominal surgery and is the operation of choice for the majority of patients with gallstones. Many more procedures are already being established but we must be careful to practice the best surgery for the patient and not feel obliged to use a particular technique just because it is available.

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Laparoscopic cholecystectomy: experience in a district general hospital

S J Kirk, S B Kelly, S A A F Aly, V K Sharma, P G Bateson,
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Accepted 4 February 1992.

SUMMARY

In many centres laparoscopic cholecystectomy has become the procedure of choice for symptomatic gallbladder stones. By comparison with conventional cholecystectomy it appears to be associated with minimal morbidity, shorter hospital stay, earlier return to work and a better cosmetic result. The present study reviews the results of the first 50 laparoscopic cholecystectomies performed at Altnagelvin Area Hospital.

INTRODUCTION

The management of symptomatic gallstones has undergone a remarkable change in the past 18 months. The first cholecystectomy was performed by Langenuch¹ in 1882 and is still the 'gold standard' method for dealing with the diseased gallbladder. Alternative treatments which have been advocated recently for gallstones include dissolution therapy,^{2,3} extracorporeal shockwave lithotripsy,^{4,5} percutaneous cholecystectomy⁶ and cholecystolithotomy.⁷ All of these techniques, however, leave the gallbladder *in situ* with the possibility of stone recurrence.

Laparoscopic cholecystectomy was attempted in 50 consecutive patients and was successfully completed in 47 cases. Three patients (6%) were converted to open cholecystectomy. There was one death in the series. Apart from this case, the initial findings confirm the feasibility of this technique and demonstrate a low morbidity with minimal requirement for postoperative analgesia. These results compare favourably with other recent experience.^{8,9}

PATIENTS AND METHODS

Between February and October 1991, laparoscopic cholecystectomy was performed on 50 patients (10M : 40F) with symptomatic cholelithiasis; mean age was 44.9 years (range 22–68 years). Acute biliary infection was present in two patients; one of whom had acute cholecystitis (48 hours duration) and the

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other had cholangitis ten days prior to surgery, and had an ERCP performed two days prior to laparoscopic cholecystectomy. Diagnosis was made by ultrasound scan in 46 patients and by oral cholecystogram in four patients. Preoperative liver function tests were performed on all patients. With one exception; all patients were admitted one day prior to surgery.

Suitability for this procedure was determined using pre-defined selection criteria similar to those described by Zucker et al.¹⁰ Absolute contraindications were previous upper abdominal surgery, pregnancy, bleeding disorders, choledocholithiasis, jaundice and empyema of the gallbladder. Sixteen patients had previous abdominal surgery (7 hysterectomy, 6 appendectomy, 1 laparoscopy, 1 caesarean section and 1 lower midline incision — operation not known). All patients received prophylactic antibiotics (cephradine 500 mg or ceftioaxime 1 g) with induction of anaesthesia, and subcutaneous heparin 5,000 units subcutaneously eight hourly during the perioperative period until ambulatory.

Laparoscopic cholecystectomy is performed by two surgeons and one assistant; all procedures were performed under general anaesthesia with the patient supine. To minimise the risk of bladder or stomach injury, a nasogastric tube and urinary catheter were inserted in the first 21 patients. The remaining 29 patients were instructed to empty their bladder immediately before operation and only a nasogastric tube was inserted.

Pneumoperitoneum is established by inserting a Veress needle through the midline just below the umbilicus and approximately five litres of carbon dioxide is insufflated into the peritoneal cavity to maintain a pressure of approximately 15 mmHg (Solos rapid insufflator). To facilitate this, the patient is placed in the 20° trendelenburg position. The Veress needle is replaced by an 11 mm trocar and sheath through which is passed a laparoscope which is attached to a video camera. After a full laparoscopic examination of the abdomen, and under direct intraperitoneal vision, a second 11 mm diameter trocar is inserted just to the right of the midline and falciform ligament four finger breadths below the xiphoid. A third trocar (5 mm diameter) is then placed two finger breadths below the right subcostal margin in the midclavicular line and a fourth trocar (11 mm) is placed in the anterior axillary line three finger breadths below the right subcostal margin. The remainder of the procedure is performed by video dissection displayed on a television monitor.

Grasping forceps are inserted through a portal placed in the anterior axillary line, and the fundus of the gallbladder is grasped and pulled gently to the right and up over the liver edge to expose the gallbladder. The patient is then positioned in the 20° head-up position and rotated slightly to the left to allow the viscera to fall away from the gallbladder thus further improving the exposure. Through the upper portal in the mid-clavicular line, the surgeon inserts another pair of forceps which grasp the neck of the gallbladder and pull it gently to the right. This manoeuvre exposes Calot's triangle and by a combination of blunt and sharp dissection using forceps or diathermy scissors placed through the upper midline portal, the cystic duct and artery are exposed. The exposed artery and duct are divided between 6 mm titanium clips. Intraoperative cholangiography was not performed routinely. Using electrocautery the gallbladder is dissected from the gallbladder bed and removed via the anterior axillary portal. If a large stone is encountered the gallbladder neck can be delivered on to the skin. It is then

opened and following aspiration of bile, the stone is crushed by an instrument placed inside the gallbladder. Alternatively, the incision can be enlarged slightly using a grooved director and a scalpel and the gallbladder removed. The gallbladder bed is inspected for haemostasis, irrigated with saline and if necessary a drain may be placed.

RESULTS

Laparoscopic cholecystectomy was successful in 47 patients. Of the three failures, one had dense intra-abdominal adhesions, one had a very fibrosed gallbladder and in one patient it was not possible to create a pneumoperitoneum due to obesity. Operative time varied from 20 to 195 mins (Table I). In six patients trauma to the gallbladder resulted in bile leakage. A subhepatic suction drain was inserted in four patients. In one patient the gallbladder perforated when it was being removed from the abdomen and a large gallstone slipped back into the peritoneal cavity. The sub-umbilical incision was extended and the stone was removed manually. Blood loss in all procedures was minimal.

TABLE I
Total operative time from induction of anaesthesia

| <i>Operative time (mins)</i> | <i>Number of patients</i> |
|----------------------------------|-------------------------------|
| 0 – 60 | 12 |
| 61 – 120 | 26 |
| 121 – 180 | 5 |
| > 180 | 4 |

Wound problems were confined to bruising around the trocar sites. Oxygen saturation fell in one patient following induction of anaesthesia and during the operation. The cause for this was unknown but it was felt to be related to the laparoscopic procedure. Otherwise we did not identify any significant pulmonary complications. Three patients complained of shoulder pain up to 24 hours post-operatively. One patient developed chest pain two days postoperatively and required treatment for unstable angina.

There was one death, a 68-year-old female who had a difficult operation due to adhesions of the omentum and transverse colon to the gallbladder. She developed septicaemia due to a large subphrenic and subhepatic collection of fluid. Approximately one litre of bile and blood was aspirated under ultrasound control three days postoperatively. She was treated with metronidazole, ceftioaxime, and gentamicin intravenously, but her condition gradually deteriorated and she died two days later. At autopsy, there was no evidence of damage to anatomical structure including the bile ducts, hepatic ducts or cystic artery.

Forty-four patients received intravenous crystalloid solutions for one postoperative night, two patients for 48 hrs and one patient for five days (subsequently died). Hospital stay was compared to the last 50 open cholecystectomies carried out by the same surgical team. This was shorter in the laparoscopic cholecystectomy

group, mean 4.2 days (range 3–10 days), compared to the open cholecystectomy group, mean 8.0 days (range 3–18 days) (Table II). The patient in whom oxygen saturation fell remained in hospital for seven days and the patient with unstable angina stayed nine days. Intramuscular postoperative analgesic requirement was as shown (Table III). The patient who developed the subphrenic fluid collection and subsequently died had seven injections of morphine/cyclizine followed by intravenous morphine infusion. Most patients took simple non-narcotic analgesia up to discharge.

TABLE II

Total hospital stay in 47 patients having laparoscopic cholecystectomy (mean 4.2 days, range 3–10 days), and 50 patients having open cholecystectomy (mean 8.0 days, range 3–18 days)

| <i>Hospital stay days</i> | <i>Laparoscopic cholecystectomy (n = 47)</i> | <i>Open cholecystectomy (n = 50)</i> |
|-------------------------------|--|--|
| 3 | 15 | 1 |
| 4 | 19 | 4 |
| 5 | 5 | 6 |
| 6 | 6 | 9 |
| 7 | 1 | 5 |
| 8 | 0 | 4 |
| 9 | 0 | 9 |
| 10 | 1 | 4 |
| 11 | 0 | 2 |
| 12 | 0 | 1 |
| 13 | 0 | 2 |
| 15 | 0 | 2 |
| 18 | 0 | 1 |

TABLE III

Total number of postoperative injections for analgesia per patient (either morphine tartrate 10mg with cyclizine tartrate 50mg/ml (44 patients) or dihydrocodeine tartrate 50mg/ml (3 patients))

| <i>Number of injections</i> | <i>Number of patients</i> |
|---------------------------------|-------------------------------|
| 0 | 3 |
| 1 | 9 |
| 2 | 13 |
| 3 | 9 |
| 4 | 11 |
| 5 | 1 |
| 7 | 1 |

DISCUSSION

Apart from one death in this series, our initial experience with laparoscopic cholecystectomy has been most rewarding, particularly in terms of patient recovery, reduced hospital stay and minimal requirements for postoperative analgesia. The procedure however, takes longer than open cholecystectomy, but with increasing experience operating time should decrease.

The requirement for postoperative analgesia is minimal, 95% of our patients requiring four or less injections for pain relief. In addition, postoperative morbidity is relatively trivial and infrequent, self-limiting shoulder pain and wound bruising being most notable. The length of postoperative hospital stay in our series is longer than that reported by Reddick and Olsen¹¹ who performed 60% of their (carefully selected) patients as day cases. This was due to our relative caution with the early patients, whom we wished to observe well into their recovery period in case they developed as yet unidentified complications. With increasing experience in the management of these patients, we have found that, even in our small series, the duration of hospital stay has decreased.

Preliminary results from many centres suggest that laparoscopic cholecystectomy has important potential, but larger series are required before its permanent place in the management of gallbladder stones can be established. Whenever difficulties arise which render continuation of the procedure unwise or unsafe, the surgeon should have no hesitation in converting to a standard open cholecystectomy. This should be regarded as an option rather than a complication and if appropriately exercised, it should limit the risk of adverse outcome.

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Initial experience of laparoscopic cholecystectomy in a district hospital

L D McKie, I Samuel, J W R Peyton, R Campbell, M Lutton,
D Strehorn, H McNeill

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SUMMARY

Fifty-five consecutive unselected patients were submitted for laparoscopic cholecystectomy, and the procedure completed laparoscopically in fifty cases. The outcome is presented with particular reference to the duration of surgery, postoperative pain and nausea, the length of hospital stay and the time taken to recover normal activities. This technique is shown to have major advantages over conventional gallbladder surgery for the majority of patients.

INTRODUCTION

Cholecystectomy has been the treatment of choice for symptomatic gallstones for many years. The operation of cholecystectomy has a record of safety, but it is painful for the patient, and is expensive in terms of hospitalisation and time taken off work. It is also associated with a certain morbidity in terms of chest problems and thromboembolic complications in the postoperative period. In recent years attempts have been made to reduce the impact of this operation by using techniques such as gallstone dissolution and lithotripsy. These have so far proved rather unsatisfactory, in that only a minority of patients are suitable for these types of treatment, and they all leave a diseased gallbladder in place, which means symptoms can continue and gallstones may reform. More recently, advances in television camera technology have made possible the operation of laparoscopic cholecystectomy. Since this procedure was first reported from Paris in May 1988,¹ it has been adopted in many centres world-wide and, although so far only small series have been reported, it does appear to offer a considerable benefit in terms of patient discomfort, hospitalisation time, time off work and, ultimately, cost.^{2, 3}

Between May and August 1991, 55 cholecystectomies have been performed at South Tyrone Hospital, 50 of them laparoscopically. We present the results of our experience with this new technique to date.

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PATIENTS AND METHODS

Fifty-five consecutive patients presented with gallbladder disease and were offered cholecystectomy according to the usual criteria. There were 41 females and 14 males aged 19 to 75 years (mean age 46). One patient was operated on as an emergency with severe biliary colic, the others were all booked via the outpatient clinic. All patients had liver function tests and ultrasound scans of their bile ducts performed preoperatively, and in all cases the liver function tests were normal and ultrasound revealed no evidence of stones in the common bile duct. All patients were offered laparoscopic cholecystectomy, with the proviso that conversion to an open operation would take place if this proved impossible or too difficult. There were no exclusion criteria in this series.

All patients were managed according to the following regimen:

PREMEDICATION: All patients received suppositories containing 100mg diclofenac sodium (Voltarol — Geigy Pharmaceuticals) two hours before surgery. In addition, where the patient seemed very anxious, an anxiolytic agent such as diazepam 10mg or tempazepam 20mg was also given orally. A few patients with severe dyspeptic symptoms received ranitidine 150mg.

ANAESTHESIA: This was induced with a standard dose of sodium thiopentone. All patients were intubated with the help of a muscle relaxant and respiration was controlled throughout the operation. Anaesthesia was maintained with N₂O₂ and halothane or isoflurane, with additions of droperidol and an opiate analgesic in appropriate doses. The following parameters were monitored: ECG, non-invasive blood pressure, O₂ saturation (pulse oximetry), and end-tidal CO₂ (capnography).

SURGERY: The actual technique of laparoscopic cholecystectomy was similar to that described by Reddick and Olson,⁴ except that a diathermy hook was used for the dissection rather than a laser. Four trocars were inserted into the abdomen, one at the umbilicus, one in the epigastrium just to the right of the falciform ligament, one just below the right costal margin in the mid-clavicular line and one lower down in the right flank. Ligaclips were used on the cystic artery and duct, except in two cases where the duct was very large, where a catgut 'Endoloop' was used instead. No operative cholangiograms were performed on any of these patients. At the end of the procedure, the gallbladder was withdrawn via the umbilical incision. In five cases the operation was converted to an open cholecystectomy, due to adhesions in three patients, a very inflamed gallbladder in one, and an empyema in the fifth.

Patients were interviewed on the first postoperative day and information recorded regarding the incidence of nausea and vomiting, and the severity and location of pain, and all drugs administered.

In the fifty patients who had successful laparoscopic cholecystectomy the time taken to perform the various stages of the operation was recorded. Patients were discharged when they were mobile and on a normal diet — usually the following day. They were all reviewed one week later and a questionnaire completed. Each patient was asked how long it had taken them to return to normal activities and how much analgesia they had required. They were also asked to score on a visual analogue scale the severity of their symptoms (from 0 to 100) from their

gallbladder disease, and their experience of the operation (from 0 = 'What operation?' to 100 = 'The worst experience imaginable!'). In those patients in whom it was applicable, the time taken before returning to work was also recorded.

RESULTS

50 laparoscopic cholecystectomies were performed. The times taken to perform the various stages of the operation were as follows:

| | <i>Minimum</i> | <i>Median</i> | <i>Maximum</i> |
|-------------------------------|----------------|---------------|----------------|
| Anaesthetic time (mins) | 45 | 98 | 190 |
| Operating time (mins) | 30 | 70 | 170 |
| Gallbladder dissection (mins) | 15 | 40 | 145 |

30 patients (78%) were discharged the following day, and a further six (12%) went home on the second postoperative day. Of those patients not discharged the next day, one had had a recent myocardial infarction and was transferred to a medical ward. One patient already suffered from multiple sclerosis which delayed recovery, a few patients complained of postoperative nausea with subsequent delay in discharge, and some patients remained in hospital briefly because of social reasons. The mean duration of postoperative hospital stay was 1.3 days. There were no surgical complications in any of these patients, and none developed any respiratory or thromboembolic problems.

Of the 50 patients having laparoscopic cholecystectomy, 26 (52%) suffered from nausea and of these 19 actually vomited. All the 19 patients who vomited were given specific antiemetics (cyclizine 50mg or metoclopramide 10mg). While 13 patients settled with one injection, six required repeated injections of either drug. Forty patients complained of pain in the right upper quadrant, but only two felt that the pain was severe and unbearable, while the rest claimed that the pain was slight to moderate. In addition, 25 also reported slight generalised 'ache' all over their abdomen and 13 complained of generalised 'stiffness' all over their body. Shoulder pain occurred in 14 patients (28%), mainly on the right side, but in three cases it was bilateral. Amongst 40 patients who did complain of pain, only 2 needed an opiate analgesic for relief, the rest were pain-free with mild analgesics such as diclofenac suppositories and/or paracetamol orally.

Time taken to return to normal activities ranged from one day to 14 days (median four days). Time taken to return to work (for those in whom it was applicable) ranged from three days to 21 days (median 12 days). As most of these operations were performed on a Wednesday, this meant a return to work on the second Monday after the operation for the average patient. On the visual analogue scale, the mean score awarded to the operation by the patients at review was 23 (range 3 to 76), compared with a mean score of 67 (range 0 to 95) for the symptoms of their disease.

Some comparative data were obtained by reviewing the case notes and anaesthetic records of fifty patients undergoing conventional surgery (open cholecystectomy with operative cholangiogram) prior to beginning the laparoscopic programme. This showed a median total anaesthetic time of 60 minutes and a mean postoperative hospital stay of 5.4 days. It was also noted in this

group that there was one postoperative chest infection which was significant in that it delayed the patient's discharge from hospital by one week.

DISCUSSION

Recently reported results from the United States⁵ have suggested that laparoscopic cholecystectomy is the treatment of choice for the majority of patients. The conversion rate to open operation at experienced centres has been reported at 3.5 – 6.9%.^{2,5} In our series the conversion rate was 9%, which seems reasonable for a centre where the technique is just being developed. It would be expected that with increasing experience, the conversion rate would fall. The procedure appears safe and no surgical complications were experienced.

One point of concern to surgeons is that the operation takes longer than a conventional cholecystectomy. This is certainly true, but in our experience the extra time spent was not felt to be excessive. With experience, dissection became quicker, and a graph plotted of gallbladder dissection time against experience for one surgeon does appear to show such a trend, but this was not statistically significant.

Postoperative nausea and vomiting during the first 24 hours is the most disturbing side-effect. Whilst no 'in-depth' study was carried out, it is our impression that this is unrelated to the type of anaesthetic drugs used or the duration of the anaesthetic. Pain appears not to be a problem. All but two patients were comfortable with simple analgesics only, which is a distinct advantage over an open procedure, particularly for patients with diminished respiratory function.

We feel that laparoscopic cholecystectomy is a safe, cost-effective alternative to open surgery for the majority of gallstone sufferers. We routinely offer this operation to all patients, with the proviso that conversion to an open operation may be required. The reduction in postoperative pain is dramatic, and the only problem encountered was some early nausea and vomiting. Laparoscopic cholecystectomy may well come to be the treatment of choice in the future.

POSTSCRIPT

As of 5th February 1992, 101 laparoscopic cholecystectomies have been completed on 108 patients submitted to this procedure (conversion rate 6.5%). Of the last fifty patients, only two have required conversion to open surgery. There have been no significant complications, and the mean duration of postoperative hospital stay remains 1.3 days. This work represents the learning experience of two consultant surgeons, two registrars and eleven visiting surgeons who have attended two training courses held at South Tyrone Hospital.

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Deliberate self-poisoning presenting at Craigavon Area Hospital: 1976 and 1986

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SUMMARY

Deliberate self-poisoning presenting at Craigavon Area Hospital was compared in the years 1976 and 1986. A decline in the use of benzodiazepines and increase in other agents, notably paracetamol, is reported. Alcohol is frequently associated with deliberate self-poisoning, while severe mental illness is less common. Diagnostic practice may have effects on bed occupancy in this group.

INTRODUCTION

Deliberate self-poisoning is one of the largest public health problems of our age.¹ It is a source of considerable morbidity and mortality as well as consuming scarce medical resources. Environmental and cultural factors are known to influence the rate of self-poisoning.² For this reason it may be advantageous to study the problem in a relatively stable population over time.

We wished to see if there had been a change in demographic characteristics and psychiatric diagnosis in this condition over a decade. This is important at both national and local level to ensure adequate planning of resources. Local studies may show differences in the incidence or characteristics of deliberate self-poisoning which increase our knowledge of its causes. The types of drug used by these patients change, depending on their availability by prescription or direct sale from the dispensing chemist. Changes in drugs used can have a profound effect on mortality, as has been seen with the barbiturates.

METHODS

Craigavon Area Hospital is a district general hospital serving three district council areas of total population 150,000. There are several towns with a large rural hinterland. No other hospital deals with self-poisoning for this area. It is usual for cases of self-poisoning to be referred to hospital because of difficulty in ascertaining the quantity of drug taken. All cases are seen in the casualty department and most remain overnight for observation. The more severe cases of poisoning may require transfer to a medical or intensive care unit.

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All casualty records for the years 1976 and 1986 were retrospectively surveyed. Cases of deliberate self-poisoning were identified and all relevant medical and psychiatric notes obtained. Deliberate self-poisoning was defined as a deliberate non-fatal drug overdose in the knowledge that it would be potentially harmful and that the amount taken was excessive. Demographic and clinical data were recorded, including drugs taken, clinical diagnosis and outcome. Data was analysed using the SPSS computer package and chi-squared test was used for comparison between years.

RESULTS

Demographic data

There were 265 recorded episodes of deliberate self-poisoning in 1976 (1.82/1000 of population) compared to 228 in 1986 (1.42/1000). Other data (unpublished) suggests that self-poisoning increased after 1976 and declined in the mid-1980's in the area served by this hospital. Deliberate self-poisoning was most common in the 30 – 39 year age group in 1976 but in the 10 – 19 year age group in 1986. (Figure). The sex distribution was unchanged, remaining at female/male = 1.5/1 in both years.

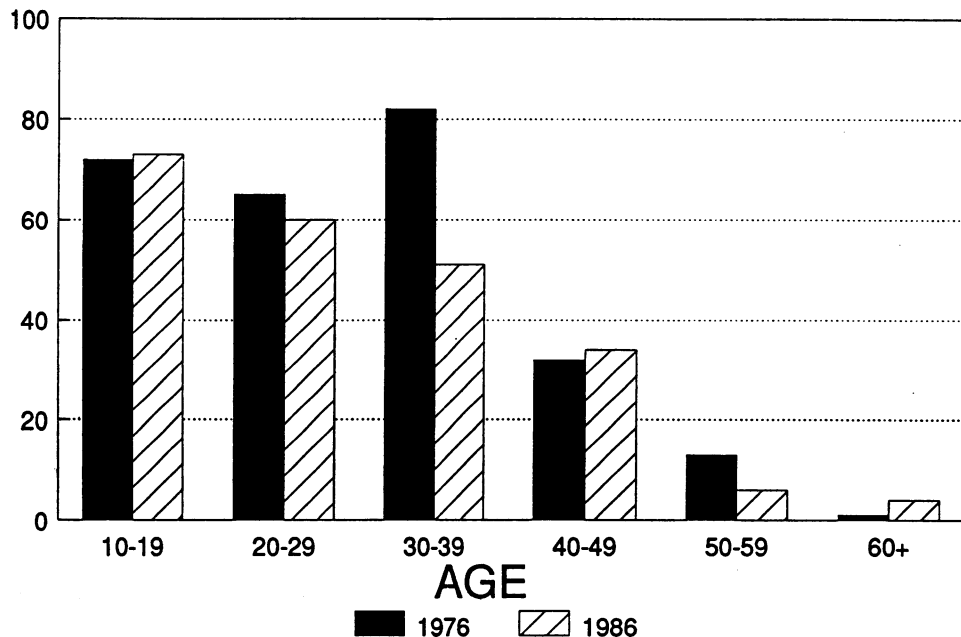


Figure. Age distribution of cases of deliberate self-poisoning treated at Craigavon Area Hospital in 1976 (265 cases) and 1986 (228 cases).

Drug and alcohol use

We do not consider excessive intoxication with alcohol alone as a form of deliberate self-poisoning, as its motivation is different in the majority of cases. Alcohol was taken (with another drug) in 36% of episodes classified as deliberate self-poisoning in 1976, and in 38% in 1986. It remains an important association and may be the main cause in many cases.³

The drugs used for deliberate self-poisoning are shown in Table I. Benzodiazepines alone were commonly used both in 1976 and 1986, but declined markedly from 40.8% of all episodes in 1976 to 26.8% in 1986 ($X^2 = 4.38$, $p < 0.05$). The number of episodes in which benzodiazepines were used (alone or in combination with other drugs) fell from 155 in 1976 to 97 in 1986 ($X^2 = 4.07$, $p < 0.05$). This fall in benzodiazepine use occurred both in those above and below thirty years of age.

Paracetamol alone was used in 0.8% of episodes in 1976 and 9.2% in 1986. This increase is also significant ($X^2 = 7.43$, $p < 0.01$). The use of paracetamol in combination with other drugs was also greater in 1986 than in 1976. There was an increase in anti-inflammatory drug use in 1986, and drug combinations in overdose were seen more often in 1986 (35.9%) than in 1976 (22.6%).

TABLE I
Drugs used for deliberate self-poisoning (%)

| | 1976 <i>n</i> = 265 | 1986 <i>n</i> = 228 |
|---|------------------------|------------------------|
| Benzodiazepines | 108 (40.8%) | 61 (26.8%) |
| Paracetamol | 2 (0.8%) | 21 (9.2%) |
| Aspirin | 17 (6.4%) | 9 (3.9%) |
| Neuroleptics | 5 (1.9%) | 2 (0.9%) |
| Anti-depressants | 17 (6.4%) | 9 (3.9%) |
| Anti-convulsants | 5 (1.9%) | 3 (1.3%) |
| Anti-inflammatory | 2 (0.8%) | 12 (5.3%) |
| Combination, without psychotropic drugs | 7 (2.6%) | 32 (14.0%) |
| Combination, with psychotropic drugs | 53 (20.9%) | 50 (21.9%) |
| Unknown, others | 49 (18.4%) | 29 (12.8%) |

Diagnosis

Case note diagnosis for the deliberate self-poisoning patients is shown in Table II. Situational disturbance refers to a short period of distress related to a specific stress, reactive depression refers to a more prolonged episode of low mood with characteristic clinical features which often requires intervention. Situational disturbance was diagnosed more often in 1986 than in 1976 ($X^2 = 11.5$, $p = 0.0007$). It is likely that situational disturbance in 1986 included some patients who would have been diagnosed as reactive depression in 1976.

Alcoholism and situational disturbance account for the majority of cases of deliberate self-poisoning. Severe mental illness such as schizophrenia and endogenous depression account for a small but steady proportion. There was no significant difference between these disorders in 1976 and 1986.

TABLE II
Diagnosis of deliberate self-poisoning patients (%)

| | 1976 <i>n</i> = 265 | 1986 <i>n</i> = 228 |
|------------------------------|------------------------|------------------------|
| Situational reaction | 78 (29.4%) | 107 (46.9%) |
| Reactive depression/neuroses | 66 (24.9%) | 26 (11.8%) |
| Personality disorder | 18 (6.8%) | 18 (7.9%) |
| Alcoholism | 61 (23.0%) | 30 (13.2%) |
| Benzodiazepine dependence | 2 (0.8%) | 3 (1.3%) |
| Other drug abuse/dependence | 3 (1.1%) | 1 (0.4%) |
| Schizophrenia | 2 (0.8%) | 5 (2.2%) |
| Endogenous depression | 15 (5.7%) | 7 (3.1%) |
| Mental handicap | 0 (0.0%) | 3 (1.3%) |
| Unknown | 20 (7.5%) | 28 (11.9%) |

Assessment and outcome

79.5% of our patients were referred to psychiatric services in 1976 and 71% in 1986 (Table III). There was a significant ($X^2 = 7.2$, $p = 0.007$) decrease in those referred in 1986 which may be related to improved assessment by staff in the Accident and Emergency department. Some patients left before psychiatric assessment was possible (1976 = 5%, 1986 = 9%). These figures are lower than in other studies^{4,5} and may reflect the ease with which assessments can be obtained in this hospital.

TABLE III
Assessment by psychiatrist (%)

| | 1976 <i>n</i> = 265 | 1986 <i>n</i> = 228 |
|------------------------|------------------------|------------------------|
| Seen by psychiatrist | 211 (79.5%) | 162 (71.0%) |
| Not referred | 16 (6.0%) | 36 (16.0%) |
| No data | 25 (9.5%) | 9 (4.0%) |
| Left before assessment | 13 (5.0%) | 21 (9.0%) |

The outcome of the patients in this study is shown in Table IV. The most noticeable feature is the much lower admission to the psychiatric unit in 1986 which must reflect change in diagnostic practice, as noted above. This was statistically highly significant ($X^2 = 14.3$, $p = 0.0002$). Correspondingly there was an increase in those discharged with no follow-up and those requested to attend out-patient clinics. At least one-third of patients failed to attend out-patient appointments in both years.

TABLE IV
Outcome of patients (%)

| | 1976 n = 265 | 1986 n = 228 |
|---|-----------------|-----------------|
| Discharged/no follow-up | 46 (17.4%) | 86 (35.5%) |
| Out-patients | 43 (16.2%) | 48 (21.1%) |
| Admitted to psychiatric unit | 120 (45.3%) | 36 (15.8%) |
| Left contrary to advice (before or after assessment) | 31 (11.7%) | 50 (22.0%) |
| Unknown | 25 (9.4%) | 8 (3.5%) |

DISCUSSION

The purpose of this study was to review the demographic characteristics, clinical diagnosis and outcome of patients who self-poison within a specific catchment area. The reasons for this are to be aware of change in drug use, the frequency and type of psychiatric disorder diagnosed, and the resources taken up by this activity. Most studies are from large urban centres and their findings may not apply to rural areas. The major limitation of this study is the retrospective information collection. Despite this, good clinical notes enabled accurate demographic and drug data to be collected in over 90% of cases. Even prospective studies, in this group of patients, have 20% of patients lost to follow-up.⁶

There was a marked decline in the use of benzodiazepines for deliberate self-poisoning. This has been noted to a lesser degree in some,⁷ but not in all,⁸ recent studies of similar nature. The most obvious reason is the documented reduction in benzodiazepine prescribing⁹ following on from fears of dependency.¹⁰ Although starting from a low baseline compared with similar studies,¹¹ the increase in paracetamol poisoning is worrying. This is occurring later than in other UK centres. Paracetamol is toxic and is likely to produce fatalities if not treated promptly. The inverse relationship between benzodiazepine and paracetamol poisoning is disturbing, though no causal connection can be attributed on this evidence. An increased use of non-steroidal anti-inflammatory drugs for self-poisoning occurred and has been observed in other centres.¹²

Alcohol is frequently related to deliberate self-poisoning. Our results are similar to other studies^{11, 13} in this respect. It is likely that alcohol acts as a disinhibiting agent in many cases where self-poisoning would not otherwise occur. The correlation between increase in self-poisoning and relaxation of licensing laws strengthens this association.³

Our patients were younger in 1986 than in 1976. This has been seen in other centres and is reflected in the peak age of self-poisoning between samples in the 1970s^{14, 15} and 1980s.^{5, 16} There is evidence that this trend may be changing with a decline in self-poisoning particularly in young females.¹⁷ The female/male sex ratio was 2/1 in early studies.¹³ Our ratio of 1.5/1 is similar to more recent reports.⁴ It has been noted that the trend is toward unity⁸ (for unknown reasons),

and a similar trend has been reported from another study in N. Ireland.⁵ Retro-spective diagnosis in these patients is particularly difficult as mental state can change rapidly in the hours/days after admission. Diagnoses were based on clinical interviews carried out by several psychiatrists. No standardised diagnostic criteria were used. The aim was to study clinical practice only, and significant findings were observed. More patients were diagnosed as having transient situational disturbance as against reactive depression/neurosis in 1986, and this diagnostic change was reflected in service utilization, particularly in decreased psychiatric admissions. This change may represent improved psychiatric training with greater confidence in diagnosing minor disorders without resorting to hospital admission for assessment.

Despite retrospective diagnosis, this study is atypical in regard to the large number of patients seen by a psychiatrist, so that a psychiatric diagnosis was frequently available. Most of our patients have no major mental illness though a few suffer from severe depressive illness or psychosis. It is reassuring, given the diagnostic problems, that this agrees with previously published work.^{18, 19} The years 1976 and 1986 were chosen to allow time for demographic trends in deliberate self-poisoning to become evident. This data will also act as a baseline for further assessments to be carried out at 5-yearly intervals.

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The changing pattern of cervical cancer in Northern Ireland 1965 – 1989

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SUMMARY

There was a change in pattern of and increased prevalence of cervical cancer in Northern Ireland from 1965 to 1989, characterised by an increased incidence in women under 40 years. These changes occurred despite special screening of younger women, although the screening programme has probably prevented an even greater increase in incidence of the disease. To reduce the incidence of cervical cancer, not only systematic screening but also cervical smears at more frequent intervals would be required.

INTRODUCTION

Community cervical screening commenced in a random fashion in Northern Ireland in 1965. The effect of this screening on the incidence or mortality from cervical cancer is unknown, as the registration of cancer, especially of the cervix, is very incomplete in the province.¹ We therefore assessed the incidence of cervical cancer in Northern Ireland from 1965 to 1989 by a study of biopsy records to determine the impact of 25 years of cervical screening on changes in the pattern of the disease.

METHODS

Biopsy reports of all cases of cervical cancer occurring in Northern Ireland from 1965 to 1989 were reviewed in a survey of the records of the four histopathology laboratories in the province. Microinvasive cases were excluded, and invasive cases included only when the depth of invasion was considered by the histopathologist to be more than 5 mm. This decision was made because it became clear that in an appreciable number of biopsies intraepithelial neoplasia involving the endocervical crypts was difficult to differentiate from microinvasion, especially if there was an associated inflammatory reaction. There was also a marked variation in the frequency with which microinvasion was reported between different laboratories, also reflecting this difficulty in interpretation. Cases of adenocarcinoma were only included if the pathologist considered that the tumour was definitely cervical rather than endometrial in origin. During the study other potential sources of inaccuracy became apparent. In some cases the distinction between microinvasive and fully invasive disease was not clear from the report.

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In others, carcinoma secondary to the cervix had been miscoded as primary cervical cancer, or diagnostic problems arose because the patient had a history of carcinoma in another organ. Whenever possible any uncertainty in diagnosis was resolved by a review of the biopsy or examination of the patient's hospital notes.

By these means patients were grouped according to their year of diagnosis, age and the histological type of the tumour. In 37 cases (2.4%) the patient's age could not be determined and these cases were excluded.

RESULTS

The age-specific incidence rates of cervical cancer, excluding microinvasive cases, for five year periods from 1965 – 89 are shown in the Table. During the period 1975 – 89, the incidence in women aged less than 40 has more than doubled. The increase first affected those under 30 years, the rate not increasing in those aged 30 – 39 until 1980. From 1975 – 89 cervical cancer declined in those aged 45 – 59.

TABLE

Annual incidence rates of cervical cancer per million women according to age group and period

| Age (years) | Period | | | | |
|-------------|----------|----------|----------|----------|----------|
| | 1965 – 9 | 1970 – 4 | 1975 – 9 | 1980 – 4 | 1985 – 9 |
| 20 – 24 | 0.0 | 0.0 | 11.3 | 9.7 | 12.4 |
| 25 – 29 | 17.7 | 23.9 | 41.5 | 31.1 | 34.1 |
| 30 – 34 | 28.1 | 50.1 | 46.7 | 99.4 | 108.5 |
| 35 – 39 | 92.2 | 72.4 | 81.0 | 101.4 | 162.5 |
| 40 – 44 | 155.7 | 100.5 | 79.7 | 157.9 | 208.3 |
| 45 – 49 | 174.3 | 220.1 | 139.8 | 137.2 | 107.0 |
| 50 – 54 | 251.1 | 242.0 | 169.4 | 179.4 | 155.6 |
| 55 – 59 | 205.7 | 282.4 | 224.9 | 195.5 | 174.4 |
| 60 – 64 | 192.7 | 169.5 | 156.7 | 198.1 | 221.6 |
| 65 – 69 | 164.4 | 135.4 | 175.2 | 182.5 | 243.3 |
| 70 – 74 | 140.9 | 137.3 | 206.7 | 116.4 | 180.9 |
| 75 – 79 | 115.0 | 121.7 | 105.4 | 61.7 | 136.0 |
| 80 + | 62.4 | 87.6 | 107.9 | 68.7 | 84.0 |

The histological type of cervical cancer according to age group for each five year period is shown in the Figure. From 1965 to 1989 about 10% of all the cervical cancers in each five year period were adenocarcinomas or adenosquamous tumours. In 1980 – 84 there was a fivefold increase in adenocarcinoma affecting women aged less than 45 years compared to 1965 – 79. This increase was most marked in the 20 – 39 year age group. This increased incidence became even more marked in 1985 – 89 when adenocarcinoma occurred about eight times more frequently in those under 45 years, and in women aged 20 – 39 years these tumours constituted almost 20% of carcinomas.

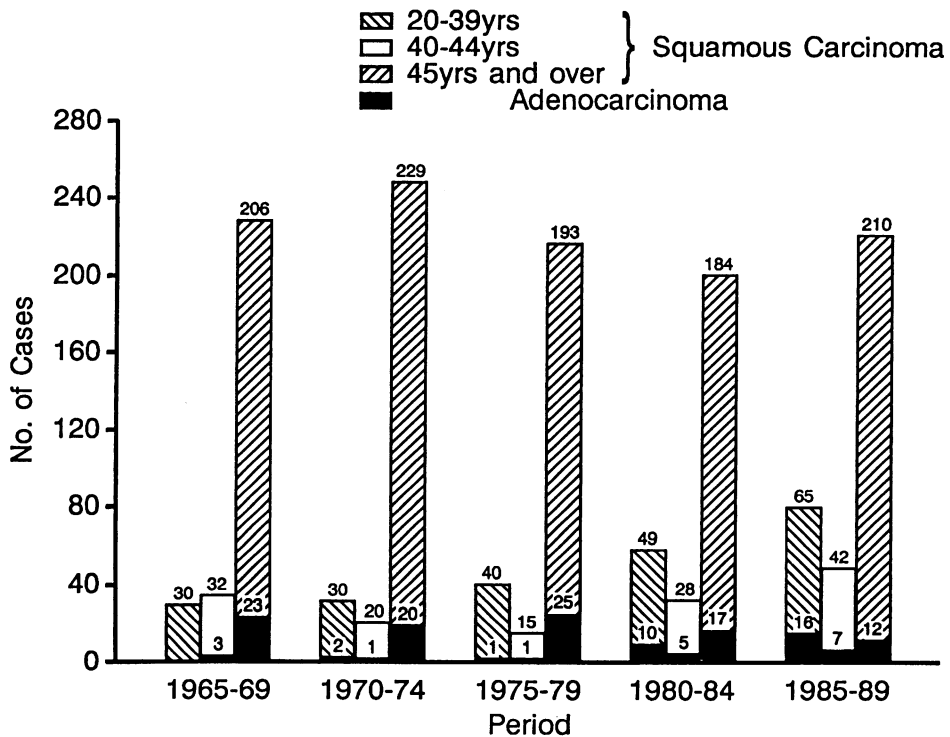


Figure. Distribution of histological types of carcinoma according to age groups and period. Adenocarcinoma includes 10% of adenosquamous tumours. Undifferentiated carcinomas are included with the squamous tumours. The figures on top of the columns indicate the number of each histological type.

DISCUSSION

In 1989 there were 547,941 women aged 20 years and over resident in Northern Ireland. Although this survey is based on a smaller population than those dealing with cervical cancer in Britain, its precision is increased by being based on biopsy reports. Northern Ireland is also a relatively discrete geographic area and is less affected by cross-boundary population changes which can influence results in larger regions. Moreover, most previous reports have relied on mortality rates which are subject to diagnostic inaccuracy and incomplete registration. Registration of cancer mortality varies in its completeness in Britain and, as in Northern Ireland, cervical cancer deaths are probably especially poorly recorded.^{1, 2}

Our results clearly show that since 1975 there has been a very marked change in the pattern of cervical cancer, characterised by an increased incidence of squamous carcinoma in young women followed five years later by a striking increase in adenocarcinoma in the same age groups. A similar increase in mortality rate among younger women was noted in England and Wales³ but the corresponding rise in incidence rates appears to have occurred five or more years earlier than in Northern Ireland. Almost certainly incidence rates in the province would have been appreciably higher in the absence of screening. Our results, for

example, do not include microinvasive cases which were detected by cervical smear and prevented from progressing. Also, from 1965–89 this laboratory detected moderate or severe dyskaryosis in about 4,600 women, which if untreated would also have increased the cancer incidence rate.

It is particularly disappointing to find that the incidence of cervical cancer in Northern Ireland has not decreased among younger women. During the 25 years of screening we estimate that about 1.25 million cervical smears have been examined in the province and a laboratory survey shows that 67% of these were from women 20–39 years old, a population comprising 226,194 in 1989. Despite this intensive screening effort it is women of this age group who are now showing a rising cancer incidence. Twenty years ago, in 1971 Brown and Lynch reported a population survey in the province which showed that even then 40% of women under 40 years had been screened at least once.⁴ In Britain it was estimated that by 1981 nearly 80% of women under 35 years were being screened.⁵

Our results suggest some possible causes for this relative failure of screening. The incidence rates clearly indicate that the screening programme was being conducted in the face of an increasing rate of occurrence of cervical cancer, probably more pronounced than indicated by our incidence rates. Also of concern is the relatively marked increase in the proportion of adenocarcinoma in younger women. These tumours are much harder to detect than squamous carcinoma in their preinvasive stages. This change in pattern of adenocarcinoma might reflect an underlying change in the biology of the disease which if associated with a shortened intraepithelial phase of squamous tumours would make them more difficult to detect by screening. Such a possibility has been suggested by others but so far not substantiated.⁶

Two other aspects of the screening programme in Northern Ireland could well be of importance in its relative failure. It was random in nature and probably less effective in attracting those young women most at risk, but a gross failure in this respect would be required to account for our results. Furthermore, doctors and patients were advised that smears should be repeated at five year intervals.

Although even a single negative smear has been said to have considerable protective value,^{7,8} there is evidence that false negative smears are not infrequent and that the protection given by one negative result is relatively low.^{9,10} Our findings show that random screening has been inadequate in the prevention of cervical cancer in Northern Ireland and we feel that even with systematic screening, a five year interval between smears will prove too long to counter an increasing incidence of disease.

We hope that a current study of the screening history with review of cervical smears from all those who developed cervical cancer from 1982–89 will provide more insight into some of these issues.

We thank the histopathologists, gynaecologists and radiotherapists of the province for access to biopsy reports and hospital notes. We are also grateful to Dr Carol McCaugherty of the Eastern Health and Social Services Board for advice and to Miss Jennifer McCullough for secretarial assistance.

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Renal transplantation in Northern Ireland 1968 – 1990

D Middleton, C Cullen

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INTRODUCTION

The first renal transplant was performed in Northern Ireland in 1968. From 1968 – 1990 a total of 618 transplants were carried out on 535 patients at the Renal Unit, Belfast City Hospital. This report analyses various factors affecting recipient and donor, to determine how these factors have changed during the past 22 years and to ascertain their effect on graft survival.

MATERIALS AND METHODS

From 1968 – 1990 a total of 618 transplants were performed at the Renal Unit, Belfast City Hospital. Of these 568 were cadaveric and 50 were live related transplants. There were 491 first, 67 second and 10 third cadaveric transplants. The figures for live transplants were 44 first, 5 second and one fourth transplant. The details of all transplants are held on a computerized data base in this laboratory. Actuarial graft survivals are calculated from the data base using the Log Rank survival programme¹ which is used in a version suitable for personal computers.² Death with a functioning graft was taken as graft failure, which is the usual practice in most centres in analysing graft survival.

RESULTS

Fig 1 shows the source of the cadaveric kidneys (local or imported from another centre) used for Belfast recipients in each year. The number of cadaveric donors obtained from within Northern Ireland between 1972 – 90 is given in Fig 2. The number of donors per million population was 21·8 in 1987 and 16·0 in 1990. Kidneys from a local donor need not necessarily be used for a local recipient, but reliable records of local donors whose kidneys were not used locally are only available from 1972. The number of cadaveric donors received from each of the local hospitals for each of the years 1982 – 1990 is shown in the Table.

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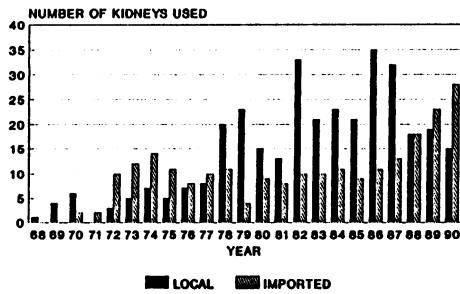


Fig 1. Source of cadaveric kidney used for Belfast recipient.

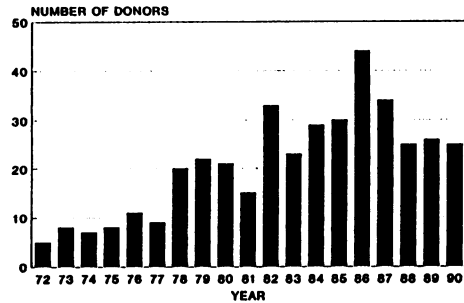


Fig 2. Local cadaveric donors obtained each year.

TABLE

Cadaveric donors from local hospitals 1982–90

| | 1982 | 1983 | 1984 | 1985 | 1986 | 1987 | 1988 | 1989 | 1990 | Total |
|----------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|------------|
| Royal Victoria | 19 | 14 | 15 | 18 | 29 | 26 | 17 | 17 | 15 | 170 |
| Belfast City | 2 | 1 | 1 | 2 | 3 | 2 | 6 | 2 | 3 | 22 |
| Ulster | 7 | 3 | 3 | 1 | 1 | 2 | — | 1 | 1 | 19 |
| Altnagelvin | 1 | 1 | 1 | 2 | 2 | 1 | 1 | 2 | 1 | 12 |
| Mater | 2 | 3 | 3 | 1 | 3 | — | — | — | 1 | 13 |
| Craigavon | 1 | — | 1 | 3 | 3 | — | 1 | — | 2 | 11 |
| Tyrone County | 1 | — | 4 | 1 | — | — | — | — | 1 | 7 |
| Mid-Ulster | — | — | — | 1 | 2 | 2 | — | — | — | 5 |
| Coleraine | — | 1 | — | — | — | 1 | — | 1 | — | 3 |
| Erne | — | — | — | — | — | — | — | 3 | — | 3 |
| South Tyrone | — | — | — | — | — | — | — | — | 1 | 1 |
| Waveney | — | — | — | — | 1 | — | — | — | — | 1 |
| Downe | — | — | — | 1 | — | — | — | — | — | 1 |
| Ards | — | — | 1 | — | — | — | — | — | — | 1 |
| Total | 33 | 23 | 29 | 30 | 44 | 34 | 25 | 26 | 25 | 269 |

A comparison of the age of cadaveric donors, irrespective of their source, transplanted to local recipients during different time periods is given in Fig 3a. A breakdown of the age of recipients transplanted within the same time periods is also shown (Fig 3b).

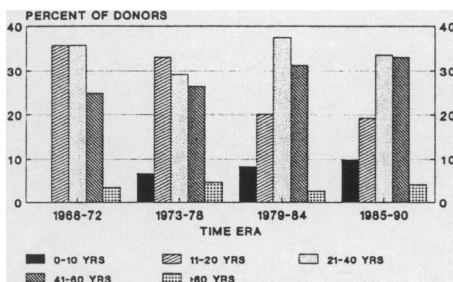


Fig 3a. Age of donor used for Belfast recipient 1968–1990.

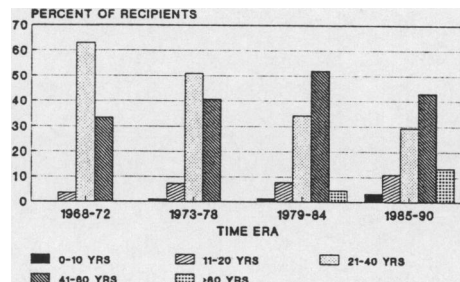


Fig 3b. Age of recipient transplanted 1968–1990.

The graft survival obtained with donors of different age groups and recipients of different age groups is shown in Fig 4. The oldest donor was aged 70 and the youngest was one year old. Whereas 65% of kidneys used from donors aged 21–40 were retrieved from local donors, 65% of kidneys from donors older than 60 were imported. A similar percentage of local and imported kidneys came from donors aged 1–10, 11–20 and 41–60. The oldest recipient was aged 71 and the youngest aged 2 years.

There was no difference in actuarial graft survival or patient survival for first cadaveric compared to retransplant second or third cadaveric transplants (Fig 5).

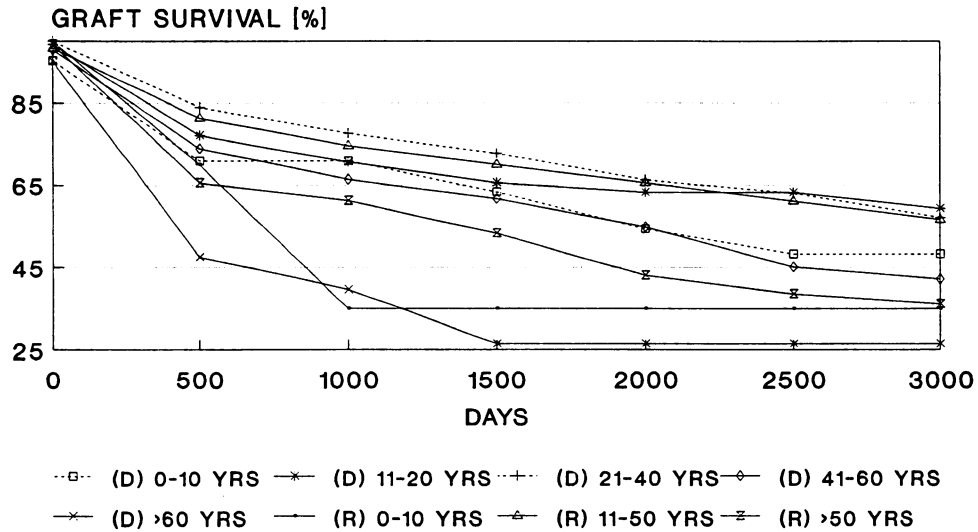


Fig 4. Age of donor (D) or age of recipient (R) and graft survival first cadaver.

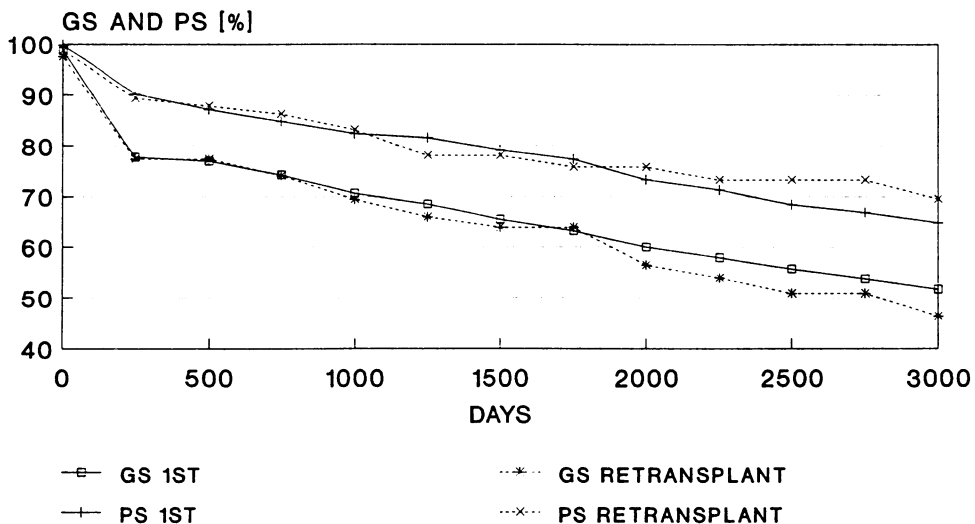


Fig 5. Actuarial graft (GS) and patient (PS) survival in 1st and retransplant cadaver transplants.

Of the 568 cadaveric donor kidneys 334 were local and 234 were imported. No difference was found in graft survival for first cadaveric transplants, whether the donor kidney was local or imported. Of the 568 cadaveric transplants, 345 were to males and 223 were to females. There was no difference in graft survival in first cadaveric transplants between males and females.

DISCUSSION

The number of local cadaveric donors increased from the commencement of renal transplantation in Belfast to a peak in 1986, whereas the number of imported kidneys received was relatively constant. Since 1987 the number of local donors has declined but the number of imported kidneys has increased. The reason for the latter increase is the adoption by more than 90% of transplant units in the United Kingdom of a scheme whereby if one kidney is beneficially matched (not more than one HLA – A or B antigen mismatched) to a recipient in another centre, it must be offered to that centre.

It is difficult to find a reason why the number of local kidneys procured has decreased since 1986. Analysis of the local hospitals supplying the donors shows the reliance placed on the Royal Victoria Hospital. There is a noticeable fluctuation in the number of donors obtained from most hospitals in each year, which may reflect staff movement. If all the local hospitals were able to offer each year the maximum number of donors they obtained in any one year, a total of 64 donors per annum would be obtained. This would easily fulfil the annual requirements for kidneys for local patients as the number of patients on the waiting list appears to have stabilised at 70 per annum. However, the maximum number of local donors ever achieved was 44 in 1986, (28·2 per million population).

The decrease in the number of donors has resulted in the number of donors per million population in Northern Ireland being below the national average in 1990, whereas in 1987 Northern Ireland was ranked second. During the same period the number of donors in the Republic of Ireland has increased from 11·9 per million in 1987 to 22·5 per million in 1990. This increase has coincided with the appointment of a Transplant Co-ordinator. The appointment of a similar Transplant Co-ordinator in Northern Ireland is imminent.

The age of donors used has changed during the time period under study. In the period 1968 – 72 no donor aged 10 years or less was used, whereas 10% of donors used between 1985 and 1990 were of that age. This reflects the larger number of children now transplanted at this unit. The percentage of donors aged 41 – 60 has gradually increased during the 22 year period with a corresponding reduction in the percentage of 11 – 20 year old donors. The graft survival in recipients receiving kidneys from donors aged 41 – 60 is comparable to the graft survival in recipients receiving kidneys from younger donors, and the use of these donors should be encouraged. Only recipients receiving kidneys from donors aged over 60 years have a poor graft survival. These kidneys were allocated on the basis of matching, and no evidence was found that they were given to recipients at greater risk. A higher percentage of kidneys from donors aged over 60 are imported and it may in future be more appropriate to refuse these donors unless they are a good match to the recipient.

The percentage of patients transplanted aged 21 – 40 has reduced during the time period analysed, with an increased percentage of recipients being

transplanted in the other age groups. Graft survival of patients aged over 50, or under 11, is not as good as patients aged 11 – 50. The latter age grouping was used as no difference in graft survival due to age was found within that group. The complications involved in transplanting young children (size of graft, post-operative management) would explain the poorer graft survival of that group. If a recipient dies with a functioning graft it is taken as graft failure which would explain the poorer graft survival in the older patients. Nevertheless it is still both feasible and reasonable to treat older patients by transplantation. The method of analysis used in this study is more stringent than that used in some studies which consider death with a functioning graft as lost to follow-up.

Overall there is a very good graft and patient survival obtained at this unit. Even when patients are re-transplanted good graft survival is obtained. Thus the only barrier to transplantation is lack of donors. We would hope that this study would encourage our colleagues to continue to offer donors to this unit. Of course much remains to be done in educating the public. A recent survey showed a 30% rate of refusal by relatives.³

The work reported here is the result of a team effort. The success of transplantation at Belfast City Hospital is due to the combined efforts of medical, surgical, nursing and laboratory staff. We therefore gratefully acknowledge the work of all staff, both past and present of the Renal Unit, Belfast City Hospital. We thank all colleagues who have contributed to the offer of donors during this period.

We thank Mr C Craig, Renal Unit, Belfast City Hospital for supplying additional information, Dr J Douglas for helpful suggestions, Miss K Mills for typing the manuscript and Professor Mary G McGeown for continuous advice and inspiration. We are indebted to the Northern Ireland Kidney Research Fund for generous support.

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Educational supervision of pre-registration house officers

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SUMMARY

An annual survey of the educational supervision of pre-registration house officers has been carried out since 1987 by the Northern Ireland Council for Postgraduate Medical Education and the Queen's University of Belfast. Educational supervision was considered by house officers to be unsatisfactory in 27% of medical posts and 52% of surgical posts. Regular teaching was provided at least weekly in 77% of the posts, but 50% of house officers wanted more experience of managing common medical emergencies. Over one-third felt administrative duties were excessive.

There is a strong argument that hospitals should designate within clinical units a consultant with responsibility for educational supervision of the pre-registration house officers. Creation of this hospital counterpart of the General Practitioner trainer — the educational supervisor — would enhance the educational value of the pre-registration year. This might avoid withdrawal of approval for training purposes from some pre-registration posts.

INTRODUCTION

In 1954 the General Medical Council made it a legal requirement that medical school graduates, in order to achieve full registration as medical practitioners, would have to undertake supervised hospital duties for one year. In recent years disenchantment with this apprenticeship-based hospital system has grown, with criticism of hours of work for the junior doctor, the specialised nature of some of the training posts and the failure of consultants to adopt an active approach to the training element of the pre-registration year. The Educational Committee of the General Medical Council produced guidelines dealing with basic medical training in 1986, which recommended that juniors should have a designated trainer and a close, more structured form of supervision. Nevertheless documented

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deficiencies in pre-registration training continue to surface, indicating that the GMC requirements have not been implemented.

A management efficiency study of the clinical workload of pre-registration house officers in Edinburgh Royal Infirmary resulted in the appointment of three extra pre-registration house officers.¹ In four Thames regions a postal questionnaire illustrated deficiencies in pre-registration training in terms of work pattern, and lack of education on important topics including resuscitation, pain control, and the breaking of 'bad news'.²

This study provides further information on the educational content of the pre-registration year based on the experiences of house officers in Northern Ireland.

METHOD

The Northern Ireland Council for Postgraduate Medical Education carries out an annual survey of the 160 pre-registration house officers in conjunction with the Faculty of Medicine of the Queen's University of Belfast. Each house officer is asked to complete a questionnaire concerning the regularity and relevance of teaching, the degree of supervision, the size of clinical and non-medical workload and their confidence in managing medical emergencies. The data from the responses is categorised by hospital, specialty and unit, and forms the basis of a report to the Pre-Registration House Officer Committee, chaired by the Dean of the Faculty of Medicine.

In 1990 a three year evaluation was carried out for the years 1987 – 1989. House officers were asked to assess the level of supervision which they had received using a scale of 1 – 4, where 1 was interpreted as being poor and 4 excellent. For the purposes of this paper the results are categorised into either medicine (including paediatrics) or surgery (including gynaecology). A chi-squared test was performed for the two groups to test the null hypothesis that there was no difference in the level of supervision between the two major specialty groups.

In a second question, house officers were asked if they thought that there were too many administrative duties involved in their posts. A chi-squared test was applied to test the hypothesis that there was no difference in the amount of non-medical work existing between the two major specialty groups.

To assess the relevance of teaching, the house officers were asked if they felt confident in dealing with common medical emergencies. In response to the question on confidence in dealing with common emergencies, 46% reported that they felt fully competent and 50% thought that although they felt competent, they would have preferred more experience. Over 4% claimed that they had been given insufficient opportunity to gain first-hand practical experience of such emergencies.

RESULTS

Over the three year period 57% of the pre-registration house officers responded to the questionnaire, covering all the hospitals and units in the Province. The Figure illustrates the replies to the level of supervision question, indicating a marked difference between the medical and surgical specialties, particularly at the extreme ends of the scale. Twenty-seven percent of house officers in medical

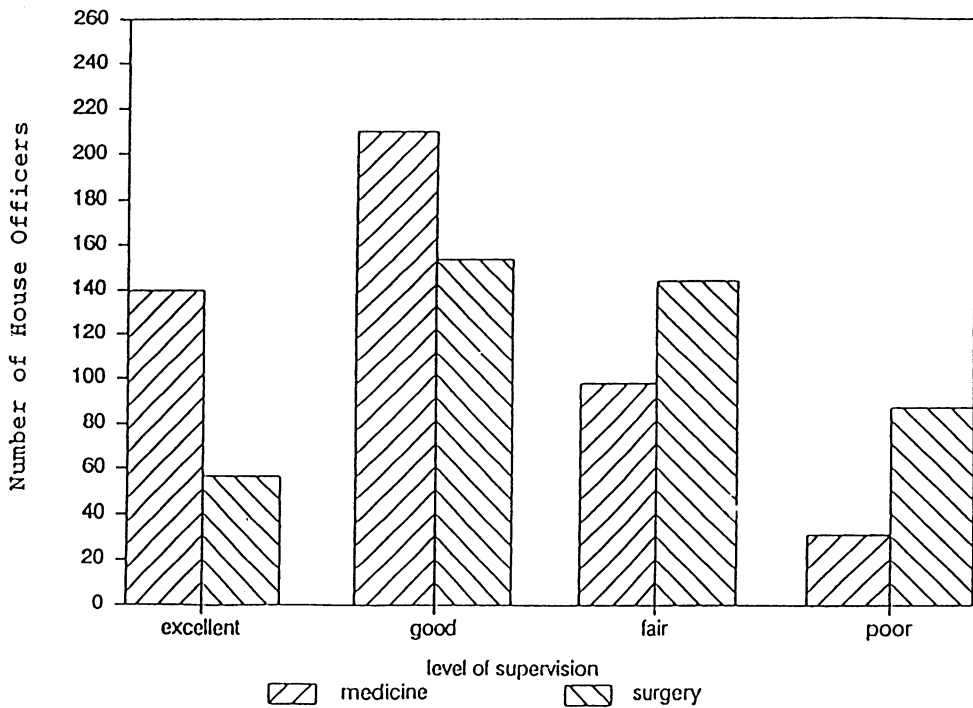


Figure. Level of supervision as perceived by pre-registration house officers 1987 – 1989.

posts and 52% of those in surgical posts thought that the level of supervision was only fair or poor. The difference between the two groups lie at the extreme ends of the supervision levels; surgical posts were much less likely to offer 'excellent' supervision and were nearly three times as likely to yield reports of poor supervision. One in five of all surgical posts reported supervision of a poor standard.

Over one-third of the house officers in both major specialties felt that the amount of administrative duties was excessive. There was no difference regarding non-medical duties (Table I).

TABLE I

Administrative duties carried out by house officers

| <i>Too many duties</i> | <i>Medicine</i> | <i>Surgery</i> |
|------------------------|-----------------|----------------|
| No | 310 (64%) | 307 (63%) |
| Yes | 172 (36%) | 176 (36%) |

The regularity of teaching, including teaching ward rounds, as perceived by junior house officers is shown in Table II. Seven percent had rarely received teaching and a further 7% were too busy with clinical or administrative duties to attend, even if teaching was available.

TABLE II
Teaching content of post

| Frequency of teaching | |
|------------------------|----------|
| More than once weekly | 88 (38%) |
| Weekly | 90 (39%) |
| Every 2 – 3 weeks | 14 (6%) |
| Monthly | 7 (3%) |
| Seldom/never | 16 (7%) |
| Lack of time to attend | 16 (7%) |

DISCUSSION

The results of this study indicate that house officers in Northern Ireland during 1987–1989 considered educational supervision unsatisfactory in 27% of medical posts and 52% of surgical posts, and that over one-third felt administrative duties were excessive in both posts. Half would have preferred more experience of common medical emergencies. These results and the studies from Edinburgh¹ and elsewhere suggest that there is scope for improving the pre-registration year experience with regard to the nature of work involved, the personnel management role of the consultant physician or surgeon and the content of formal training. The difference between medical and surgical posts might partly reflect the different nature of the work in the two specialties, surgeons being absent from wards for longer periods due to operating theatre commitments.

What action should be implemented by hospitals with responsibility for pre-registration house officers? We would suggest that hospital medical staff should designate within each clinical unit a consultant with responsibility for educational supervision of the unit's pre-registration and senior house officers. The pre-registration house officer joining a hospital unit should receive an initial briefing by the designated consultant which should encompass the clinical organisation of the unit and what is expected of the house officer. Specific advice should be given about resuscitation, discharge letters, practical procedures, autopsies and any other protocols special to the individual unit. The educational supervisor should conduct a monthly interview with pre-registration house officers lasting approximately 30 minutes during which the opinion of the house officer should be sought regarding adequacy of training and supervision, and feedback should be given on individual performance. The house officer should be encouraged to keep a career journal; this could prove helpful as it requires the house officer to document the training received in an individual post, his/her opinion of the training, and also the supervising consultant's opinion on the house officer's performance.

The house officer's career intentions should also be explored and basic guidance given by the educational supervisor. Parkhouse and Ellin³ have shown that career choice and change of choice is mainly determined by domestic circumstances, awareness of promotion prospects and self-evaluation of aptitude and ability.

Preliminary career advice to the pre-registration house officer should therefore aim to assist in assessment of his/her potential for a given career, and provide basic information about career prospects in the declared field of interest. Other appropriate advice might include information on local short-listing criteria for senior house officer posts and what to expect at interview for these posts. For detailed career advice and counselling the individual house officer should be referred to the specialty advisor or clinical tutor with access to relevant information including DHSS census data, regional strategic plans etc. Educational supervisors should ensure that formal training and/or hospital induction courses cover topics relevant to the tasks of the pre-registration house officer,^{1, 4, 5} should give adequate attention to communication skills,^{6, 7} and ensure that the house officer can recognise and initiate emergency treatment of common conditions. (Table III).

TABLE III

| |
|--|
| SUGGESTED ROLE OF THE EDUCATIONAL SUPERVISOR |
|--|

To provide for pre-registration house officers —

Initial briefing covering clinical organisation of the unit.

Specific advice about any protocols special to the unit (autopsies, practical procedures etc).

A monthly interview to assess adequacy of training being received and to give feedback on performance.

Encouragement to keep a career journal.

Basic career advice and information.

Information on senior house officer posts and guidance on interview techniques.

Formal training including management of common medical emergencies, pain relief and communication skills.

In conclusion, although some have expressed misgivings about the General Medical Council recommendations for basic medical training,⁸ the findings of this study strengthen the case for extending the trainer/trainee system from general practice to hospital medicine, accepting that this could not be on the one-to-one basis that pertains in general practice. The trainee (the pre-registration house officer) needs to have a model of good practice, adequate supervised clinical experience, time for study, regular feedback on performance and preliminary career guidance.⁹ Ways need to be explored of minimising the clerical/administrative component of the pre-registration house officer workload. The hospital counterpart of the general practitioner trainer — the educational supervisor — could fulfil a valuable role which would enhance the educational value of the pre-registration year. This might help the difficult problem of reconciling the competing needs of clinical workload and continuing medical education, thus avoiding the spectre of some pre-registration house officer posts having approval for training purposes withdrawn.

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Junior medical posts in the NHSSB: what the doctors think

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Accepted 10 March 1992.

SUMMARY

This survey of house officers in the Northern Health and Social Services Board in Northern Ireland demonstrated that they have complaints not just about the number of hours they work. Thirty-nine per cent noted poor standards of food and/or accommodation. Many complained about doing routine "non-medical" work and thought that their working conditions would be improved by nurses having more responsibility for managing intravenous medication and the employment of phlebotomists. Doctors expressed concerns about a lack of career counselling and availability of training in research methods in their posts.

INTRODUCTION

"Junior Doctors — the New Deal", a series of guidelines aimed at reducing the number of hours junior doctors are contracted to work, and also at improving their working conditions, was published in June 1991.¹ It is based on the proposals contained in the "Heads of Agreement" signed by representatives of consultants, the Royal Colleges, junior doctors and the National Health Service Management Executive.² In order to put these guidelines into effect a committee was established within the Northern Health and Social Services Board (NHSSB) to examine the working practices of junior doctors.

METHODS

In June 1991 all 91 senior house officers and 20 junior house officers employed by the NHSSB received a questionnaire about their job. As well as asking specific questions about the tasks undertaken by the house officer it sought their views about changes that might improve their working conditions and their subjective opinions about their job. The questionnaire was accompanied by an explanatory letter signed by the Director of Public Health and the local British Medical Association junior doctors' representative and a stamped addressed envelope for its return. Junior doctors at each of the eight hospitals involved had been informed

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by the BMA representative that these questionnaires were to be circulated. Ten days after dispatch a junior doctor from each hospital was contacted and asked to encourage any of his/her colleagues who had not completed and returned the questionnaire to do so.

RESULTS

Fifty-nine completed questionnaires were returned from 11 junior house officers and 48 senior house officers (a 53% response rate). All specialties in NHSSB hospitals were represented with 19 doctors working in medicine, 12 in surgery, 9 in psychiatry, 6 in obstetrics and gynaecology, four in paediatrics, three in cardiology, two in geriatrics, two in accident and emergency, one in anaesthetics and one in ear, nose and throat surgery.

On-call rotas varied with two doctors (3%) being on call 1 night in 2, 28 (48%) on call 1 in 3, 12 (20%) on call 1 in 4, four (7%) on call 1 in 5, four (7%) on call 1 in 6, six (10%) on call 1 in 7, one (1%) on call 1 in 12 and two (3%) on call 1 in 13 (one doctor did not complete this question). Sixteen respondents (27%) were working on rotas entailing more than 83 hours on duty each week.

When on-call between the hours of 11 pm and 7 am the doctors in this sample were telephoned an average of 3.6 times, got up from their bed to attend patients 3.8 times and had 4.9 hours sleep. Ten (17%) of the respondents regularly get up at night to give intravenous medication.

Twenty-three respondents (39%) noted that they were not satisfied with some aspect of their on-call accommodation. The specific complaints were predominantly about the standard of the living and washing facilities provided (14 doctors) and others included poor access to telephones (6 doctors), unsatisfactory security in rooms (3 doctors) and lack of married accommodation (2 doctors).

During the preceding 12 months, senior house officers in this sample had taken an average of 6.4 days of study leave. Six respondents (10%) had experienced difficulty in obtaining appropriate study leave. Sixteen doctors (27%) had time allocated for training or study in their weekly timetable.

Table I shows the responses given by doctors when asked if provision of the stated services on their ward would have an important beneficial impact on their working conditions (a score of 1 = very important, 4 = unimportant).

TABLE I
Degree of importance in improving working conditions

| | Very important | | | Unimportant |
|---------------------------------------|-------------------|----------|----------|-------------|
| <i>Service provided</i> | <i>1</i> | <i>2</i> | <i>3</i> | <i>4</i> |
| Employing phlebotomists | 26 | 13 | 7 | 7 |
| Employing filing clerks | 8 | 6 | 8 | 25 |
| Nurses giving intravenous medication | 24 | 19 | 9 | 5 |
| Clerks completing investigation forms | 20 | 6 | 22 | 5 |

(figures relate to numbers of doctors responding).

Table II shows the time junior doctors spent between the hours of 9 – 5 during a normal working day on selected tasks.

TABLE II

Time junior doctors spend during a typical working day on different activities (notionally 9 – 5)

| <i>Activity</i> | <i>Time spent (hours/minutes)</i> |
|---|---------------------------------------|
| Attending ward rounds | 2 hrs 17 mins |
| Clerking patients | 2 hrs 22 mins |
| Taking blood | 43 mins |
| Administration of intravenous drugs | 39 mins |
| Managing intravenous lines | 31 mins |
| Telephone calls and whether doctors thought they should deal with them: | |
| (a) doctor required | 45 mins |
| (b) doctor not needed | 30 mins |

Table III illustrates the replies junior doctors gave to the questions about aspects of their work.

TABLE III

Senior house officer and junior house officer responses

| <i>Statement</i> | <i>True</i> | <i>False</i> | <i>Don't know</i> |
|---|-------------|--------------|-------------------|
| My number of contracted hours is excessive | 36 | 20 | 3 |
| The training provided in my post is good | 35 | 16 | 8 |
| Too much of my time is spent doing tasks inappropriate for a doctor | 32 | 26 | 1 |
| I get sufficient training in medical research methods and statistics | 2 | 54 | 3 |
| I have access to a microcomputer at work | 12 | 43 | 4 |
| I worry about litigation due to medical negligence because of tiredness | 38 | 16 | 5 |

Table shows number of JHOs and SHOs responding to each statement.

DISCUSSION

This study shows that while generally uncritical of many aspects of their job, junior doctors do have some complaints about their posts. In Northern Ireland 39% of junior medical staff (at 31st March 1991) were contracted to work for more than 83 hours per week (Northern Ireland Department of Health — personal communication). This is the upper level specified in "Junior Doctors — the New Deal".¹ Over one quarter of respondents in this survey were contracted for more than this level. The survey had a moderate response rate but the respondents did

closely reflect both the range of specialties practised in the NHSSB and therefore also the different on-call rotas these posts entail. It is unlikely that doctors responding are only those particularly dissatisfied with their posts. There is no reason to believe that these findings are not representative of Northern Ireland where the intended reductions in hours of work of junior staff should reduce the strain caused by working long hours in busy and onerous jobs.

As well as reducing the numbers of contracted hours of junior staff the "New Deal" document aims to reduce the amount of time doctors spend doing tasks that could be more appropriately performed by others. As Table I shows, doctors in this sample spend a considerable time completing forms, giving intravenous medication and on telephone calls that do not, in their opinion, need a doctor's involvement. Changing who performs different tasks requires negotiation between the professions involved. Nurses are the group most likely to assume many of the tasks currently done by junior medical staff. This will require national as well as local agreement. Where it is agreed that nurses will assume some of the workload of junior doctors, additional training and accreditation will be required. The current system, where different employers allow nurses to assume different levels of responsibility, is clearly unsatisfactory and revision of national training curricula for nurses may be appropriate. As nurses assume what are currently "medical" tasks they of necessity have to shed duties of a "non-nursing" nature. Clearly such changes in working practice will have implications for other disciplines, will take some time to implement and will probably require the employment of extra staff.

Demonstration of published research is becoming increasingly important in the advancement of junior doctors' careers. Pursuing this research may require study leave and this is something about which there were few complaints from respondents. Nonetheless, it is pertinent to note that most doctors felt that training provided in research methods was deficient and indeed most do not have access to computing facilities at work. Employers are responsible for training their workforce and developing good personnel management practice. Thus training programmes for their junior staff in computing and research skills should be developed and the junior doctor's perceived lack of career guidance should also be addressed.

Employers are also responsible for providing appropriate on-call and canteen facilities and it appears that in this sample there are shortcomings in these areas. The changes in medical indemnity situation over the past two years also appear to be causing some anxiety for juniors and clear advice in this regard should be forthcoming.

This study shows that good employers should not concentrate solely on reducing the contracted hours of work but should address issues such as the content and extent of study leave, appropriateness of tasks performed at work and the living conditions of junior staff. Good employee relations in this field should foster a well-trained workforce with high morale and a pleasant workplace environment.

We acknowledge the help of our colleagues on the NHSSB committee examining the working conditions of junior doctors and thank those junior doctors who co-operated with our survey.

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Acoustic neurinoma surgery in Belfast 1986–1989

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SUMMARY

Forty-seven acoustic neurinoma tumours have been operated on in 46 patients in the years 1987–1989. This is a considerable increase over the prevalence in the preceding ten years. Twenty-six were classified as large tumours, 18 as medium and one was small. Surgical excision was complete in 16 and incomplete in 31 cases. Two patients died in the early postoperative period. Facial nerve function was preserved in 36 (80%) of cases; of these, 27 (60%) had good function and nine (20%) fair function. Useful hearing was preserved in only two patients. The overall complication rate has been low and often of a transitory nature.

INTRODUCTION

Acoustic neurinoma presents a challenge both in diagnosis and treatment. The diagnostic challenge is to detect early and therefore smaller tumours, whilst the surgical challenge is to remove what is a benign tumour with as little morbidity as possible.

Reports of these tumours date back to 1830, when Sir Charles Bell¹ published the first clinical and autopsy case of cerebellopontine angle neurinoma. The first successful operation on an acoustic neurinoma was performed by Ballance² in 1894. Cushing³ developed an interest in these tumours in the early twentieth century and by performing partial removal was able to reduce to 20% the previous operative mortality of 80%. His technique was refined by Dandy,⁴ one of his students, who removed the capsule after enucleation of the tumour interior with a mortality rate of 22%, although all his patients were deaf and had permanent total facial paralysis.

Further advances were made in the 1960s when House⁵ developed the trans-labyrinthine approach with microsurgical removal. He achieved a 5% mortality

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rate, and preserved facial nerve function in almost 95% following total tumour removal. Recent advances have been mainly in the field of diagnostic techniques, allowing smaller tumours to be identified. These twin improvements, in surgical technique and in diagnosis, have led to a further reduction in mortality and overall morbidity. Facial nerve function is commonly preserved and in selected patients preservation of hearing may now be a realistic goal. In the ten years between 1976 and 1986 40 acoustic neurinomas were removed surgically in the Royal Victoria Hospital,⁶ an average of four per year. In the last three years 47 tumours have been removed, an increase to over 15 per year. The reasons for this increase may include heightened awareness among the medical community, the more widespread provision of CT scanners and increased expectations by patients.

The results of the surgery in the last three years are reported.

PATIENTS AND METHODS

Forty-six patients were operated on for 47 tumours between September 1986 and February 1990. Three patients had bilateral tumours: in one the first side had been dealt with before September 1986 and in a second only one side has been operated on so far. There were 18 males and 28 females. Ages ranged from 19 to 73 years with an average age of 48 years.

Symptoms on presentation are shown in Table I. All patients complained of deafness. The next most common symptoms were unsteadiness and tinnitus. Rotatory vertigo occurred in eight patients. Other symptoms included headache, facial numbness or pain, diplopia, blurring of vision and nausea and vomiting. The first symptom was deafness alone in 33 patients, unsteadiness in six, tinnitus in three, and both deafness and tinnitus together in another four.

TABLE I
Symptoms on presentation in 46 patients

| | <i>n</i> | (%) |
|----------------------|----------|-----|
| Deafness | 46 | 100 |
| Unsteady gait | 24 | 52 |
| Tinnitus | 23 | 50 |
| Rotatory vertigo | 8 | 17 |
| Headache | 7 | 15 |
| Facial pain/numbness | 4 | 8 |
| Diplopia | 2 | 2 |
| Blurred vision | 2 | 4 |
| Vomiting | 2 | 4 |
| Nausea in morning | 1 | 2 |

Physical signs on presentation in addition to deafness are shown in Table II. Twenty-one patients were ataxic on walking with a positive Romberg's test, and a further four showed arm ataxia. Nystagmus was present in 15, decreased corneal reflexes in nine, and decreased facial sensation in seven patients. Other signs

included papilloedema (4), facial weakness (3), absent or diminished gag reflex (3), and vocal cord paresis or hypoglossal paralysis. Eighteen patients showed no physical signs whatsoever other than deafness.

TABLE II
Physical signs on presentation in addition to deafness

| | <i>n</i> | <i>(%)</i> |
|------------------------------|----------|------------|
| Ataxia — gait | 21 | 45 |
| — arm | 4 | 8 |
| Nystagmus | 15 | 32 |
| Decreased corneal reflex | 9 | 19 |
| Decreased facial sensation | 7 | 15 |
| Papilloedema | 4 | 8 |
| Facial weakness | 3 | 6 |
| Absent/diminished gag reflex | 3 | 6 |
| Vocal cord paresis | 1 | 2 |
| Hypoglossal paralysis | 1 | 2 |
| No other signs | 18 | 39 |

Three clinical stages are recognised. In stage I only the eighth nerve is involved with deafness and tinnitus being the only symptoms. Stage II is marked by the presence of other neurological signs or symptoms usually cerebellar ataxia and trigeminal nerve or nucleus involvement. Stage III tumours cause raised intracranial pressure and usually papilloedema.

In this series there were 18 stage I, 25 stage II and 4 stage III tumours on presentation. One tumour was small (less than 1 cm), 20 were medium sized tumours (1 – 2.5 cm) and 26 were large (greater than 2.5 cm diameter). The patient with the small tumour had previously had surgery on the opposite side for an acoustic neurinoma, with nystagmus preoperatively and was therefore classified as stage II.

All reported tumours were removed by a combined neurosurgical and otological team using the suboccipital approach.

RESULTS

There were two postoperative deaths, which occurred in patients who had large tumours. A 58-year-old female died a few hours postoperatively from a brain stem infarct, and a 63-year-old male developed chest problems in the early postoperative period and eventually died of respiratory failure.

Sixteen tumours were completely removed and 31 incompletely removed (Table III). Most complete removals were in medium sized tumours. Of the incomplete removals 11 were subtotal, 17 were partial capsular and three were intracapsular. Reasons for incomplete removal included age or debility, fluctuating vital signs, swelling of the brain or bleeding at surgery, attempts to preserve auditory or facial nerve function and tumour adherence to the brain stem.

TABLE III
Tumour size and stage at presentation and surgical excision

| | Tumour stage | | | Excision | |
|--------|--------------|----|-----|----------|------------|
| | I | II | III | complete | incomplete |
| Small | — | 1 | — | — | 1 |
| Medium | 11 | 9 | — | 13 | 7 |
| Large | 7 | 15 | 4 | 3 | 23 |

Facial nerve function

Postoperative facial nerve function in 45 patients is shown in Table IV. Function was graded as absent, fair when weakness and asymmetry were marked and good when normal or near normal. The two patients who died in the early post-operative period are not included. Facial nerve function was preserved in 36 (80%) of patients. Of these function was good in 27 (60%) and fair in 9 (20%). Nine patients had no residual function (20%). Two of the patients with fair results are expected to improve further. In the 15 patients where tumour excision was complete, facial nerve function was preserved in 14, of whom 11 had a good result. Of the nine patients with no postoperative facial nerve function eight had large tumours, seven of which were incompletely removed.

TABLE IV
Facial nerve function postoperatively. (Two cases who died have been excluded)

| | Excision | Good | Fair | None | Total |
|--------|------------|------|------|------|-------|
| Small | Incomplete | 1 | — | — | 1 |
| Medium | Complete | 10 | 3 | — | 13 |
| | Incomplete | 4 | 2 | 1 | 7 |
| Large | Complete | 1 | — | 1 | 2 |
| | Incomplete | 11 | 4 | 7 | 22 |
| | Total | 27 | 9 | 9 | 45 |

Hearing

If a speech discrimination score of 50% is considered to be essential for useful hearing, then 14 patients has useful hearing preoperatively. This includes one ear with a score of less than 50% in a patient who had bilateral tumours and total loss of hearing in the other ear, his only hearing ear therefore was considered useful. Hearing was preserved in this patient and in only one other. All other patients had no useful hearing postoperatively. Two patients were referred directly to neurosurgery from medical and neurological clinics and because of the large size of the tumours audiometry was not performed. These two patients have been excluded from consideration of hearing.

Complications

The overall complication rate has been low. Twenty-one patients had no complications whatsoever. There were no cases of wound infection or meningitis. Fifteen patients showed ataxia to some degree which has completely settled in eight so far, improved in a further five but continues to present a problem in two cases who need an aid to walk. Dysphagia was present in five patients initially, and has resolved in two of them. Four of the patients with dysphagia, plus one other patient, had vocal cord paresis postoperatively, two of whom have settled. Other complications included headache and facial pain in four cases and single cases of dysarthria or blurred vision. Cerebrospinal fluid otorrhoea occurred in one patient due to incomplete waxing of mastoid air cells, which required further surgery.

To date two patients who had partial capsular removals have shown evidence of regrowth on CT scanning and will probably require further surgery.

DISCUSSION

The mortality rate for acoustic neurinoma surgery via the suboccipital route remains low, and was 4.3% in this series. Facial nerve function was preserved in 80% of the 45 patients, and in 14 of the 15 patients where tumour excision was complete. The likelihood of hearing preservation is low. Postoperative morbidity has been low and often of a transient nature. It is well recognised that surgery on smaller tumours results in fewer complications. However, although Brainstem Evoked Response Audiometry can indicate, and CT scanning can detect small tumours, it is interesting to note that only one tumour was picked up at this stage. Whilst this compares favourably with the previous 10 year period where 78% of the tumours were large at presentation,⁶ it would decrease morbidity if these tumours could be detected earlier. Hearing preservation is only likely with small tumours. The tumour size distribution here is similar to that reported in other series. Cohn et al⁷ noted no change in size distribution from 1979 to 1986, whilst Glasscock⁸ noted a decrease in large and an increase in medium tumours but no change in the number of small tumours presenting between 1975 and 1985. Similarly Thomsen⁹ had only one small tumour in 300 cases, the majority being large tumours greater than 2.5 cm.

An advantage of the translabyrinthine approach is that it avoids damage to the cerebellum and therefore produces less ataxia postoperatively. Thomsen⁹ noted a 7% incidence of ataxia using this approach. Ataxia was a problem in only two of our cases both of whom were elderly. Cerebrospinal fluid leakage is a relatively common complication in other series, especially with the translabyrinthine approach, occurring in up to 20% of cases.¹⁰⁻¹³ This occurred in only one patient in this series (2.2%). Facial nerve function was preserved in 80% (60% with a good result) which is similar to other series.^{9, 14, 15} Hardy et al¹⁴ pointed out that the presence of preoperative facial weakness and a tumour size greater than 2.5 cm diameter are poor prognostic signs for facial nerve recovery.

It would seem that despite the surgical advances, increased diagnostic ability and more widespread provision of this technology, little apparent effect on early diagnosis has occurred. Perhaps education of the medical community to refer earlier the patients complaining of asymmetrical hearing loss is the way forward.

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Surgical palliation of proximal malignant biliary obstruction

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INTRODUCTION

Proximal malignant biliary obstruction can be cured only by surgery. Such treatment may involve hepatic resection and is associated with significant morbidity and mortality. Unfortunately up to 90% of patients with this problem are either unfit or unsuitable for a curative procedure.¹ Conventional surgical decompression for these patients with obstructive jaundice via a cholecystojejunostomy, choledochojejunostomy or hepaticojejunostomy below the confluence of the right and left hepatic ducts may be impossible because of extension of the tumour.² Normally, these patients receive a stent using a radiological percutaneous trans-hepatic cholangiography procedure (PTC) or an endoscopic route at ERCP. In a small proportion of cases stenting may not be possible by either route. We believe that an attempt at palliation should be made, as otherwise these patients will die within a few months with a very poor quality of life.³ Such palliation can be achieved by a bilioenteric anastomosis either to the left main hepatic duct or to the segment III duct, draining the left lobe of the liver, lateral to the ligamentum teres. These techniques were first described by Hepp and Couinaud in 1956,⁴ but initially were rarely performed outside France. There has been more interest since 1984 following a report by Blumgart and Kelley on the use of these techniques in benign and malignant disease.⁵ We describe these procedures and their successful use for palliation in three patients with proximal malignant biliary obstruction.

OPERATIVE TECHNIQUE

The key to understanding these two approaches is a knowledge of the ductal anatomy of the liver (Fig 1). The anatomy of the left hepatic duct and its branches is usually constant, unlike the frequent anomalies found around the common hepatic and cystic ducts.⁵ The left hepatic duct always has an extrahepatic course along with its accompanying artery and vein beneath the quadrate lobe (segment IV). The segment III duct is found with an accompanying branch of the portal vein within the liver at the base of the ligamentum teres.⁶

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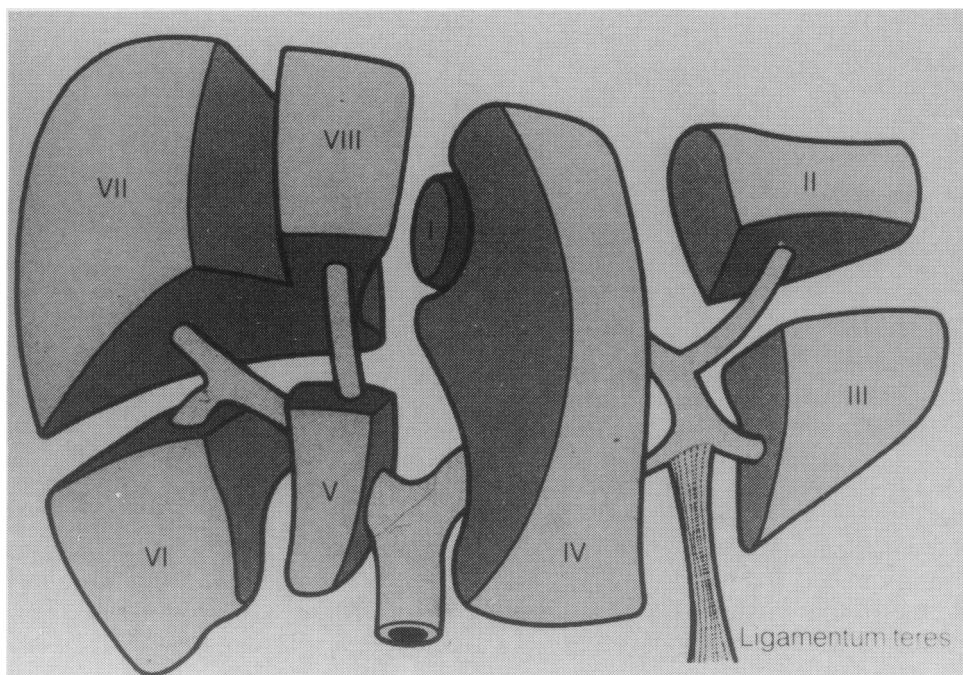


Fig 1. Segmental anatomy of the liver.

At laparotomy, through a "rooftop" incision the liver is elevated to display its undersurface. A decision as to which bypass should be performed — left main duct or segment III — is then made dependent upon the extent of the tumour. For the left main duct approach, this duct and the confluence are exposed at the base of the quadrate lobe. The duct becomes visible as the hilar plate is lowered by dissection around the structures of the left portal triad. A Roux-en-Y loop of jejunum and the left hepatic duct is incised longitudinally and a side-to-side anastomosis is performed.

The ligamentum teres approach to the segment III duct is used when there are difficulties in exposing the left main duct. This may be due to tumour mass, adhesions, bleeding or a large overhanging quadrate lobe. It allows anastomosis at a reasonable distance from the malignant lesion, lessening the risk of further biliary obstruction. Downward traction on the ligamentum gives access to the segment III duct. Needle aspiration or intra-operative ultrasound is used to assist with its identification. The duct is then incised and the anastomosis performed as above. A tubal splint may be placed across the anastomosis.

Case 1

A 77-year-old man was admitted with a five day history of painless, obstructive jaundice after three months of anorexia and weight loss. He was cachexic and had 4 cm hepatomegaly. His liver function tests confirmed an obstructive pattern with a bilirubin of 194 $\mu\text{mol/l}$ (3 – 18). An ultrasound scan demonstrated dilated intra-hepatic ducts and a PTC revealed complete obstruction of the extrahepatic biliary

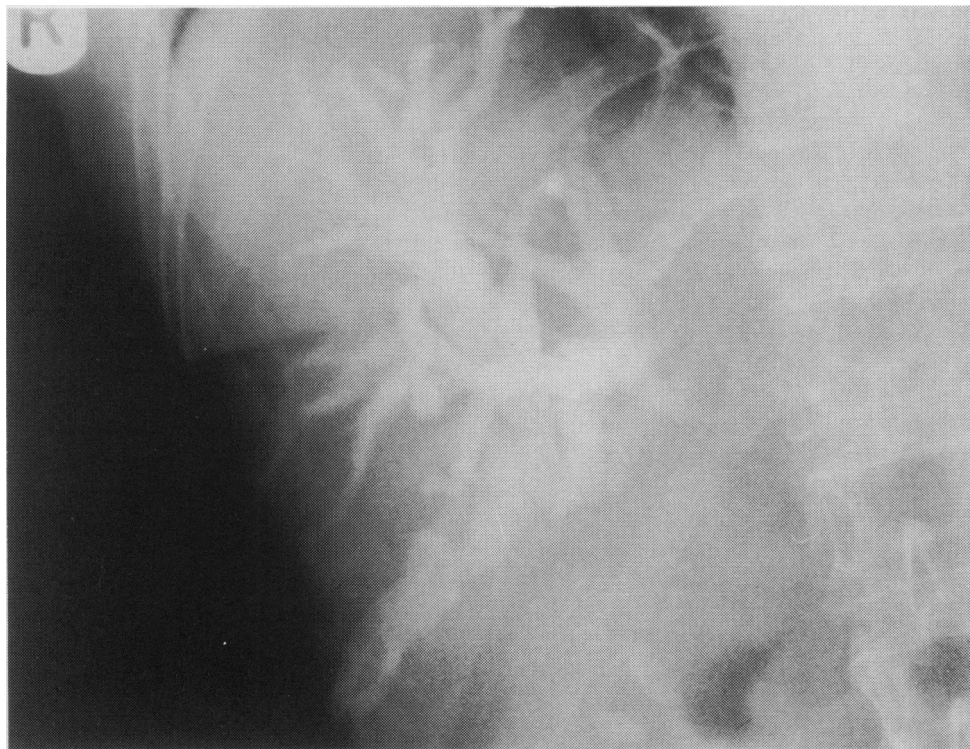


Fig 2. Pre-operative proximal biliary obstruction at PTC.

tree just below the porta hepatis (see Fig 2). An abdominal CT scan showed normal liver parenchyma, dilated intrahepatic ducts and a mass in the pancreatic head. Stenting from above or below was impossible. After treatment with intravenous fluids and vitamin K, he underwent laparotomy. There was a large tumour in the pancreatic head with gross lymphadenopathy encasing the common bile duct up to the porta hepatis. A segment III hepaticojejunostomy was performed using a 70 cm Roux-en-Y loop of jejunum. Postoperatively he received intravenous antibiotics and total parenteral nutrition. A postoperative cholangiogram via a trans-anastomotic tube (which had been brought out percutaneously) revealed decompression and drainage of the left hepatic ductal system. A simplified illustration demonstrates the drainage route (Fig 3). Postoperatively he made a gradual recovery and his jaundice settled. He had a good quality of life until he suddenly deteriorated and died eight weeks later.

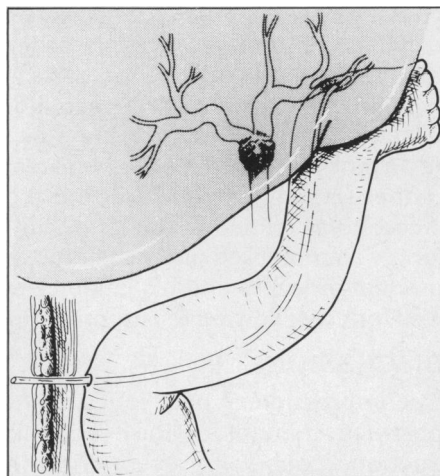


Fig 3. Simplified illustration demonstrating the drainage route.

Case 2

A 64-year-old man was admitted with a two year history of episodes of obstructive jaundice. He had had a cholecystectomy eleven years previously and a heavy alcohol intake until five years beforehand. An ultrasound scan during his first episode of jaundice revealed a 5 cm mass in the right lobe of his liver with dilated intrahepatic ducts. A PTC was normal as was his serum alphafetoprotein. Fine needle aspiration of the lesion under ultrasound control revealed atypical hepatocytes but no definite malignancy. On this admission he had painless obstructive jaundice, and an ultrasound scan and CT scan showed a 7 cm lesion in the right lobe with dilated intrahepatic ducts and a mass lesion at the porta hepatis. His serum alphafetoprotein was elevated at 288 ku/l. ERCP demonstrated a filling defect at the porta hepatis. He was felt to have multifocal hepatocellular carcinoma but stenting was impossible. Subsequently his condition deteriorated and he became increasingly jaundiced and clinically septic. He was referred for surgical assessment four weeks after becoming jaundiced. At this stage his liver function tests were grossly obstructive with a bilirubin of 164 $\mu\text{mol/l}$ and his coagulation screen was markedly deranged. He was treated with intravenous fluids, broad-spectrum antibiotics, vitamin K and "renal" doses of dopamine. At emergency laparotomy a large mass in the right lobe of his liver and a smaller mass posterior to the porta hepatis were identified. A segment III hepaticojejunostomy was performed for palliation. The lesion was not biopsied because of his bleeding diathesis. The first 48 hours postoperatively were very stormy with severe renal impairment and septicaemia, but he gradually recovered. His jaundice settled and he was discharged on the eighteenth postoperative day. For the next seven months he was well, and walked up to five miles daily. He then began to lose weight and developed epigastric pain requiring oral morphine sulphate. Eight months following his operation, he suddenly deteriorated and died.

Case 3

A 71-year-old man was admitted with a three month history of episodes of obstructive jaundice. Two years previously he had been found to have a cholangio-carcinoma and had a cholecystectomy, resection of the affected area of his common bile duct and a hepaticodochojejunostomy performed. On this admission his liver function tests again showed an obstructive pattern with a bilirubin of 64 $\mu\text{mol/l}$. Ultrasound and CT scans suggested intrahepatic duct dilatation and PTC demonstrated obstruction of his hepaticojejunostomy due to recurrent tumour. This was unsuitable for either dilatation or stenting. A laparotomy was performed revealing recurrent tumour around the anastomotic site. Access was gained to the left main hepatic duct. The blind end of the old Roux-loop was mobilised and anastomosed to the left duct. Postoperatively he made a gradual recovery and his jaundice settled. He went home after 19 days and was well for the following ten months. His jaundice then recurred and he died at home.

DISCUSSION

Decompression of both lobes of the liver should be the aim in surgical relief of obstructive jaundice. However, unilobar decompression as obtained with the left duct approach will prevent cholangitis and resolve cholestasis in over 95% of cases.⁷ The surgical approaches described are also valuable in the treatment of benign disease such as recurrent biliary strictures and sclerosing cholangitis.⁵

The second case illustrates that investigation of jaundice should be viewed as an emergency. Biochemical confirmation, ultrasound scan and cholangiography (via ERCP or PTC) should be performed within 48–72 hours. Undue delay will increase both the morbidity and mortality rates following surgical or other intervention. As Professor Michael Trede states “The cause of obstructive jaundice should be diagnosed before the sun sets twice’ (personal communication).

Surgery provides the only chance of long-term cure in proximal malignant biliary obstruction. Potentially curative resection is only possible in about 10% of patients with hilar neoplasms.^{1,3} This may involve some form of hepatic lobectomy with restoration of biliary continuity by single or bilateral hepaticojejunostomy. The 30 day hospital mortality rates are 10–20% in experienced hands.³ However, there are few long-term survivors and the mean survival time is 18–24 months.¹

There are a variety of means of providing palliation in the 80–90% of patients who have incurable disease. Insertion of tubing across the biliary stricture at laparotomy has been widely performed. Straight, Y or U-tubes have all been used. Despite the varieties of shape and material used in these tubes, they often rapidly become blocked by tumour growth and sepsis is very common. These procedures give an average survival of nine months but the morbidity rate is 26% and the 30 day mortality is 35%.^{1,8}

Percutaneous transhepatic biliary drainage involves radiological insertion of a drainage catheter into the biliary tree non-operatively. The results are very poor with morbidity rates of 78%, 30 day mortality of 56% and a mean survival of only 3 months, no better than if left untreated.⁹ The obstruction may be relieved by insertion of a stent at PTC. This is successful in approximately 70% of patients and will prolong survival to around 8 months. The 30 day mortality is 30% and the morbidity rate is 13%.¹⁰ An endoprosthesis may also be inserted endoscopically at ERCP. This route is more popular worldwide as the incidence of haemorrhage and biliary leaks are less than at PTC. In proximal lesions the success rate is 89% in experienced hands with a morbidity rate of 17%, 30 day mortality of 22% and mean survival of eight months.⁷ Internal biliary drainage has been combined with local irradiation to the tumour using an iridium-192 wire. This has been reported to lengthen survival to between 12 and 18 months.¹¹

Surgical palliation using the above-described approaches to the left ductal system gives a mean survival of eight months.^{3,5} Cholangitis is less common than in treatment with endoprosthesis. The 30 day mortality rate is 30%. We believe that palliative hepaticojejunostomy should be used for patients with proximal malignant biliary obstruction in whom stenting is not possible. These patients will die within 3 months if not treated. Even in elderly patients, palliation is worthwhile because of improved survival and an enhanced quality of life by relief of the pruritis and nausea of obstructive jaundice.

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Shrinkage of uterine fibroids by preoperative LHRH analogue injection

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SUMMARY

Six patients with large uterine fibroids were given a single subcutaneous implant of an LHRH analogue (goserelin 3.5 mg) prior to elective hysterectomy. Overall fibroid volume decreased by 30–47% within six weeks of implantation. All patients reported improvement in their symptoms of pressure and pain, and were rendered amenorrhoeic prior to surgery.

INTRODUCTION

Goserelin, a high potency luteinizing hormone-releasing hormone (LHRH) analogue is available as a slow release subcutaneous implant (Zoladex ICI), which has been shown to produce consistent, reversible suppression of the pituitary-ovarian axis.¹ The release of follicle stimulating hormone and luteinizing hormone from the pituitary gland is under the control of pulsatile release of LHRH from the hypothalamus. Continuous administration of LHRH analogues causes pituitary gonadotrophes to become desensitised, which induces a state of hypogonadotrophic hypogonadism, a process known as pituitary down-regulation. As a result of this oestrogen output from the ovary decreases and serum oestrogen levels fall to values in the postmenopausal range.

LHRH analogues are increasingly advocated in the management of common oestrogen-dependent gynaecological conditions, notably uterine fibroids,^{2,3} endometriosis^{4,5} and menorrhagia,⁶ and may also be effective in the management of metastatic breast cancer.⁷ Recent work^{2,3} has shown the effectiveness of both buserelin and goserelin in shrinking uterine fibroids to about 50% of their original size (assessed by ultrasonic measurement), but both these studies showed regrowth of the fibroids to their former size within months of stopping therapy. This work prompted Shaw to suggest that LHRH analogues should be used as an adjunct to the surgical management of fibroids.²

The aim of the present study was to measure the effect of a single implant of goserelin on large fibroid masses prior to elective surgery, to determine its effect on the patients' symptoms and to record side effects.

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PATIENTS AND METHODS

Six patients, aged 42 – 50 years, parity 0 – 2, were recruited to the study from the gynaecological clinic of the Ulster Hospital. All had menorrhagia, dysmenorrhoea and generalised lower abdominal pain and pressure.

On clinical examination each patient had a firm abdominal mass equivalent in size to a 16 – 20 week pregnancy. Clinically these were fibroid masses and ultrasound examination supported this diagnosis. The patients were offered abdominal total hysterectomy and bilateral salpingo-oophorectomy with postoperative hormone replacement therapy as the treatment of choice. Each was given an admission date within 4 – 8 weeks.

After informed consent for the present study was obtained, each patient received a 3.5 mg goserelin implant into the anterior abdominal wall after local infiltration with 1% lignocaine. They were then reviewed weekly for ultrasonic assessment of the maximum length (a), depth (b) and width (c) of the tumour. Measurements (a) and (b) were taken in the longitudinal plane and (c) in the transverse plane. Ultrasound scanning was performed on an Ultramark 4 machine by one observer. After obtaining a picture of what was regarded as the maximum size of the mass, the ultrasonographer freeze-framed the picture and turned the screen away. The second observer performed the measurements and obtained hardprint copies of these. Fibroid volume was determined by the formula of an ellipsoid according to Shawker's method.⁸ Weekly measurements of serum FSH, LH and 17 β -oestradiol values were obtained, symptoms were reviewed and any side effects noted. Following operation the fibroid volume was measured immediately by fluid displacement in a measuring jug.

RESULTS

Overall fibroid volume estimated by ultrasound showed a 30 – 46% reduction in the study period (Table I), the shrinkage being obvious within two weeks in all cases and maximum by four weeks (Fig 1). This reduction was evident on palpation. The shrinkage coincided with the postmenopausal levels of oestradiol (< 50 pmol/l) achieved with the treatment. These levels were reached in all cases by four weeks from implantation (Fig 2). These coincided with hypogonadotrophic levels of FSH and LH (< 2 IU/l).

TABLE I

Ultrasonic estimates of original and immediate preoperative fibroid volume and percentage reduction in size

| <i>Patient</i> | <i>Original fibroid volume (ml)</i> | <i>Preoperative fibroid volume (ml)</i> | <i>Shrinkage</i> |
|----------------|---|---|------------------|
| OA | 1033 | 557 | 46% |
| JC | 951 | 581 | 39% |
| ED | 815 | 453 | 44% |
| MM | 660 | 353 | 46% |
| FS | 600 | 420 | 30% |
| JH | 542 | 350 | 33% |

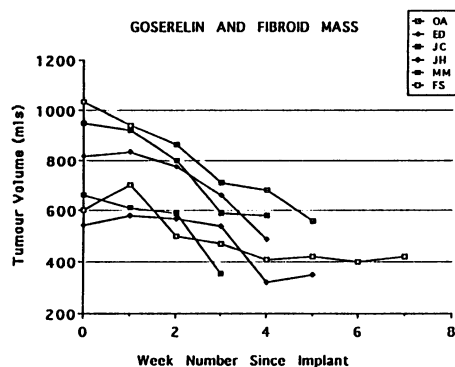


Fig 1. Ultrasonic estimate of fibroid volume in the weeks following goserelin implant up to the day before surgery.

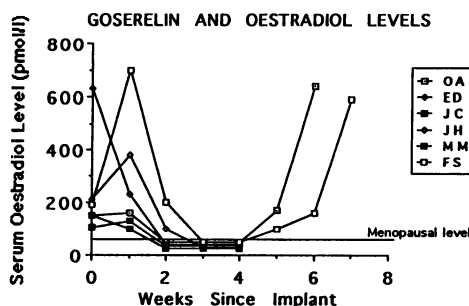


Fig 2. Serum oestrogen levels after goserelin implantation.

The estimated volume by scan prior to surgery corresponded to within 5–14% of the actual volume of the surgical specimen (minus ovaries and tubes) as determined by fluid displacement (Table II). The ultrasound volumes were all under-estimated.

TABLE II

Comparison of ultrasonic estimation of immediate preoperative fibroid volume and actual volume of surgical specimen measured by fluid displacement

| Patient | Preoperative estimated fibroid volume (ml) | Actual fibroid volume (ml) | Discrepancy |
|---------|---|----------------------------------|-------------|
| OA | 558 | 636 | – 14% |
| JC | 581 | 648 | – 11% |
| ED | 453 | 475 | – 5% |
| MM | 353 | 385 | – 9% |
| FS | 420 | 441 | – 5% |
| JH | 350 | 388 | – 10% |

All patients reported improvement in their symptoms of pain and pressure, evident between the second and third week of treatment. One patients had slight vaginal staining during the treatment, but menstruation was delayed in all patients prior to hysterectomy. One patient was just commencing menstruation at the time of hysterectomy, 61 days after her implant. The only side effects noted were hot flushes (four patients) which coincided with the low oestradiol levels.

Despite shrinkage of the fibroid mass in all patients, a debulking procedure (myomectomy) was required in two patients in order to gain access to the uterine pedicles. Postoperatively the only complication was one self-limiting episode of paralytic ileus. Histopathology confirmed the diagnosis in all cases: in two endometriosis was found in the ovaries.

DISCUSSION

We report major shrinkage of large uterine fibroids following a single subcutaneous implant of goserelin. Some studies have suggested that medical management of fibroids with a LHRH analogue is an alternative to surgery^{9, 10} but others^{2, 3} have shown complete regrowth of fibroids within months of cessation of this therapy. The long-term use of LHRH analogue with subsequent hypooestrogenism has not been studied but this therapy does induce a premature menopause with the associated problems of climacteric symptoms and, in the long-term, potential atherosclerotic and osteoporotic changes. We would agree with Shaw² who suggested that the LHRH analogue should be used as an adjunct to surgical management. The implant could be repeated at four-weekly intervals if surgery was delayed. Surgery also allows full histological assessment with a firm diagnosis and rules out malignancy.

Important aspects of this combined chemosurgical approach include full patient compliance and acceptability, improvement in symptoms and absence of serious side effects. Goserelin is expensive (£114 per implant) but this must be weighed against other positive factors and potential savings. The cessation of menses over the treatment period should allow anaemia to be corrected by simple haematinics and might save the need for blood transfusion in some patients with severe menorrhagia.⁶ Surgery could also be made easier in several ways. Matta *et al* have shown by Doppler blood flow studies that the blood supply to fibroids and the uterus is decreased after a LHRH analogue.¹¹ In our series, a debulking procedure (myomectomy) still had to be performed in two cases but blood loss was not a problem. Access to the uterine pedicles should be easier when fibroids are smaller and the surgical incision might be modified from a longitudinal midline incision to a transverse lower abdominal incision.

One important factor in the selection of patients is to exclude the possibility of an ovarian tumour. Fibroids have a characteristic ultrasonic appearance and shrinkage is obvious both clinically and ultrasonically within three weeks. It is unlikely, therefore, that a misdiagnosis would be made, but with this chemosurgical approach, surgery would be performed within a few months and the time factor involved would make little difference in prognosis.

The fibroids appeared to demarcate prior to shrinkage and one huge mass appeared to separate into the various fibroids making up the mass. It has been shown that fibroids have increased cytoplasmic oestrogen receptors compared to adjacent myometrium¹² but our impression on serial scanning was that the myometrium shrank around the fibroid first, which allowed the fibroids to demarcate.

Concern has been expressed regarding the potential side effects of the LHRH agonists. The only side effects recorded by our patients were tolerable hot flushes and these were offset by relief of pain and pressure. In theory, long-term usage of a LHRH analogue could be associated with bone loss or coronary artery disease. Our patients did not show any change in serum calcium or alkaline phosphatase during the short period of this study. Van Leusden and Dogterom did not find any bone loss following six months of continuous treatment with a LHRH analogue.¹³ We contend that this chemosurgical approach to large fibroids is safe, effective and beneficial to the patient. One implant produces significant benefits and this

schedule could be incorporated into standard clinical practice. Close follow-up of patients preoperatively seems unnecessary as there are few side effects.

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Communication of discharge information for elderly patients in hospital

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SUMMARY

The delivery of the discharge note to the general practitioners following discharge of elderly patients from a geriatric medical unit was studied over a two-month period. Handwritten discharge notes were received by the general practitioners in 75% of cases, and the delay was reduced to a median of two days by the use of a pre-printed envelope. Postal communications were also received by 89% of general practitioners after a median delay of two days. A dual system of hand-delivery and postal delivery would ensure faster and more complete receipt of information.

INTRODUCTION

Accurate communication of information from hospital is important, particularly for the elderly who may require considerable support from medical and other professional services following discharge. We have audited our own established patterns of communication of this discharge information before attempting improvements. The present discharge note is handwritten by the house physician and is intended to be delivered by hand by the patient or a relative to the general practitioner. The note includes details of medical treatment received, and lists discharge medication. Its early receipt by the general practitioner is particularly important if a prescription is to be issued to follow the three-day supply prescribed on discharge by the hospital. This survey investigated the current system of communication of information, the result of introducing envelopes with pre-printed advice regarding hand delivery and finally the value of postal communication.

METHOD

In phase I we studied 78 consecutive patients discharged over a two-month period from the geriatric medical unit to homes in urban Belfast. Each subject at the time of discharge was provided with a discharge note in a plain envelope containing details of medical diagnosis and discharge drug information. The

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patient or carer was advised to deliver the letter to the general practitioner as soon as possible. The envelope also contained a letter to the general practitioner in which to record the date and time of receipt of the discharge note and a stamped addressed envelope for its return.

In phase II we studied 71 consecutive patients discharged over a further two-month period. The same procedure as in phase I was followed except that the discharge note envelope was pre-printed with the reminder in capitals: "BY HAND: DO NOT POST: Please have this envelope delivered to your Doctor's surgery as soon as possible. The contents include a clinical summary of your admission for your Doctor." The patients for whom returned communications from the general practitioners were not received were identified. A further identical discharge note was posted to the general practitioner who was invited to complete the time of receipt of this discharge communication and return the information in an enclosed stamped addressed envelope.

TABLE

Delay, and failure of delivery of discharge information to general practitioners with comparison of plain envelope (phase I) and pre-printed envelope (phase II)

| <i>Results</i> | <i>Phase I — Plain envelope</i> | <i>Phase II — Pre-printed envelope</i> |
|----------------------------------|-------------------------------------|--|
| Discharged patients | 78 | 71 |
| Mean age (years) | 78.2 | 78.4 |
| No returns of information | 19 (24%) | 18 (25%) |
| Information returned | 59 (76%) | 53 (75%) |
| Delivery to general practitioner | | |
| same day | 4 (7%) | 9 (13%) |
| within 2 days | 19 (24%) | 30 (42%) |
| within 3 days | 26 (33%) | 36 (51%) |
| Mean delay (days) | 4.90 | 2.96 |
| Range (days) | 1 – 23 (median 4) | 1 – 10 (median 2) |

RESULTS

Details of the delay and failure rate of delivery of discharge information to general practitioners are shown in the Table. The results of the 78 patients in phase I (plain envelope) were compared with the 71 patients enrolled in phase II (pre-printed envelope). There was a similar failure rate of receipt of information by the general practitioner in phase I (24%) and phase II (25%). However, the mean delay of delivery of information is markedly different, 4.9 days in phase I and 2.9 days in phase II. The 18 patients in phase II for whom no initial communication was acknowledged from the general practitioners, but for whom a postal communication was separately sent, generated 16 returns (89%) with a mean delay of 2.1 days (range 1 – 14, median 2 days).

There was no apparent relationship between younger age of the patient and successful delivery of discharge note: the mean age of the 112 patients from whom the general practitioners received a handwritten communication was 80·6 years, in comparison to a mean age of 76·0 years for the 37 subjects from whom no communication was received. The mean mental score of those patients for whom the discharge note was successfully delivered was 7·3 in comparison to a mean score of 6·9 for those for whom no communication was received. Twelve (25%) of the 49 patients living alone failed to deliver the discharge note.

DISCUSSION

Initial diagnostic and drug information should be conveyed promptly from the hospital to the general practitioner after discharge. This is particularly important with regard to the elderly in view of their high re-admission rates and the incidence of medication problems.¹ Studies of the method of delivery of the initial discharge information has previously revealed conflicting results. A survey of discharges from a medical ward reported that hand delivery was quicker (mean 2·0 days) than postal delivery (mean 4·5 days) and the information was received by 97% of the general practitioners.² However, a mean delay of 4·3 days for hand delivery, with failure to arrive at all in 17% of cases has also been reported,³ and similar delays were found in other surveys of hand delivery, van delivery, or post.^{4, 5, 6} Some of these differences may reflect the varying proportion of elderly subjects included in these studies. Our survey in a geriatric ward setting confirms the inadequacy of relying on a hand-delivered initial communication. Use of a printed instruction note on the discharge envelope improved the delivery to the general practitioner within three days from 33% to 51%, but 25% of communications were still apparently not received. In contrast 89% of the posted discharge notes were acknowledged by the general practitioners. Age of the patients, mental score and living alone did not influence the speed of delivery of the discharge note.

Present systems of delivery of the initial discharge communication for elderly patients often fail or are slow. A dual system using an initial discharge communication for hand delivery to the general practitioners in a pre-printed envelope, with a second copy being simultaneously dispatched by first class post from the hospital should achieve both faster and more complete delivery. Future developments including facsimile transfer of discharge notes merit further study.

We acknowledge the assistance of the nursing staff and general practitioners involved in the care of the elderly in this study.

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Commentary:

A personal view of the hospital service

J Maguire

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"To write a prescription is easy, but to come to an understanding with people is hard" Franz Kafka.

It took some time before I knew what my illness was and even longer to appreciate how serious it might be. The diagnosis involved an extensive battery of tests and several visits to the hospital over a period of three months in 1989. Then it was revealed that I had a rare complaint known as the Carcinoid Syndrome and that my liver was heavily metastasised from an unknown primary source.

As it became clear that I was likely to have an ongoing contact with the hospital, both as an in-patient and an out-patient, I thought, perhaps naively, that I might link the experience to one of my professional interests — Improving the Quality of Service in the Public Sector — a major emerging theme for public servants. Here was an opportunity to observe service first-hand as a customer from inside a major teaching hospital.

I did not know then that in the course of two years I would undergo major surgery, chemotherapy twice, four hepatic embolizations, three acute crises (one of which confirmed me as a diabetic), innumerable examinations and tests, an assessment (and a rejection) at a national liver transplant centre as well as many problems at home just trying to lead a tolerable life. As it turned out, my observations became a distraction in some ways from the illness itself and, in a way, were therapeutic.

Not that it was a rigorous scientific study but rather a collection of anecdotes, conversations with other patients and my own straightforward observation. Not scientific, but true nonetheless and perhaps of some interest to those who work in the service and find it hard to see themselves as others see them. Maybe of increasing interest as they face up to the prospect of meeting prescribed standards of service under the Citizens Charter.

The concept behind the commentary was the notion of "the moment of truth", borrowed from bull-fighting and so successfully developed by Scandinavian Airline Systems as they transformed the airline's service to customers. To explain, an organisation experiences a moment of truth when the service provider and the service consumer meet. In a hospital thus there would be literally thousands of "moments of truth" every day as telephonists, receptionists, porters, cleaners, orderlies, nurses, doctors and many others made contact with patients and friends

James Maguire, former Director of the Public Service Training Council.

With acknowledgement to the Western Health & Social Services Board who commissioned the article for internal discussion.

of patients. Quality of service is primarily a perception of the “customer” or “client” so the challenge was to see if the hospital was a “customer” or “client” centred organisation. Did it deliver a quality service in the eyes of the critical customer?

But what does a patient expect by way of service? That was a starting point and it had to be a personal view. What I wanted was

- the best possible medical attention
- being cared for as an individual human being
- to be comfortable and free from pain
- to be reasonably well fed
- access to my relations and friends
- courteous and efficient contact with the hospital
- to understand and participate in managing my illness.

This last point may not be shared by all patients but many agree with my view that it is an important factor in coping and being positive.

Coincidentally with my own assessment, I noted from various staff newsletters in the hospital that management was promoting a “better service” campaign and encouraging staff to generate ideas for improvement. What disturbed me though, was that in all my sojourns in the hospital and from questioning many patients and nurses, I never heard of any organised approach to patients to let them articulate their views on service. It seems obvious that if you are going to improve customer service, you have to be clear on who the “customers” are, and consult them thoroughly about their needs. This is the starting point in improving service quality. I wonder did management even think of instituting a sample of “exit” interviews of patients?

Given the range of staff who have contact with the patients, here are some of my observations.

Contacting the hospital

The first point of contact with an organisation is critical. It makes a considerable impact on perceptions. Usually it is by telephone. For me it was a variable experience ranging from the courteous, informed and helpful to the uninterested, clueless and verging on being rude. Two times in five I would be left hanging, consigned to a ‘black hole’ of telecommunications, so that I had to make the call again, hopefully to a more efficient telephonist. It made me wonder how often managers or doctors took it upon themselves to test the service from outside to see what a customer’s experience was. I wondered too, given the variety of responses, what training was given, what drills were established and who monitored performance? It seems a clear-cut case for skills training based on good role models and regular monitoring. Easy to do and providing more satisfaction to staff and customer alike.

Many receptionists too seemed to lack the basic skills of receiving customers. Perhaps most noticeable was the absence of a greeting and the lack of eye contact. The latter possibly was due to the arrival of computer terminals on the desks which seemed to have a fatal fascination for the ladies concerned. Here, on the part of many staff, there seemed to be a problem of attitude and a need for good basic training.

Car parking

Car parks were clearly for the benefit of the staff who took up all the spaces nearest the hospital. The customers, (patients), relatives, friends, the elderly and infirm were consigned to parks farthest away from the hospital. No parks were reserved for visitors. When the hospital management made a bit of a song and dance about a new car park opening for the benefit of visitors, it was only too clear to see a week later, from the number of early morning parkers, that 80% of the spaces were taken up by the staff. Again the question "who are the customers?"

The medical service

I can only assess the clinical side of my experience as a layman and I am bound to say it was one of technical excellence. Doctors were highly skilled, concerned to do the best possible job and clearly had pride in the clinical expertise of the hospital. At the top end they seemed to relate well to one another and to see themselves as a team. As a customer the satisfaction and confidence in their expertise was high. This part of the core service was right. But was it enough?

Woody Allen reminded us that human beings were divided into mind and body. The mind embraced the noble aspirations like poetry and philosophy but the body had all the fun. We all know however that this is not true. Mind and body are inseparable and nowhere is this more important than in being ill. Too often I got the impression that when most doctors looked on patients they saw only the disease and not the human being. This may be a question of training, it may be a defense mechanism against the constant pressures of dealing with pain, or simply lack of resources and time, combined with bureaucratic pressures of the system. Not once in two years did anyone discuss with me how I felt about my illness, its impact on my life psychologically and the problems it caused at home. Consultants must have been aware of these difficulties but I felt they simply did not have the time to advise and counsel me. A sign of an overloaded system. Or, possibly they did not have the interactive skills necessary for this aspect of the job. Certainly my experience in King's College Hospital of a young consultant trying to break it to me that I would not be accepted for a liver transplant was a classic example of the absence of these skills. Yet he thought he was doing a fine job!

A plus point though for the consultant immediately concerned with me. He recognised my need to understand the ailment and what was being done for me and kept me fully briefed and involved in the treatment. His sensitivity however did not appear to be characteristic of the profession and too often doctors retained a certain mystique about what was really going on. Many patients need to know about what's going on and many want to share the responsibility of recovery.

Various conversations with the professionals also convinced me that consultants in particular would be so much more efficient if they had the right type of administrative and secretarial back-up. But who has ever studied their particular needs and who has ever taken it upon themselves to train secretaries in the skills that are needed? And who has ever thought about teaching a doctor to use a secretary, or to use his time effectively? Accepting that my contact was limited, the right type of back-up did not seem to be there. If ever I saw an opportunity for a productive study it was here.

In certain respects some of these criticisms apply to nurses. Highly proficient on the technical side of their jobs, they simply did not have the time to relate to patients except perhaps when they were making beds! Dedicated people,

overloaded for the sake of economy but not in a position to do a well rounded job. But in spite of the system and lack of resources this is the core service of the hospital and it is a prodigious service in spite of the obstacles.

Housekeeping and catering

Of all places you would expect a hospital to observe the highest standards of hygiene and no doubt this is true. I had only minor complaints on this score but they could easily have been rectified. In my ward there were two toilets for 20/24 patients and I accept that nothing could be done in the short term to improve this ratio. But cleaning was simply inadequate for the usage — once a day and no real check on standards. Indeed in one toilet over the two years no-one had ever bothered to replace the toilet roll holder and as a result toilet rolls were usually on the floor.

One of the trivial irritations too was the poor light from my reading lamp — 25 watts was simply not enough on a winter's day. Then I discovered that the bulbs had been changed from 40 to 25 watts to save money! A piddling saving if there ever was one! The situation was compounded too when I learned that lamps could only be changed by an electrician. It makes me wonder what stage we have got to when an orderly or a nurse cannot change a light bulb!

Catering managers, I know, labour under the restrictions of a budget but much more could be done by way of variety and presentation of food. The rest of the world seems to have passed the hospital by. Standards generally were poor and accepted too readily by patients because of their gratitude and obligations to staff. Perhaps if management had to undergo a hospital regime for a week or so they would take more interest in standards. But the recurring problem for me was cold meals! The trolley would arrive from the kitchen and sit for 15 – 30 minutes before meals were served. This was not a shortcoming on the part of the nurses — they simply had not the time to do the job that was required of them. Thank goodness for the microwave but really some check should be made at the point of delivery to patients. Patients certainly were not treated as customers when it came to being fed.

Improving service management

Quality service is an attitude of mind and it takes time to change attitudes. It needs a change in values too and this must be both a top-down and bottom-up process. Above all it needs recognition of who the customers are.

In Japanese management philosophy the employee learns to regard the client as the one who pays the salary. Consequently problems are regarded not only in terms of how they affect the organisation, but in how they affect the client. The hospital employee then, from porter through to consultant, has to empathise with the patient's needs and adapt accordingly. It's a continuous process. The hospital service is on the way, but has still a long way to go if it is to be truly customer-centred.

A word of caution though. Current thinking lays great emphasis on standards and measurement of performance. This could lead to a bureaucratic trap and a messy paper system. Once you apply widespread statistical measurement you run the danger of changing the true service culture of the organisation. Positive circles can easily become vicious circles! Perhaps what we need is more "management by simplicity", so that there are extremely clear signals about the standards of key norms that lead to quality service — and top management leads by example.

Historical Review

“Who fears to speak of ninety-eight”?

Hume Logan

Presidential Address to the Ulster Medical Society, 10 October, 1991.

Tonight, ladies and gentlemen, I ask you to cast your minds back 200 years — to 1791 — the year of the Mutiny on the Bounty, the death of Mozart and the publication of Boswell's *Life of Johnson*. Dublin, the second city in the British Empire, had a population of 200,000. It was grand and elegant. In contrast, Belfast had a population of 18,000 and was small and squalid.

On this day, 10th October 1791, 200 years ago, two men boarded the coach in Dublin at 10.30 am. Few could have imagined the carnage which would result from their trip to Belfast. The two men were Theobald Wolfe Tone and Thomas Russell. Wolfe Tone is the most famous name in Irish history. He was the son of a Protestant coach builder in Dublin and his ambition was to join the army. His father opposed this and insisted that he entered Trinity College, Dublin, to train as a lawyer. Later he eloped with Matilda Withering of Grafton Street, and went to the Middle Temple in London for two years to become a barrister. When he returned to Ireland he joined the Leinster Circuit but he did not really like the law and was a failure as a barrister. He was more interested in politics and became involved with the Whig Club in Dublin which had been started by Lord Charlemont who was also the commander of the Volunteers. Tone started to write political pamphlets and as a result he was appointed secretary to the Catholic Committee which was then campaigning for catholic emancipation. Tone wrote a pamphlet entitled *An Argument on Behalf of the Catholics of Ireland* in the summer of 1791, which brought him to the attention of the radical section of political thought in general and of the Volunteers of Belfast in particular. This



Fig 1. Theobald Wolfe Tone (1763 – 1798). (from Madden, *The United Irishmen*, 1846: Reference 10).

was a group, all protestant at first, which had been reactivated to protect the island from invasion by the French, but which had become much more active in promoting catholic emancipation, free trade and freedom of the Irish parliament from its English counterpart. Some of the Volunteer leaders invited Tone to come to Belfast to discuss the setting up of a radical group there. (Fig 1).

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Fig 2. Thomas Russell (1767–1803).
(from Madden, *The United Irishmen*, 1846:
Reference 10).

Tone brought with him Thomas Russell, a Protestant born in Cork who had met Tone in the gallery of the House of Commons and they became firm friends. Unfortunately he had gone bail for an American who had absconded and he had to sell his army commission to pay the forfeit. He was a tall handsome man who, while in the army had been posted to Belfast where he became very popular. (Fig 2). He met many of the liberals at this time and it was through him that Tone was invited to Belfast by the leaders of the Volunteers. These were mainly well-off merchants who could afford their own uniforms and arms. The organisation in the north was purely protestant

and mainly presbyterian as the catholics were not allowed to carry arms and the protestants did not really want them to do so as arms were a symbol of supremacy. It must also be noted that while they supported catholic emancipation they were staunchly against the catholic church. When Tone came to Belfast they were loyal to the Crown, but later this was to change.

The Volunteers were commanded by Lord Charlemont who had formed the Dublin Whig Club in 1789. This movement spread to Belfast the following year largely to control the exuberance of the Volunteers and they were much less committed to catholic emancipation. The co-founder and secretary in Belfast was Dr Alexander Henry Halliday. (Fig 3). He was born in 1728, the son of the minister of the First Presbyterian Church in Rosemary Street. He matriculated in Glasgow in 1743 and graduated there in 1751. Where he received his medical education is not known as there was not any clinical teaching in Glasgow till 1794 when the infirmary was opened. He wrote in 1751 to Dr Cullen who had been appointed Professor of Medicine in Glasgow stating that he had been in Paris at the time of the appointment; it might therefore be assumed that Halliday was studying in Paris before his graduation. Later he practised in Belfast and travelled all over the province of Ulster, charging one guinea per mile — despite this he was very popular. He had a long association with the Charitable Society and was President of the Linenhall Library from 1792 to 1798. Martha Matier wrote to her brother Dr William Drennan, perhaps a little uncharitably "I think they had an eye to his books more than to himself when they paid him the compliment".



Fig 3. Dr Alexander Halliday (1728 – 1802).
(Reproduced by kind permission of Mrs Martin
de Bartolomé).

In 1770 Halliday was involved in an episode when Lord Donegall raised the rents on his estates in Co Antrim. The leases fell due at that time and fines were imposed on top of the leases. The tenants agreed to pay the increased rents but did not have the ready cash for the fines. This led to rich speculative merchants from Belfast taking over the leases and subletting the lands for profit. A co-operative organisation, the Hearts of Steel, incensed by this carried out various outrages on the cattle and farms of the tenants and when caught were prosecuted by the speculators. One of these was David Douglas, who was arrested by Mr Waddell Cunningham for haughing the cattle of his partner Mr Gregg, and was lodged in the barracks in Belfast. This resulted in a meeting of the Hearts of Steel in Templepatrick at which it was resolved to proceed to Belfast and free Douglas. Men having been collected *en route* to Belfast, some 1200 surrounded the barracks in Barrack Street and sent in a message demanding the release of the prisoner. This being denied they went to Cunningham's house at the lower end of Royal Avenue, broke into it and proceeded to break up the furniture. Dr Halliday mixed with the crowd and expostulated with them but was taken prisoner. He agreed to go to the barracks and try to procure the release of Douglas, promising that if he failed he would return and surrender himself as a hostage. At this stage the barrack gate was thrown open and shots were fired into the crowd by the soldiers, killing five and wounding nine of them. Halliday's interference prevented further firing but as he had not returned the crowd set fire to Cunningham's house and threatened to destroy Halliday's as well. The risk of the town being burnt down was so great that the prisoner was released and the crowd dispersed. When related it is not usually disclosed that Halliday had a lease for 539 acres with a rent of £120.6.0 and a fine of £500. Perhaps his efforts were not entirely altruistic.

The journey from Dublin took Tone and Russell thirty-six hours. On arrival they arranged a meeting for Friday, 14th October in Sugarhouse Entry, off High Street, to formulate the resolutions to be put to the inaugural meeting of the Society of United Irishmen, which took place on the 18th October. Tone is therefore credited with formation of the society — but did he? A year earlier he had formed a small club of nine people to discuss politics in Dublin. Two of the members were doctors — Whitley Stokes of whom Tone wrote "The very best man I have ever known" and who later became Professor of Medicine at the Royal College of Surgeons. The other doctor was William Drennan about whom I have already addressed this Society. Drennan had worked in Newry and then moved to Dublin. Later, on his return to his native Belfast he was a founder member of the Belfast Medical Society and was instrumental in the foundation of the Royal Belfast Academical Institution and therefore of our medical school. Drennan had earlier written to his brother-in-law about setting up a secret society and through their political club Tone must have known of this: I would submit that the true founder of the United Irishmen was William Drennan.

Tone left Belfast after the foundation of the society and returned to Dublin where further societies were set up. He did not return to Belfast till 1795 when he was on his way with his wife and family to exile in America. This was part of a deal made with the government when he had been associated with William Jackson who had come to Ireland as an emissary of the French. Before embarking he went out to the Cavehill and near McArt's Fort he and some of the founders of the society (Russell, Neilson, Simms, McCracken) made "a most solemn obligation . . . never

to desist in our efforts until we have subverted the authority of England over our country, and asserted our independence". Having been fêted in Belfast and presented with £1500 Tone and his family sailed to America. He did not stay long in the New World as he was pressed to go to France and try to encourage the French to invade Ireland. He soon became friendly with General Hoche, and with his support the Directorate mounted a force to invade Ireland in 1796. This was a disaster, the commander's ship was separated from his troops, and when they gained the Irish Coast the seas were so rough many could not even enter Bantry Bay. Those who did get into the bay were unable to disembark their troops, and ultimately the ships cut their cables and fled back to France.

The threat of invasion and its ultimate occurrence frightened the authorities. The Militia Act of 1793 had produced by ballot a force of catholics officered by protestants to act as local police. However, the threat of invasion had taken them from their local role to a national one, and consequently they had to be replaced by yet another group — the Yeomen. These units were formed in 1796 and were made up largely by loyalists and Orangemen. Political activity had driven some of the catholics into the United Irishmen and the societies were becoming much more organised and were arming to the best of their ability. The government needed to get information about what was going on and who was involved in the secret societies. They did this by bribing members of the societies to inform on their colleagues. Soldiers were also quartered in the local houses — something which was hated by the inhabitants. Intimidation was widespread and took the form of flogging, hanging, the application of pitch caps and burning of home-steads. These atrocities were perpetrated by the soldiers and often initiated by the local magistrates who were loyal, Orange in outlook and anything but impartial.

By 1797 things were becoming so serious that General Lake issued a proclamation which in effect established martial law, protecting informers and encouraging all arms to be given up under threat of death. The object was to disarm the rebels but it probably had the opposite effect and it was said there were 280,000 United

Irishmen at this time. They were poorly armed as most had hardly enough to live on. Local blacksmiths forged pikes which were hidden in thatched roofs, the houses of the better off were raided, their guns stolen and their garden statues taken to melt down into musket balls.

By this time the United Irishmen were highly organised. Each local group sent a representative to a higher group and ultimately to provincial groups and a directory in Dublin. One of the leaders there was Lord Edward Fitzgerald, a brother of the Duke of Leinster and a member of the premier family in Ireland. Wealthy and influential, the Duke owned Carton, a grand house in the country, and Leinster House in Dublin which is now the seat of Dail Eireann. In 1798



Fig 4. Lord Edward Fitzgerald (1763–1798).
(British Museum).

Lord Edward was thirty-five. He had spent most of his life in the army and had travelled widely in Europe as well as in Canada and America. He was married with a young family and had been a member of the Irish parliament. Uncharacteristically here was an outstanding member of the ascendancy who had become a leader of the revolutionary United Irishmen. (Fig 4).

Early in 1798 plans were being made for the rebellion and the government were fully aware of what was happening, due to its informers. On 12th March a meeting of the directory was to take place in Oliver Bond's house in Bridge Street in Dublin. The government were informed about this meeting by a man called Reynolds, a confidant of Lord Edward, and the whole directory were arrested at one time. The only one to escape was Lord Edward — for which no explanation has ever been given. He may not have been present when the police arrived or he may have escaped. It was said that he was breakfasting with his friend Dr Macneven and they were preparing to go to the meeting when the police arrived and arrested Macneven but let Lord Edward go. Realising he was in danger of arrest he seems to have made his way to Dr Kennedy's, from where he is known to have left with Surgeon Lawless. He spent some time in a widow's house beside the canal and then moved around houses in central Dublin. By this time it was early May and the rebellion was due to start. Lord Edward had been on the run for two months and £2000 was offered for his arrest. This was too much for some and on 19th May the authorities arrived at the house of a feather merchant called Murphy to arrest Lord Edward. He was resting in bed when they arrived but defended himself with a dagger which he had hidden in the bed. He stabbed Captain Ryan in the groin but Major Sirr arrived and shot Lord Edward in the right shoulder. His wound having been dressed by Dr Adrien he was taken by sedan chair to the Castle where he was examined by the Surgeon General — George Stewart. As he had been captured by civil authorities they demanded he be taken to Newgate gaol where he was attended twice daily by Stewart and Dr Lindsay. Ryan died on 30th May and Lord Edward's condition started to deteriorate on 1st June, the thirteenth day of his confinement. It was decided that Dr John Armstrong Garnett should be employed to live in the prison and look after Lord Edward. Garnett left a diary which accurately recounts all that happened. On 2nd June a man called Clinch who had been a militia officer and had gone over to the rebels was hanged for treason outside Lord Edward Fitzgerald's cell: the noise involved in erecting the gallows and in the execution seemed to upset the prisoner. Garnett noted that Lord Edward was very agitated. His tongue was thrust forward and his jaws closed with "the most rigid spasm". After a time he became much better and was able to eat a little. It was recorded that "his pulse flutters excessively". On 3rd June Garnett was able to read to him, and he had requested "the account of our Saviour's death". Later in the day he had been rational but then developed twitching and in the evening his breathing became more difficult and he raved. Around midnight he was visited by an aunt and his brother; he was said to know them and embraced the brother but at 2.00 am he succumbed. It has been recorded "an inquest was held in Newgate on the body of Lord Edward Fitzgerald and on the evidence of Surgeon Leake a verdict returned of death from water in the chest". There is other evidence to suggest that a post mortem was carried out but I have been unable to obtain the details. However, I have no doubt that Lord Edward Fitzgerald died of tetanus as all his symptoms were typical of this disease.

Whatever the cause it was a great loss to the rebels as he was the only man who had sufficient military experience to direct the strategy for the rebellion which had already started at the time of his death.

It is interesting to look at the history of these doctors who looked after Lord Edward. Dr John Adrien was a United Irishman and was educated in Paris. His son became the first Professor of Medical Jurisprudence in the Royal College of Surgeons. Dr William James Macneven was a member of an old Ulster catholic family transported to Connaught by Cromwell. He had an uncle in Germany who was a doctor and had married well and became Baron Macneven. He brought his nephew to the continent and educated him in Prague and Vienna where he graduated in 1783 at the age of 20 years. He returned to Dublin where he practised until he was arrested in 1798 and taken to Kilmainham Jail and later transported to Fort George in Scotland. On his release in 1802 he went to France and joined the French army but resigned in 1805. He then emigrated to New York where he was appointed Professor of Midwifery at the College of Physicians and Surgeons in 1808. Three years later he changed to the chair of chemistry and in 1816 added the chair of *Materia Medica*. He died in 1841.

Surgeon William Lawless was born in 1764 and was indentured to Michael Keogh in 1781 for five years. He was elected Professor of Anatomy and Physiology in the Royal College of Surgeons in 1794, the year he joined the United Irishmen. After the arrests at Bond's house he became a member of the new directory but was informed that there was a warrant out for his arrest and so he left Dublin. On approaching his mother's house his sister signalled from the drawing room window that Major Sirr was there searching for him, so he went to the house of a Mr Byrne at Kimmage where he was concealed in a garret. To help him leave Ireland his brother sent a carriage for him, and dressed as a woman he went with the three Miss Byrnes to Dublin, where he changed into a sailor's suit. As he had a long easily recognisable face he carried a cable on his head and he was not recognised by Major Sirr whom he passed on the way to the quay. He boarded a ship and went to France and after his arrival joined the French army, attaining the rank of Brigadier General in the Irish brigade, having been decorated with Legion of Honour by Napoleon.

George Stewart was born in Co Tyrone in 1752 and his father was the High Sheriff at that time. He set up practice in Dublin in 1773 and was Surgeon to the Charitable Infirmary on Inns Quay and Jarvis Street. He was one of the group who started the Dublin Society of Surgeons in 1780, their main function being to petition for a Royal Charter to separate the Surgeons from the Barbers, which led to the founding of the Royal College of Surgeons in 1784. He was the eighth President of the College in 1792 having been appointed State Surgeon in 1785 and Surgeon General to the Forces in 1787. A bust of George Stewart was placed in the College on the motion of the great Abraham Colles as Stewart had encouraged him to change from being a physician to being a surgeon.

John Armstrong Garnett was born at Thurles in 1767, the son of the master of Tipperary Grammar School. He obtained his letters testimonial in February 1798 and in 1803 he became Surgeon to Dean Swift's Hospital and to the General Dispensary, and later that year Professor of Surgical Pharmacy. He left Ireland in 1811 because of poor health and died in 1831 from paralysis. It should also be

recorded that William Dease, first Professor of Surgery at the College, was a member of the United Irishmen.

Lord Edward Fitzgerald was arrested on 19th May and the rebellion started on 23rd May despite the arrest of all the leaders in Oliver Bond's house two months earlier. There was no doubt at this time that the main feeling for the rebellion was in the south: in the north there had been a diminishing revolutionary fervour. Nothing happened in Ulster until 6th June when a number of insurgents attacked Larne where the garrison was 20 men and one officer. The insurgents were making progress when information was passed to them that Carrickfergus Castle had been captured. This was incorrect but the rebels decided to abandon the attack on the garrison and proceed to Donegore hill to join the main body which was to attack Antrim. This group was led by Robert Simms, who had been in charge of the United Irishmen in County Antrim, but he did not agree with the rebellion at that time and Henry Joy McCracken was hastily chosen to replace him. (Fig 5).



Fig 5.
Henry Joy McCracken (1767–1798).
(Ulster Museum).

McCracken was born in High Street, Belfast in 1767 and so at the time of the rebellion he was 30. His father was captain and part owner of a ship which traded between Belfast and the West Indies. His mother was Anne Joy, a daughter of Francis Joy who had started the Belfast Newsletter in 1737 and she had two brothers, Henry and Robert.

Lord O'Neill had convened a meeting of magistrates for the 7th June in Antrim to discuss the rebellious state of the country. The rebels therefore decided to attack Antrim on that date. Through their informers the authorities knew to expect this event and preparations were made. McCracken gathered his men at Roughfort, but there were only 20 of them, many fewer than had been expected. The enthusiasm for rebellion had waned — "some were seized with most violent bowel complaints, cramps, rheumatic pains; the wives of several given out to be on point of death, others suffered ankle strains etc. Some of these, notwithstanding their piteous wailings, were forced along to the ranks, while others after a few hearty kicks were suffered to remain". The rebels proceeded from Roughfort to Templepatrick where two cannons from the earlier Volunteer days were hidden in the old presbyterian church. One of these was unserviceable because it did not have a carriage, the second carriage was very poor but was just the same. By the time they arrived at Antrim some 5000 men from the surrounding country had joined the party.

McCracken set up his flag in the old graveyard and proceeded into the town up the main street. The battle went well for the rebels, who were winning until the

cavalry were released to attack them. The cavalry thought they were surrounded and galloped off towards Ballymena. Rebel reinforcements from Randalstown arrived at this critical time but were confused and consequently retreated. The battle was over, the rebels being defeated. The whole battle lasted from 2.45 to 4.00 pm. The rebel force may have amounted to 5000 men of whom 100 were killed during the battle and 200 during the retreat. The bodies were carried in carts to a sandy burying ground near Lough Neagh. A Yeoman officer in charge of the burying asked the driver of a cart sitting on his ghastly load "where the devil did these rascals come from!" A poor wretch feebly lifted his head and said "I come frae Ballyboley". He was buried with the rest.

Lord O'Neill was present at the battle and after emerging from the Market House to cross to the Castle was piked by a rebel. It was said that a Dr Morton and his family were hiding in the cellar of their house on the corner of High Street and Massereene Bridge when there was a loud hammering on the door. The doctor thought it was the rebels who had come to shoot him, but it was some soldiers who had come to seek help for Lord O'Neill who they feared was dying. Another account states that his lordship was taken to Dr Bryson's where he sat on the steps, and as there was no answer from within he was carried back to the Market House. This account stated that he was then taken by boat to Shane's Castle but in fact he probably died eleven days later in Antrim Castle as stated in the Belfast Newsletter. The surgeons were fourth in order of the cortege after the firing party, Lord O'Neill's horse and the clergy, all of whom preceded the hearse drawn by six horses. I have been unable to trace the names of the surgeons. What the doctors, if any, did at the battle does not seem to have been recorded. Dr Agnew owned a public house in Templepatrick where the United Irishmen met. He was warned that he was wanted by the army and escaped, but his house was burned down. Dr Thomas Houston of Carnall was away treating a patient and did not get back till 2 pm when he set out for the battle — presumably it was over when he arrived. Later he removed bullets from one Samuel Barron of Ballylinney.

McCracken led the remainder of his men out of Antrim with the intention of going on to Ballymena and helping the rebels there. However, he was diverted to Slemish with 100 survivors, the rest having melted away. Ultimately 20 men set off with him heading at first for Saintfield but diverted again to Colin mountain. After some weeks in hiding it was arranged that he would leave the country by boat from Carrickfergus, but when he got there he was recognised by a Yeoman as he crossed the Green. At first imprisoned in the Castle he was later brought to Belfast for trial, where he was found guilty of treason and hanged at the corner of High Street and Cornmarket. His body was not mutilated and was given immediately to his family. At this time there had been talk of reviving people who had been drowned, and the famous Dr James McDonnell was sent for. He did not come but sent his brother Alexander. Despite efforts "to restore animation" McCracken did not respond and he was ultimately buried in the church yard in High Street.

County Down was as militant as County Antrim. The first United Society in the county was formed in Saintfield in 1792. and the rebels grouped there the day after the battle at Antrim. They heard that the army and yeomen were coming from Comber and they ambushed them in the demesne of Mr Nicholas Price — Saintfield House is still in the same family, the children from the Ulster Hospital

were treated there after the air raids in 1941. The rebels at this stage were under the command of Dr Jackson from Newtownards who led them to Creevy Rocks. A party of rebels also set out from Donaghadee and Bangor area to take the garrison in Newtownards but failed to do so. They retreated to Conlig and then returned to Newtownards to find the garrison gone. Having been joined by groups from Killinchy and Killyleagh they spent the night at Scrabo and then proceeded to Creevy Rocks. On Sunday the Rev Birch preached a sermon on the text "Cause them that have charge over the city to draw near, even every man with his destroying weapon in his hand". He was tried for this but got off because his brother, a doctor in Ballybeen, was a friend of Lord Londonderry.

General Nugent in Belfast proclaimed that all loyalist prisoners were to be released by the rebels and all arms given up or he would burn all the houses around Saintfield and everyone who was armed would be put to the sword. He proceeded to Saintfield, carrying out his threat. The rebels meanwhile had gone to Ballynahinch under the command of Henry Munro — a draper from Lisburn — and Nugent followed them. The first battle was at Bells Bridge and the army then captured Windmill Hill near the centre of the town. (Fig 6). One rebel was hanged from the sail of the windmill. The rebels retreated into Montalto, the estate of Lord Moira, and the army proceeded to get drunk in the town. Munro was encouraged to attack the army from Ednavady during the night but would not do so. Nugent attacked at 4 am but Munro beat the army troops back into the centre of the town. The loyalist buglers sounded the retreat, but the rebels mistook this for the signal to attack and they retreated. Nugent was the first to appreciate the situation and pursued the rebels who fled.



Fig 6. The windmill at Ballynahinch.

The Battle of Ballynahinch was fought between 5–7000 rebels and 2–3000 troops. Of the rebels 300 were killed in the battle and 200 in the retreat. Nugent claimed that six soldiers had been killed and seventeen wounded — an absurdly low figure. Munro escaped but was betrayed and ultimately hanged in Market Square, Lisburn across the street from his own shop. Little is known of the man but he presented two jugs to a local masonic lodge, which later had to be disbanded because of its radical political views.

A total of 129,636 armaments were captured by the army, including 70,000 pikes, 48,000 guns, 4,400 pistols and 4,100 swords, with 22 pieces of ordinance — these figures give some indication of the number of rebels involved.

Of course, the two battles which have been described in Ulster were a very small part of the rebellion. As I have already stated Ulster had lost its will to fight but this was not the case in the south-east, in Wexford and Wicklow. A very bloody battle had started in May and continued on through July. In Ireland there were probably

190,000 soldiers, militia and yeomen involved on the loyalist side, and perhaps 280,000 rebels. The exact number of deaths is unknown but Madden gave a figure of 70,000; Dublin Castle estimated a figure of 20,000 and the real figure was probably in the region of 30,000. The official military figure not including the yeomen was 1,060 deaths.

The object I set for this address was to establish what happened to the injured on both sides in this rebellion. Sadly I have failed completely, as I have been unable to unearth any information regarding the injured on either side. In the case of the rebels this is fairly easy to understand. Many women attended the battles at Saintfield and Ballynahinch. They made the food for the men and dressed the wounds of the injured and then accompanied them home where they were hidden — if they were found by the army they would have been shot. A letter written to the Marquis of Downshire from an officer of the Yeomen in Hillsborough stated that they were going out to hunt rebels after the Battle of Ballynahinch — this may explain why some of the injured were thought to have been taken to the Isle of Man or Cumberland. Many of the injured rebels were said to have been shot by their colleagues to prevent information and names falling into army hands.

After the *débat* of the French invasion in 1796 Tone returned to France where he was promoted to the rank of Adjutant-General. Napoleon saw the wisdom of invading Ireland and forces were gathered at Brest, La Rochelle and Dunkirk. All had great difficulty in getting out from the ports, so it was not until 23rd August that General Humbert landed at Killala Bay in Co Mayo. He made his way to the Bishop's Palace at Killala and on to Castlebar where he routed the natives in an action subsequently known as the "Races of Castlebar". He had not had the support of the rush of rebels to join his forces which he had expected, and did not have a clear strategy, and his force was defeated at Ballinamuck. He was to have been supported by two other groups. One, led by Napper Tandy, landed on Rutland Island off the coast of Donegal. Tandy approached the town and took it without bloodshed. He found that the postmaster was an old friend of his and together the two got drunk: the next morning he was carried back on to the ship insensible and the ship sailed for France. The nine ships in the Brest fleet with 3000 men on board were defeated off Lough Swilly on 12th October. Amongst those captured was Tone who was landed at Buncrana on 3rd November.

Wolfe Tone was taken to Derry and then transferred in chains to Dublin. He complained bitterly of this as he claimed that he was an officer in the French army and should therefore be treated as a prisoner of war. He was tried on 10th November, found guilty and condemned to death. He requested in a statement which he made to the court that he be executed as a soldier by firing squad but on the day after his trial he was told he was to be hanged at Newgate. During the night using a penknife he cut his throat and severed his trachea. He was treated by a French immigrant surgeon called Lentaigue to whom he announced that he (Tone) was a bad anatomist. His trachea was sutured and his condition was declared not to be serious and his execution was to go ahead. However, his father obtained an injunction delaying the execution.

In the Freeman's Journal of 20th November there was the following report: *Tone*, that unfortunate and irreligious man, equally a rebel against the laws of his Country and his God, died yesterday morning in consequence of the wound which

he inflicted on himself. An inflammation, which was the result, extended to his lungs and proved mortal. The Coroner's Inquest sat on the body, and brought in a verdict of *self murder* — horrible crime. It is said, his head will be placed on the top of the New Prison, as his death does not exonerate him from such part of the sentence as can be put in execution — that is, if it shall be decided that he died in the legal possession of the military power”.

I started this address with Tone and Russell. Tone died in Dublin, Russell later in Downpatrick. Both gave their lives for their beliefs. I wonder if they knew this would be their destiny when they left Dublin this day 200 years ago.

*“Who fears to speak of Ninety-Eight?
Who blushes at the name?
When cowards mock the patriot's fate,
Who hangs his head for shame?”*

I wish to express my gratitude to Miss C Davidson for her help with the preparation of this paper. My thanks are also due to Mr N Irvine for the photographs, to Mrs E Doran, Medical Library, The Queen's University of Belfast and to the staff of the Linenhall Library, Belfast, who went to considerable trouble to help with the research.

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Historical Review

Cinderella had a champion

J M G Harley

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Society today expects the physician of the future to concern himself increasingly with social justice, the environment and government regulations. However, when attempts are made to deal with these subjects they usually focus on the 'here and now' and not the historical underpinning. The student, practitioner and public, often through the media, marvel at the contemporary medical scene with its greater scientific understanding, diagnostic and operative tools, therapeutic methods and broadened attitudes towards the whole patient. Nevertheless, they are apt to view today's practices either as having always been there or, indeed, as if they had miraculously appeared from outer space.

Through events in the lifetime of one of our colleagues I will try to illustrate that medicine is more than just medicines, that the development of medicine has not been an uninterrupted straight line of progress, and that society and the profession have exerted a mutual influence on each other. Furthermore, we may see that the effectiveness of any one doctor in relation to his patients and colleagues was, and still is, more a function of his own qualities and abilities than of any philosophy.

My story begins in the last decade of the 19th century, when on 28th July 1898, Charles Horner Greer Macafee — affectionately known as 'Mac' — was born in Omagh, Co Tyrone. At that time, this site which now contains the Royal Group of Hospitals, was parkland on which stood the County Infirmary for the Insane (Fig 1). This hospital, then in Frederick Street and known as the Belfast Royal Hospital, had celebrated its centenary in 1897, and Queen's College, Belfast, was preparing for its fiftieth anniversary in 1899.

Ireland was 'united' (partitioned in 1920). The so-called 'troubles' were ongoing but there was relative calm compared to the very turbulent 18th century which had

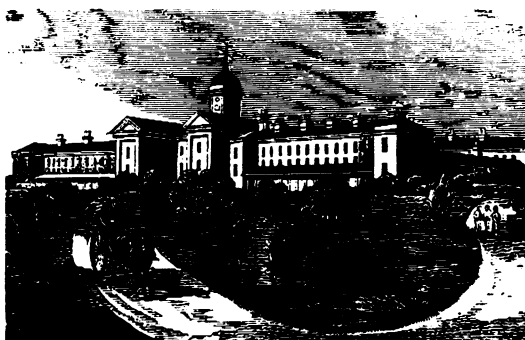


Fig 1. County Infirmary for the Insane. This building was demolished prior to the building of the Royal Victoria Hospital.

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culminated in the Rebellion of 1798. Queen Victoria's reign, which had started in 1837, was coming to an end. There had been a great increase in the population and nowhere was this more evident than in Belfast where in 1780 the population was only 13,000 — one-twentieth that of Dublin. By the year of Mac's birth, Belfast's population was some half-million — more than the Irish capital — and rated as the fastest growing city in the industrialised world. It could boast the greatest shipyard, ropeworks, tobacco factory, linen spinning mill, dry dock and tea machinery works in the world — sadly most have now gone.

The great increase in population was partly due to large families, and partly to the fall in death rate and rise in life expectancy. This latter was 44 years for men and 48 for women. It is not surprising, therefore, that the word 'menopause' was unheard of. Now, nearly a hundred years on, life expectancy has risen dramatically to 75 years for males and 81 years for females. Women can now expect to live nearly half of their lives after the menopause, and the pharmaceutical companies can look forward to making millions from hormone replacement therapy.

Divorce affected only two in every 1,000 marriages, now at least one in three. Unmarried mothers were rare — now, one in four is single. Perhaps the sentiments expressed in Tennyson's 'The Princess' need reappraisal:

"Man for the field, woman for the hearth
Man for the sword and for the needle she:
Man with the head and woman with the heart
Man to command and woman to obey".

In 1898, tuberculosis was rife, and terrible scourges such as smallpox, cholera, typhus and diphtheria that devastated populations were still common. Infant mortality was 102 per 1,000 and maternal mortality 8 per 1,000; now, 7 per 1,000 and less than 1 in 10,000 respectively. The hospitals, and the energies of the medical and nursing professions were directed mainly towards the prevention, diagnosis and, where possible, the treatment of infectious diseases. General medical cases were admitted only by special permission, and surgical problems were mostly dealt with in the patients' homes.

Midwifery had still not emerged from the dark ages. Little had changed for centuries. That it was the Cinderella of medicine can be judged by the attitudes of physicians and surgeons. Sir Henry Halford, President of the Royal College of Physicians, in a letter to Sir Robert Peel the Prime Minister, stated that the practice of midwifery was "an act foreign to the habits of gentlemen of enlarged academic education", and Sir Anthony Carlisle, Royal College of Surgeons, told a select committee that "It is an imposture to pretend that a medical man is required at a labour".

It was not until the Medical Act of 1886 that qualification in medicine and surgery also required midwifery before a student could be registered. It seems incredible that only 12 years (1886) before Mac was born, midwifery for the first time achieved the same academic status as medicine and surgery. This was only the beginning, for the universal acceptance of midwifery, still perceived as the Cinderella of medicine, was to take another 40 years.

Reluctance by the medical profession to recognise the importance of a proper knowledge of midwifery explains to some degree the absence of public support

for legislation for the control of midwives, who were likened — rightly or wrongly — to Sarah Gamp, as portrayed in Charles Dickens 'Martin Chuzzlewit' (1843). I quote: "She was a fat woman. Having very little neck it cost her some trouble to look over herself. The face of Mrs Gamp — the nose in particular — was somewhat red and swollen and it was difficult to enjoy her society without becoming conscious of a smell of spirits".

The atmosphere of apathy, intolerance and aggression against the compulsory training and registration of midwives persisted until finally, as a result of sheer persistence and determination by the midwives themselves, Parliament passed the Midwives Act in 1902, establishing the Central Midwives Board governing the training and practice of midwifery. To appreciate how significant this achievement was, one has to remember that women had no voting rights at that time. At present, most midwives are fully trained nurses who have completed a further 18 months postgraduate training and an examination in midwifery. Why, since the advent of Project 2000, they are intent on being trained only as midwives and not first as nurses, I have great difficulty in understanding, particularly as the numbers of pregnancies with medical disorders are increasing.

As if to mark the year of Mac's birth, two historical events occurred. The first caesarean section in Ireland where mother and baby were to survive, was performed in the Rotunda Hospital, Dublin in 1898 by the Master, Dr Arthur Macan. Secondly, the British Medical Journal recorded that three young ladies had applied to the Medical Professors of Queen's College, Belfast, to have the medical classes thrown open to women.

This was the political, social and medical status when Mac was born in the front bedroom of what was the First Omagh Presbyterian Manse. His father, the Reverend Andrew Macafee was the Presbyterian Minister in Omagh. Thus, Mac was the third son of the Manse to occupy the Chair in Midwifery — the two preceding him were Professors Dill and Sir John Byers. His mother was a trained nurse. There were two children from this marriage — Mac and his younger sister Edith (more commonly known as Pat) (Fig 2).



Fig 2. Rev Andrew Macafee and his children C H G and Edith; about 1902.

During his childhood Mac was always aware of the social problems of poverty and drunkenness and the inevitable political unrest. The problems of alcohol abuse were not helped by the 'spirit grocer', a common feature in those days. Women were spending more money on gin than on groceries and, as a result, their children were starving. Often his father's parishioners were taken home by their horses as they lay incapable in the bottom of a cart. Poverty was prevalent; particularly among elderly widows and spinsters. There was, of course, no National Insurance scheme until 1911. The poor depended mainly on money from the 'Church Poor Fund' and every Saturday Mac was given a number of envelopes, each containing half-a-crown, to deliver to the poor throughout the town.

Educated at Omagh Academy and Foyle College, Londonderry, Mac entered the Faculty of Medicine at Queen's in October 1916, graduating 5 years later with first class honours, taking first place in midwifery and gynaecology. The Professor of Midwifery, Sir John Byers, had died in 1920, a year before Mac qualified, and the Chair was divided into two — midwifery (Professor C G Lowry) and gynaecology (Professor R J Johnston).

During his house officer's year at the Royal, the IRA was doing its best to burn down Belfast and at least 600 corpses due to the 'troubles' passed through the mortuary. On completion of the year he decided to specialise in obstetrics and gynaecology and subsequently sat and passed the examination for Fellowships of the Royal College of Surgeons of both England and Ireland in 1927.

Mac was the first University Tutor in Obstetrics at Queen's. He describes conditions in the Belfast Maternity Hospital in Townsend Street as deplorable. While this may have been so, nevertheless, in 1921, Mr H L Hardy Greer, one of the medical staff, established the first antenatal clinic in Ireland in that hospital. Mac resigned as Tutor in 1925. He went into private consulting practice but continued his commitments in the Belfast Maternity Hospital and the Royal Victoria Hospital, where he was appointed assistant gynaecologist in 1929.

It was in 1929 that obstetrics and gynaecology made the most major advance as a specialty with the founding of its own College. Although Blair Bell and Fletcher Shaw are regarded as the founders, Professor C G Lowry and other members of the Gynaecological Visiting Society of Great Britain and Ireland played important roles. In fact, Professor Lowry was one of the 9 signatories of the 'Articles of Association' submitted to the Board of Trade.

Not surprisingly, the Royal Colleges of both Physicians and of Surgeons objected to the proposed issue of certificates for proficiency in obstetrics and gynaecology by another body. Following a Board of Trade enquiry the Colleges conceded and, at last, obstetricians and gynaecologists had their own College. Mac was a foundation Fellow and later a Vice President, as had been C G Lowry. Mac's successor Professor Jack Pinkerton also became a Vice President.

In 1930, Mac married Margaret Crymble Lowry, daughter of Professor C G Lowry and Mrs Lowry. Their best man was Arthur Eakin, a surgeon in Omagh. Margaret and Mac lived at 18 University Square. They had three children. Jeremy, the eldest, is a consultant obstetrician and gynaecologist in Leicester, and is married

with two sons. Alastair is one of our consultant orthopaedic surgeons; married with four children he lives in the family home in Donaghadee. Anne, the youngest, trained as a nurse in the Royal and is married to David Mahood. They live in Holywood, Co Down.

Mac was now very busy in private and hospital practice. To make matters worse, most of Professor Lowry's time was occupied negotiating the transfer of the Maternity Hospital from Townsend Street to the Grosvenor Road site and its amalgamation with the Royal Victoria. The new hospital was opened in 1933 and designated the 'Royal Maternity Hospital', Belfast (Fig 3) (George V, 1935), not the 'Maternity Unit of the Royal Victoria Hospital', as the media and others often incorrectly state. The gynaecological unit has always been part of the Royal Victoria.

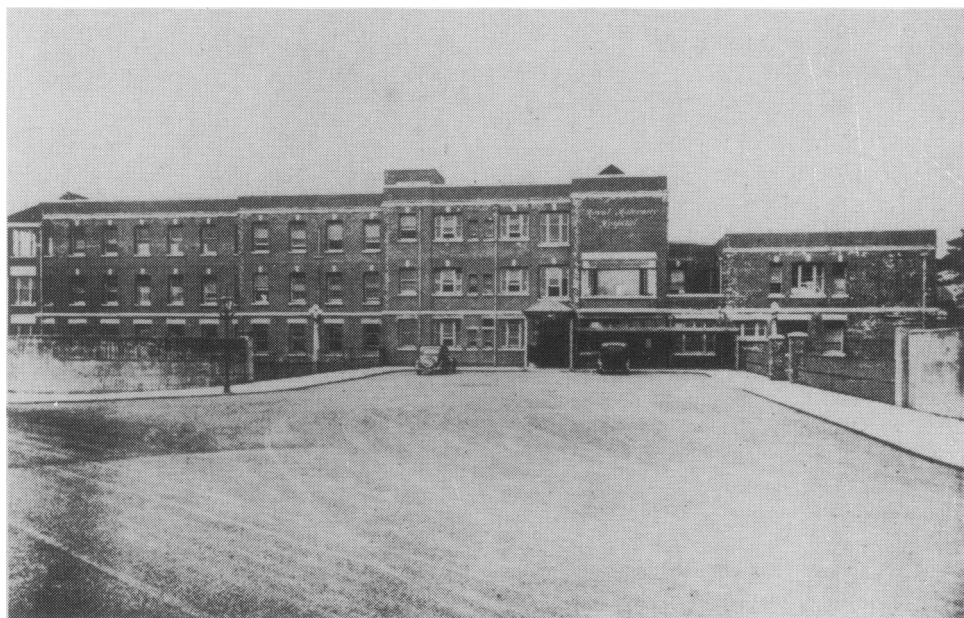


Fig 3. Royal Maternity Hospital, Belfast, in 1933.

The logo of the Royal Maternity Hospital, which some people seem desirous of eliminating for no good reason, is taken from the sculpture (Fig 4) in the main entrance hall by Miss Rosamund Praeger of Holywood, Co Down. The sculpture was commissioned by Mrs Maitland Beath in memory of her parents and daughter. The model for the sculpture was a Mrs Nancy Dowling who is holding her youngest son up high in her arms with another child at her feet. The sculpture was greatly admired by most. Mac was surprised, therefore, when Miles Phillips, an obstetric colleague from Sheffield, criticised the sculpture on the grounds that the lady should be facing the other way so as to show her left hand with a wedding ring and there should be two children at her feet — the ideal family being three. Miles Phillips commissioned Miss Praeger to do a sculpture for Sheffield with these altered specifications. Fig 5 shows what the Sheffield sculpture looks like. Note the ring and the additional children.

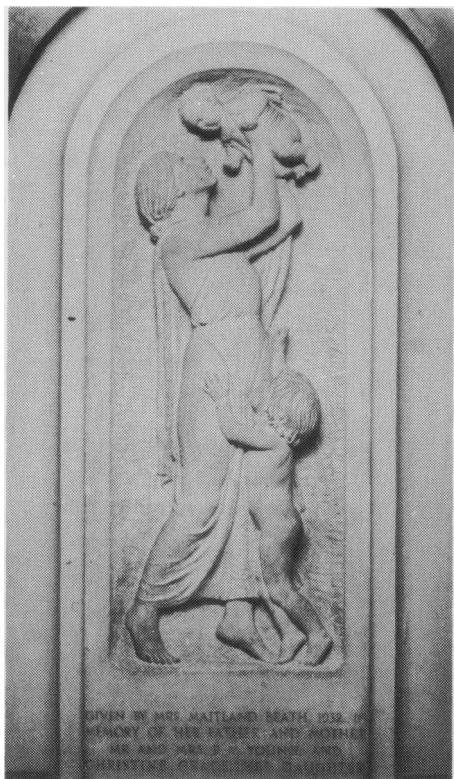


Fig 4. Sculpture in entrance hall of Royal Maternity Hospital by Miss Rosamund Praeger.



Fig 5. Bronze cast of sculpture by Miss Rosamund Praeger, commissioned by Mr Miles Phillips, Sheffield. This cast is presently in the Ulster Hospital, Dundonald.

Professor Sir Robert Johnston died in 1937 and the Chair of Obstetrics and Gynaecology reverted to one — Professor Lowry continuing in office. Mac became his Lecturer — the first in Obstetrics and Gynaecology at Queen's, a post he held until he himself was appointed to the Chair in 1945.

The 'Irish News' reporting on the 1937 Annual General Meeting of the Royal Maternity Hospital, quoted the new lecturer Mr Macafee as saying, "The mother is still the Cinderella of the Public Health Services". His reason for this remark was the proposed expenditure for maternity and child welfare of £12,542 compared with £39,540 for libraries and museums. He thought it scandalous to suggest placing books and antiques before mothers and babies, particularly when Belfast had higher mortality figures than most comparable centres.

What really upset Mac and his colleagues was that, in spite of the advent of asepsis and antisepsis, mothers continued to die from puerperal fever. The treatment of the haemolytic streptococcus was still basically glycerine, Guinness and God. However, that same year (1937) this dismal situation was, at last, to change with the introduction of Prontosil in clinical practice. The basis of Prontosil was that most outstanding chemotherapeutic agent of the century, sulphonamide. Most

patients with sepsis now survived — the fact that they turned pink for a considerable period of time was a small price to pay for life. However, the introduction of Prontosil did not affect the pneumococcus. Fortunately, May & Baker, after 692 experiments, found that sulphanilamide combined with pyridium, would kill the pneumococcus. Hence, the name 'M&B 693' for the drug introduced in 1939 which was to prove such a valuable chemotherapeutic agent in the War years.

The year 1940 saw the introduction of penicillin and the beginning of the antibiotic era which was to revolutionise the treatment of all infections and alter medicine dramatically. The penicillin story is best told by our own Sir Ian Fraser who had an important role in its introduction to clinical medicine during the Second World War.

Although commenced in 1937, it was during the War years that Mac did much of his original work on placenta praevia. Until that time, any patient admitted to hospital with an antepartum haemorrhage diagnosed as a placenta praevia was immediately delivered irrespective of fetal maturity, as it had always been believed that the mother could bleed to death. However, Mac was of the opinion that the first haemorrhage from a placenta praevia was not always a life-threatening obstetrical emergency. He believed that delivery could be postponed in many patients until the fetus was mature enough to survive, by simply putting them to bed in hospital until 38 weeks gestation. This 'expectant treatment' of placenta praevia revolutionised the outlook for mothers and babies. The maternal mortality was reduced from 26 to 5 per 1,000, and fetal loss from 500 to 12 per 1,000.

Mac, in his publication, acknowledged not only the obstetricians involved, but also the paediatrician Dr F M B Allen, later Professor of Child Health, and the nursing staff, for their interest and care of the babies, many of whom were very premature. He also paid tribute to all the patients who had stayed in hospital, some for many weeks, quoting from William Trotter's 'Collected Papers', "Let us not forget that they have borne more substantial witness than has yet been produced by any philosophers or theologians that all suffering is not in vain".

It is only when one appreciates the number of mothers and, in particular babies, that have been saved throughout the world since the introduction of 'expectant treatment', that one realises the magnitude of Mac's contribution to midwifery. The diagnosis of placenta praevia is now more accurate due to ultrasonics, and many patients who would have been kept in hospital 50 years ago may now be allowed home. Fetal mortality in placenta praevia also continues to improve, mainly because even if the patient has to be delivered prematurely, the enormous advances in the care of the premature infant by the neonatologists have virtually eliminated the vast majority of infant deaths.

Professor Lowry retired in 1945. The contenders for the post were the two lecturers, C H G Macafee and H I McClure. There was obviously a considerable amount of medical politics involved at the time as attempts were made to divide the Chair once more into Gynaecology and Obstetrics. This was refused by the Board of Curators and Mac was appointed full-time Professor with limited private practice.

At that time there was no academic clinical University departments as we know them today and the Institute of Clinical Science did not exist. However, by 1954,

mainly through the efforts of Professor Sir John Biggart, the then Dean of the Faculty, the Institute was built and the various clinical departments moved in. The relevant minutes of the Academic Council reveal that the move from the Queen's campus to the Grosvenor Road site was strongly opposed by many members, several of whom were Professors who had shown no previous affection for the Faculty of Medicine.

Almost concomitant with the establishment of this new accommodation for the University academic departments was the introduction of the National Health Service in 1948. Mac commented that the taking over of all the hospitals, which had largely been built with private monies charitably subscribed, was comparable to the rape of the monasteries by Henry VIII. He found it unusual to receive a salary for something he had done for nothing in the previous 22 years. He also thought that the Government had failed to realise how much work, both in general and consultant practice, had been carried out for nothing.

Now, 50 years on, we are faced with further reforms. Again, we are to receive payment for work that in the past was done for nothing. For example, there will be 'sessions' for administration, auditing, and many other activities, all of which were previously part of a vast amount of work done on a goodwill basis. If these 'sessions' encroach on time spent in clinical work, and I don't see how this can be avoided, then patient care will surely be affected unless additional medical and nursing staff are appointed. In the present financial climate this seems unlikely and, once again, much will depend upon the goodwill of the medical and nursing staff.

After 1948, more and more general practitioners ceased to practise obstetrics, referring their patients to hospitals which were no longer considered dangerous. Public demand for institutional confinement was such that by 1957, 70% of all deliveries were conducted in hospital — a complete reversal of the figures when the Royal Maternity Hospital opened in 1933. Now, the figure is virtually 100%. In the past few years several groups have been campaigning for a return to home confinements in 'normal' patients. I have one simple comment and that is — no labour can be considered normal until mother and baby have been safely delivered without any complication. *Normal labour is a retrospective phenomenon. Those who think differently should think again.*

The increasing demand for hospital confinements led to a recurrent shortage of beds. Many pleas for additional beds were made by Mac and his colleagues, but it was not until 1966 that the 50-bed extension opened, followed by the new labour suite in 1971, that is, 3 and 8 years respectively after Mac's retirement. Mac and his colleagues acknowledged the valuable part played by the late Mr Reginald Magee who headed the planning team.

One of the highlights of Mac's career was his appointment as Sims Black Travelling Professor of the Royal College in 1956. As such, he was to visit Rhodesia and South Africa. Unfortunately, after visiting Rhodesia he became ill and had to return home before going to Durban and Cape Town. He was so impressed with South Africa that, on his return, he advised those in the long queue for consultant posts to consider this option.

In 1958 the Royal Maternity Hospital celebrated its Silver Jubilee on this site. Lady Wakehurst planted a cherry tree to commemorate the occasion, and inspected a guard of honour. The tree is now well grown. In the same year, Malone Place Obstetric Unit was transferred to Jubilee III at the Belfast City Hospital, Mr H I McClure, having left to go to Musgrave Park. As a result, Mac was invited to join the staff, and the University's presence has continued in the Jubilee Maternity Hospital ever since.

Like all his predecessors, except Professor Burden, he was elected President of the Ulster Medical Society in 1958. I often wonder if this was why he chose the title 'Burden's Ghost' for his Presidential Address. During Mac's 18 years as Head of Department, in spite of many other duties, he continued his active role in clinical obstetrics and gynaecology. His Friday afternoon antenatal clinic attracted many complicated referrals, including patients with diabetes. Subsequently, in 1956, when Professor Desmond Montgomery of the Royal Victoria Hospital Metabolic Unit joined the clinic it became one of the world's first combined metabolic/antenatal clinics. Professor Montgomery was later honoured with a Fellowship *ad eundem* of the Royal College of Obstetricians and Gynaecologists for this work.

Mac was a very skilled operator and showed great patience when assisting juniors. His main interests were vulval and ovarian lesions, and the publication on 'The Pathology of Ovarian Tumours' in conjunction with Professor Sir John Biggart, was recognised as an authoritative work on the subject at that time. Those of us who had the privilege of working with him can remember several of his likes and dislikes and many of his sayings, a number of which have permeated down through the years.

Mac never liked husbands in the labour wards. I wonder what he would say today when husbands, partners, or whatever, are present at most deliveries. I expect that soon patients' lawyers will also attend. He also had very interesting views on infertility. His advice to newly married couples was always to go ahead and have a baby. When asked why, he used to say, "Just in case the ground should go fallow". On many a ward round when a student or postgraduate had taken the history and examined the patient but had omitted some relevant information, he would say "Did you ask her?" "Did you look?" The negative reply was followed by, "Ask and you will be told". "Look and you will see".

Just as his lasting memorial is undoubtedly placenta praevia, one of the best known of his sayings was "Let's sit on her". He often advised those uncertain what to do next about a patient, to practice "Watchful expectancy and masterful inactivity" if possible. He deplored "meddlesome midwifery".

Mac believed that "while medicine is undoubtedly a science, it is a science in which the scientist is dealing with people and not things". I'm sure he would have agreed with David Seegal who recently said, "The young physician today is so generously provided with a kit of diagnostic and therapeutic tools, his attention might be wisely directed to the question of 'what not to do' as well as 'what to do'. Like Osler, Mac also believed that "to study the phenomenon of disease without books is to sail an uncharted sea, while to study from books without patients is not to go to sea at all".



Fig 6. Charles Horner Greer Macafee, Professor of Midwifery and Gynaecology 1945–1963.

Sadly, in 1963, the Macafee era came to an end. After 42 years in medical practice, 40 of which were spent in obstetrics and gynaecology and 18 as Head of Department, Mac retired (Fig 6). His last operating session and the hospital tea party I recorded for posterity. There was also a grand function in the Great Hall of Queen's University at which his portrait, painted by Frank McKelvey, was presented by H I McClure on behalf of his colleagues.

Mac spent the first year of his retirement as Visiting Professor to the Hammer-smith Postgraduate Center in London. He was appointed Chairman of a Select Committee of the Council of the Royal College of Obstetricians and Gynaecologists to examine postgraduate training, and the quality of the 'Macafee Report' in 1966 on the subject is an example of his own ability and his expectations for others. I'm sure he would have been very proud to know

that at the present time Belfast is the largest postgraduate centre for obstetrics and gynaecology in the UK and that one of his protégés — Professor C R Whitfield now Regius Professor of Obstetrics and Gynaecology, University of Glasgow, is Chairman of the Higher Training Committee of the College. Another of our post-graduates, Dr Harith Lamki, is College Director of Postgraduate Studies.

As one would expect, Mac received many accolades. These included the CBE, an honorary DSc from Leeds University, and an honorary LLD from Queen's. There were many others, but the one which I think gave him most satisfaction was the unique honour of being the first person to receive, in the same year, the Blair-Bell Medal of the Royal Society of Medicine (the fourth ever awarded) and the Eardley Holland Medal of the College.

The death of his wife Margaret in 1968 — five years after he retired — was a heavy blow for him and the family circle but they, together with Miss Agnes Sey — better known as 'Nanny', who had been with the family from the beginning — provided him with companionship and care in the ensuing years. He moved from the family home in Donaghadee into the cottage at the rear, leaving the 'big house' for Alastair and his family. Always a keen gardener he continued to enjoy his lovely garden. He remained in contact with his friends and colleagues and regularly attended meetings of the Ulster Medical Society and the Ulster Obstetrical and Gynaecological Society. He was one of the founder members of the latter and its first President in 1952.

It was inevitable that the day would come when Mac would no longer be with us and, on 16th August 1978, in his eighty-first year, he passed away peacefully at

his home in Donaghadee. In a simple funeral service conducted by his great friend the late George Quinn, Bishop of Down and Dromore, we gave thanks for this man of deep Christian faith.

All who knew Mac will have their own memories. I personally remember that, no matter how busy, he listened patiently to the problems of everyone, and was ready to help in any way he could. His quiet manner when talking to patients was always reassuring, and his meticulous clinical examination of them was an example to us all. He had a most remarkable memory, undoubtedly due to his very genuine interest in his patients. Years later he could remember not only the face, the name and the medical condition of the patient, but he also delighted in recalling which bed she had occupied in the ward.

Generations of undergraduates and postgraduates, many of whom came from all over the world, particularly Africa and Australia, acknowledged without reservation that he was a great teacher and a distinguished medical pioneer. As a result, his name was known to obstetricians throughout the world. His personal contribution to the Belfast School of Obstetrics has been invaluable and, as a permanent tribute, Professor Pinkerton arranged the refurbishing of the old lecture theatre in the Royal Maternity Hospital and named it 'The Macafee Lecture Theatre'. Much of the funding for this was provided by one of Mac's former overseas postgraduates, Professor Tow Siang Hwa of Singapore.

Mac gave this Opening Address in 1942 on 'Medical Students and the Teaching of Midwifery'. He ended by saying, "Should the orator in a hundred years' time be a gynaecologist, I hope that he will be able to refer with pride to the contributions of the Belfast School to Medicine, and to the science and art of Obstetrics in particular". This morning we are about halfway and it is easy for me to refer with pride to the Royal Victoria and the other hospitals in the Royal Group. Their reputations as centres of excellence in medicine are worldwide as a result of the major contributions made by those who have worked and are still working in them. As main teaching hospitals they have, from their very beginnings, contributed to the fame of the Belfast Medical School, and I am confident they will continue to do so in the years ahead, irrespective of any changes.

Sir William Osler said, "Humanity owes a great debt of gratitude to those devoted men who have striven during their lives for exactness in knowledge and for practical application of such knowledge — a debt too great to pay, too great even to acknowledge". Although this may be true, I hope this address will in some small way help you to appreciate this most remarkable and distinguished man called 'Mac', a man who gave more than half-a-century of devoted service to mankind and, in so doing, championed the cause of midwifery — no longer a Cinderella. We will remember him.

No orator can produce an address entirely on his own and I would like to acknowledge the help I have had from our Archivist Dr John Logan, Norman McMullan and Ronald Wood of the Photographic Department and May Weller, without whose help there would be no oration. Last, but not least, I thank my wife and family for their support and tolerance over the past months.

Historical Review

The treatment of myxoedema with raw sheep thyroid gland and its introduction into practice in County Londonderry in 1892

Mary S T Logan, J S Logan

Doctor David Thompson¹ (1849–1936), of Fincairn and Feeny in County Londonderry, introduced the effective treatment of myxoedema to his neighbourhood in late 1892 or early 1893. He received the degree of BA from the Queen's University in Ireland in 1871, (Queen's College, Galway), and then qualified in medicine in 1873 (LRCP and S, LM, Edinburgh). In 1874 he was appointed dispensary doctor to the Feeny dispensary district. He held the post, and carried on his general practice, until 1928 when he was 79. Poor Law patients were seen at the dispensary, or, if too ill to attend, then in their homes. Other patients came to his family home in Fincairn. For many years he went about his rounds on horseback. It is said that he did the first cataract operation in that part of County Londonderry, on an old blind beggar woman. It was done on a kitchen table, with a local helper, one supposes to hold her head.



Doctor David Thompson of Feeny.

One day, when he was visiting a man with a broken leg, the neighbours asked him if he would speak to a poor woman who was dying. Not, they said, that anything could be done, because others of the family had died in exactly the same way. He went in to see her and found that she had advanced myxoedema. One wonders what form of familial myxoedema had afflicted the kinship, if indeed the family history was true. By a happy chance Thompson had recently read in his *British Medical Journal* (he was member of the Association) a report of the first

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successful treatment of myxoedema by the oral feeding of sheep thyroid gland. He at once obtained some thyroid gland from the slaughterhouse, presumably sheep's, and started her on it. She made a miraculous recovery. After that, until the dried preparation was commercially available, he always kept her supplied with fresh thyroid gland. When he was on holiday, he arranged for it to be got for her till he came home. What Thompson had read in his farmhouse home in Fincairn, that remote townland in County Londonderry, were the reports of H W G Mackenzie² of London and E L Fox³ of Plymouth in the *British Medical Journal* of the 29th October 1892.

Each had treated cases of myxoedema with great success by feeding fresh sheep thyroid gland by mouth. This was a great mercy, but it was not the beginning or end of a triumph of clinical and pathological observation. The sequence and progress of ideas and knowledge in these islands, over some thirty years, can be followed, as Thompson could, in the *British Medical Journal* of the times. It will hereafter in this text be called "The Journal".

No claim should be made for priority, but Gull's⁴ report in 1873 of two cases of "a cretinoid state supervening in adult life in women" had penetrated the consciousness of the medical profession. Gull's use of the word "cretinoid" was important, for Curling⁵ in 1850 had reported absence of the thyroid "body" in two cases of cretinism. Fagge⁶ in 1871 had shown that "sporadic cretinism" in England was not necessarily congenital and in his case there was wasting or absence of the thyroid gland. Ord⁷ in 1878 reported five cases of the cretinoid state of Gull and gave details of the postmortem findings in one. He noticed that the "thyroid alveoli" were mostly "annihilated". He gave myxoedema its name from the "jelly-like swelling of the connective tissue". "The cut surfaces yielded less fluid than their appearance would promise". As Halliburton⁸ said, it was "not the result of a watery dropsy". Ord thought it was mucinous, and temporarily that it was the swelling of the tissues had damaged the thyroid. An editorial⁹ in the *Journal* in 1882 showed that the clinical description was by then well known, the diagnosis was increasingly being made, and it was accepted that the thyroid gland was much diminished in size or absent. So did another editorial¹⁰ in 1863, but neither understood that the myxoedema was due to non-function of the thyroid gland.

Case reports in ones and twos began to be common enough in the *Journal*. Good examples are those of Oliver¹¹ and Gowans.¹² Less satisfactory but still useful is that of Heron.¹³ Coxwell¹⁴ described a case with onset at age 8. Various treatments were tried, tincture of jaborandi especially being thought to be helpful.¹⁵ Either doctors, apparently sensible and competent observers, were deceiving themselves (which is most likely), or jaborandi did do some limited good. (The botanical pharmacologists should have explored this suggestion of antimyxomatous activity in jaborandi). The myxoedematous insanity which confined a good many patients to a mental asylum was described,^{14, 16, 41} and the muscle disorder and weakness were repeatedly reported.^{11, 12, 17, 18} These features, now so seldom seen, were evident in the advanced, long-standing, untreated cases of the time. So was deafness,^{16, 18, 19} Loss of taste is mentioned.¹⁹ Myxoedematous patients lived as a rule for upwards of ten years before dying, so there was time for the disease to become advanced. In those days postmortem examination of an advanced untreated case was possible. The illustrated report of Hun and Prudden²⁰ was

valuable for the detail of the gross and the histological appearances. The residual gland tissue was remarkably cellular. This is hardly in accord with the frequent statement that the gland was replaced by fibrous tissue. Some women with myxoedema became pregnant, and one apparent case of remission associated with pregnancy may have been due to circulation in the mother of foetal thyroxine.²¹ Some reports suggested a haemorrhagic tendency in myxoedema.³¹ Gowans¹² thought his patient was going to die of the prolonged severe haemorrhage when he extracted a loose molar tooth. Tayler's patient had menorrhagia.¹⁸ The menorrhagia of the patient of Bettencourt and Servano ceased after she received an implant of sheep thyroid gland.²² These are brief and not substantial observations, but may deserve thought.

The notion that myxoedema was a form of chronic renal disease (Bright's disease) lingered for longer than it should have, but careful doctors tested the urine for protein and found none except in the terminal stage. An acute clinical observer, Doctor Clunn of Prestwich, reported a case resembling myxoedema in a woman following severe post-partum haemorrhage.²³ No doubt it was a case of anterior pituitary necrosis of Sheehan type, but the description and lesson in half a dozen lines is brilliant. Probably Sheehan's syndrome, acromegaly and renal disease did give some difficulty in diagnosis to some doctors, in the knowledge of the time.

Semon²⁴ in the *Journal* in December 1883 pointed out that E T Kocher²⁵ of Bern had produced a syndrome closely resembling myxoedema by "totally" resecting the thyroid gland. Kocher cannot have followed his patients' postoperative course closely, because he was astonished when the patients' local doctors drew his attention to what was happening. It took time after thyroidectomy for myxoedema to manifest itself. It follows of necessity that these cases had not experienced tetany or been disabled or died of it, and so the "extirpation" can only have been partial and the parathyroids spared. An almost immediate and continuing complication of a truly total operation was tetany. It is certain that in these cases the parathyroid glands had been removed. Of course the calcitonin-secreting cells of the thyroid gland had also gone. Evidently the thyroid surgeons of the time did not know of the existence and importance of the parathyroid glands, although Sandström's paper (in Swedish) on the existence, anatomy and histology of the parathyroid glands was published in 1880.²⁶ In any case they did not know of their function. That had to wait for some years. There were, however, many descriptions of "accessory thyroid glands" and one suspects these may have been the parathyroids. In his monograph von Eiselsberg²⁷ uses the word Nebenschilddrüse to mean accessory thyroid gland. In later years, especially in the plural, it came to mean the parathyroid glands.²⁸

Kocher and Ord compared their observations²⁹ and were satisfied that the naturally occurring myxoedema and the operative myxoedema were the same. They seem to have disregarded for the time being the tetany of some of the operated cases. In 1885 Horsley produced myxoedema in monkeys by total thyroidectomy.³⁰ Like Kocher he produced tremor and tetany in some, presumably again by removing the parathyroid glands, but the tetany in Horsley's monkeys was not so severe as had occurred in dogs. Horsley noticed that myxoedematous monkeys, though cold, did not shiver. He usefully reviewed the European literature. In 1888 the Clinical Society of London published an exhaustive review of myxoedema, operative and natural, and of cretinism, sporadic and

endemic.³¹ The investigative committee had been chaired by Ord. Henceforth the Society and the profession generally were satisfied that myxoedema was a consequence of atrophy of the thyroid gland, the cause of that remaining unknown. This ultimately provided ground for rational treatment of the disease. Moreover it was clear that total resection of the thyroid gland for simple goitre could no longer be practised. And it was later to be clear that thyroidectomy, total or partial, must not include removal of the parathyroid glands. It is fair to remember Munk³² who, almost alone, thought it was not thyroid deficiency that caused tetany. He seems to have produced ischaemic necrosis and denervation of the thyroid with ligatures. If the wound did not become infected, there was no tetany. Few agreed with him though he was right.

In 1890 Horsley³³ reviewed in the *Journal* experimental animal thyroidectomy by European workers, Schiff³⁴ and von Eiselsberg.²⁷ Both were preoccupied by the postoperative tetany, which killed most of the animals shortly after the operation, and which they attributed to thyroid deficiency. They had of course removed the parathyroid glands with the thyroid. Schiff had some limited success in preventing, in the short term, the tetany in his dogs by implanting beforehand a thyroid gland of another dog in the peritoneal cavity. The good effect must have been due to the unwitting associated parathyroid implant.

In only one case did he think an intraperitoneal implanted gland was "conserved and attached to the peritoneum". The rest was absorbed. Von Eiselsberg had many failures, even when he first removed one half of the thyroid, implanted that in the abdomen, and later removed the other half of the thyroid. After such procedures, however, two cats remained well. In one, after a month's good survival, postmortem examination showed the implant to be "organised, normally vascularised and with no signs of degeneration". In another, which had had no tetany or myxoedema, postmortem at three and a half months showed the implant well vascularised. He seems further to say that when the two implants, which had survived so well, were removed at a third operation, tetany occurred. This being so, parathyroid glands must have been included in the graft and none left in the neck. Horsley,³³ basing himself on this work and on his own, not realising any more than Schiff and von Eiselsberg the irrelevance of the tetany to thyroid deficiency, advised trying an implant of sheep thyroid gland in "natural" myxoedema in humans. In 1889 Bircher³⁵ in Aarau had already transplanted into the abdominal cavity of a "totally" thyroidectomised female patient a piece of "apparently normal thyroid tissue" from a goitre. There was genuine but transient improvement. Bettencourt and Serrano²² in 1890 had transplanted sheep thyroid gland beneath the skin of the chest of a myxoedematous woman, with "immediate" improvement. The onset of the improvement was so rapid that they percipiently ascribed it to absorption of thyroid gland "juice" from the graft, and not to the graft truly functioning. Vassale³⁶ in 1890 and Gley³⁷ in 1891 had tried intravenous injection in animals of an extract of "thyroid" gland soon after total thyroidectomy, "with beneficial results". These results were to abolish the tetany. The injections were given very soon after operation, before myxoedema could develop, and, unknown to them, must have included parathyroid hormone. They thought they were treating thyroid deficiency. However, the successes of the experimental transplants and injections, transient, imperfect, and not properly understood though they were, made men think that extracts of thyroid glands in some form would be effective in myxoedema. It was, after all, the age of organotherapy.

In the *Journal* in October 1891, Murray^{38, 39} of Newcastle on Tyne reported that he had made an extract of sheep thyroid gland, and had injected it subcutaneously in his patient with naturally occurring myxoedema. Striking improvement had followed. He cut up the gland into small pieces and put them in a test tube with one ml of pure glycerine and one ml of an 0·5 per cent solution of carbolic acid in boiled distilled water. The mouth of the tube was closed with a plug of cotton wool, and the mixture stood in a cool place for twenty-four hours. It was then strained through a fine handkerchief, which had previously been placed for a few minutes in boiling water. Three millilitres of a turbid, pink fluid was obtained. One and a half millilitre was injected twice a week subcutaneously and the patient improved remarkably. Others easily confirmed Murray's findings. It was no humbug. No other clinical trial was necessary to establish the principle. In the same issue of the *Journal*, Fenwick⁴⁰ reported his similar success with the injection of "ten drops of juice" of sheep thyroid gland. Possibly, indeed probably, Murray's injection included an extract of the parathyroid glands, but, if it did, it does not seem to have done obvious harm. The injection however was inconvenient, the site sometimes became septic and adverse reactions occurred. In the *Journal* for the 29th October 1892, the issue Thompson of Feeny read, Mackenzie² and Fox³ each reported the successful treatment of their patients by feeding with whole fresh thyroid gland. Fox had first given Murray's glycerine extract of thyroid gland by mouth, and, finding it worked, then the whole gland. Thenceforth the treatment of myxoedema was effective and relatively simple, though the preparations of the gland had to be developed and improved, and later standardised. Indeed there was trouble with overdosage and some deaths were attributed to that.

Some reports of the excellent effect of the new treatment came from mental asylums,^{16, 41} where patients had been confined because of the "myxoedematous madness". That must have been the first example of successful treatment of a chronic organic dementia.

One interesting report said that the new treatment diminished the size of a simple goitre. It worked well in cretinism. Whitla in Belfast tried it in exophthalmic goitre and found it made the patient worse! It was no good in psoriasis, for which there had been hopes. It should not perhaps have been surprising that the anti-myxoedematous principle of the thyroid gland is absorbed readily from the alimentary tract, and functions after absorption. The gland develops embryologically from a diverticulum of the foregut, and presumably in very early evolutionary times discharged its secretion through its duct into the alimentary tract.

Two preparations of sheep thyroid gland became official for the first time in the British Pharmacopoeia of 1898. These were Thyroideum Siccum or Dry Thyroid, and Liquor Thyroidei or Thyroid Solution. The Dry Thyroid monograph required the glands to be taken from the sheep immediately after killing, cleaned, cut up, inspected for apparent normal appearance, minced, dried at a temperature of 90 degrees to 100 degrees F., powdered, defatted and dried again. Dose of the powder was 3 to 10 grains. Monographs in subsequent editions varied the directions, but the important advance was the standardising of the powder. The British Pharmacopoeia of 1932 required the powder to contain 0·1 per cent of iodine in combination as thyroxine. The direction for standardising varied in subsequent editions but was retained to the end. The BP 1932 also allowed the preparation

to be made from ox and pig thyroid as well as from sheep. It had long been available commercially as a tablet, and commercially it had not always been made from sheep thyroid before 1932. Moreover Martindale's Extra-Pharmacopoeia, 20th edition, volume 1, stated that "thyroid preparations on the market (mostly made from sheep thyroid) are obtained from parathyroid as well as thyroid tissues". Lactose was the diluent and it was said that this inhibited some of the hormonal effect. Presumably some calcitonin was contained in Thyroid, as it was latterly called. Perhaps the process of heating to dry the gland, for powdering, and to evaporate the defatting agent, had an antibacterial effect.

But it seems likely that Thyroid might have conveyed animal infections to patients, and one would think the original raw thyroid tissue certainly did. It was not until the BP 1973 that the monograph required 1 Gram to be free from *E coli*, and 10 Grams to be free from salmonellae. After eighty-two years of being official, Thyroid was omitted from the British Pharmacopoeia of 1980. The BP 1988 contains only Thyroxine Sodium (European Pharmacopoeia Levothyroxine sodium) and Liothyronine Sodium. Thyroid preparations first appeared in the famous Belfast book *Pharmacy, Materia Medica and Therapeutics* by Sir William Whitla in the seventh edition of 1898.⁴² This was cautious of Sir William, but of course he had delayed its publication to follow the BP of that year. The treatment of myxoedema now rests with thyroxine. Even that may not be the end of the story.

David Thompson was the son of John Thompson of Fincairn and Jane Ritchie of Straid, not far away. He began attending the medical classes of The Queen's College, Belfast, in 1869, when he was 20, and the classes of the Belfast General Hospital (now the Royal Victoria Hospital) in Frederick Street in the winter of 1870. He had a happy, useful, honourable life, living with his unmarried brother and two unmarried sisters on the family farm, his parents having moved from the adjacent clachan. A keen gardener, croquet player and churchman, a shareholder in the Dungiven to Limavady railway (which never paid dividends), he was contented with an occasional holiday in Harrogate. He is buried in the kirkyard of Banagher church where his grave bears the superscription *Ardens sed Virens*.

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The Royal Medical Benevolent Fund Society of Ireland

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Succeeding generations of medical practitioners have tended to complain about their lot. In more recent years the complaints about terms of service have been directed at the administrators of the system originally devised by politicians with the acquiescence of a reluctant profession. These complaints are more concerned with the small print refinements in an already well tried system. Looking back to the early 19th century, the conditions under which general practitioners toiled can be envisaged only with difficulty. At that time their main problems were those of survival and of establishing a means of sustenance for their families. Insurance, although available, was not widely used, in part due to the additional expense and there was a perceived need for some form of benevolent fund for dispensary doctors and their families.

The purpose of this article is to review and reflect on the history of the Royal Medical Benevolent Fund Society of Ireland.

The dispensary system had originated in 1805 when Grand Juries and Infirmary Authorities in Ireland were authorised to establish dispensaries. The subsequent dispensaries “were too few, without proper organisation and supervision, and attended by doctors who were poorly paid for work which was undertaken even at the cost of their lives”.¹ Clearly the system did not work satisfactorily and in 1842 Sir George Nicholls, the Irish Poor Law Administrator, drafted a new bill which was to increase the number of dispensaries and bring them under the Commissioner’s control. It is recorded that the dictatorial Administrators were disliked by both medical and laymen, which creates now a feeling of *dèja vu*.

An attempt was made by the senior representatives of the profession in Ireland to improve the lot of the dispensary doctor, who was later to evolve into the general practitioner of the twentieth century. Such was the strength of feeling that Dr William Stokes — subsequently President of The College of Physicians of Ireland, accompanied by Samuel Cusack, a prominent surgeon, went to Westminster to give evidence to members of the House of Commons. Figures were produced which showed that in the previous twenty-five years no less than 24 % of Irish medical practitioners had died in the discharge of their duties, mainly of typhus and cholera. This figure was more than twice the mortality of army officers in combat.

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Stokes' portrayal of the Irish Dispensary Doctor was a dismal picture. "The Irish Physician is often exposed to contagion in its most concentrated force when himself, under the influence of cold, wet, fatigue and hunger, as he labours among the poor, passes from hovel to hovel in wild and thinly populated but extensive districts. He has often to ride for many hours in the worst weather and at night, enduring great fatigue while himself a prey to mental and to physical suffering; for if we add to such labour the injurious influence which the knowledge of danger must have on the system of a man feeling that he is struck down by the disease under which he has seen so many sick, and tortured by the thought of leaving a young family unprovided for, we can understand how it happens that the country is so often deprived by death, of so many of its best educated and most devoted servants".¹ Members of the House of Commons can hardly have remained unmoved by such an eulogy.

The second initiative taken by the senior representatives to improve the situation was to hold "A memorable Great meeting of the Physicians and Surgeons in Ireland".¹ Such a meeting was held in the College of Surgeons in Dublin on 29 May 1830 and brought together representatives of the College of Physicians, the Surgeons, Apothecaries and the University. Like so many things in Ireland before and since, agreement could not be reached on the main aims of the meeting. However, there flowed from the meeting a number of resolutions on which action was subsequently taken. Three effects of lasting importance resulted from some seventeen resolutions passed that day. One was to lead to the formation of district medical associations, later to become the Irish Medical Association. Another resolution was to standardise medical education and practice eventually evolved as the Medical Act of 1858, enabling the setting up of a medical register and General Medical Council in Ireland. A further resolution was "that the establishment of a relief and widows fund be recommended to the attention of the Council".

Some twelve years later on 26 May 1842 a Dr Kingsley of Roscrea set up a benevolent fund which he called "the Medical Benevolent Fund Society of Ireland".² This had a Central Council in Dublin with branches in main cities throughout the country. A Belfast and District Branch was formed at the suggestion of Dr James Sanders in February 1843, "for the relief of medical men, under severe and urgent distress, occasioned by sickness, accident or any other calamity".³ Its charter further stated that "Under circumstances of peculiar emergency, relief may also be afforded to the Widows and Orphans of Medical Practitioners". The Belfast branch comprised practitioners in Belfast, Co Antrim and Co Down. Dr Sanders typified the necessity for the Society in that he was in very active practice for eleven years, but contracted tuberculosis and died at the age of 32 years.

The Minutes of the Board of Management of the Fever Hospital in Frederick Street, Belfast, dated 11 February 1843 record that "Resolved that leave be granted to Dr Sanders to hold a meeting of medical practitioners in the Library for the purpose of establishing a branch of the Medical Benevolent Fund lately formed in Dublin". The Society thrived but not without some difficulty. Minutes of the Society show that quarterly meetings were held at first in the library of the General Hospital and subsequently in the Ulster Medical Society Room at 33 High Street. It had a President in the person of Dr Thomas Henry Purdon of

5 Wellington Place. Dr Purdon held the posts variously of Medical Attendant to the County Gaol and the managed Asylum, Senior Surgeon to the General Hospital, and Medical Attendant to the Infirmary and the Poorhouse.⁴ The early minutes of the Branch show that Dr Purdon was appointed "President for Life", a rather unusual appointment.

Obtaining voluntary subscriptions appears to have held the same difficulties for the office bearers than as it does now. Downpatrick and Banbridge were barren areas requiring "a deputation on the part of the Society with a view to obtaining additional subscriptions".⁵ Mid Antrim did not require a deputation in spite of its Scots roots. The net was cast wider to "bring the claims of the Society under the notice of the principal nobility, gentry and Members of Parliament of this Branch for their support". Some support did come from such figures as Lord and Lady Antrim, The Reverend O'Neill of Shanes Castle and some city merchants. Subscriptions of one guinea or more allowed the member to put forward names for consideration for benefit.

In 1860 the Belfast branch of the Society reported that it had collected £113.2s.6d. for the year. This sum was transmitted by the Treasurer, Dr Browne, JP, RN, to the Dublin Society headquarters for consolidation, after £2.11s.6d. had been held over for "contingency expenses". If the sums of the subscriptions were not large, neither were the annual distributions. Five widows and an elderly surgeon received sums of £16.0s.0d. for that year, at a total outlay of £57.0s.0d. by the Society.

For practical purposes medical practice as we know it now began in the United Kingdom with the Medical Education Act of 1858. This Act set up the Council of Medical Education and Registration (subsequently modified to the General Medical Council). Prior to this the practice of medicine was not clearly controlled. In 1860 the Belfast branch of the Society received an application from Mr Christy "a Doctor of Midwifery" from Co Antrim. At a subsequent meeting in 1861 the minutes show "the widow of the Mr Christy "Doctor of Midwifery" had sent forward a filled up form for relief to the Committee (her husband having died soon after his own application to this branch in November 1859) but who after due consideration and enquiry had decided no sufficient proof had been afforded that her late husband was a qualified member of the profession".⁶ Sadly this application was refused and the widow Christy's fate was unrecorded. This case does indicate the drawing to an end of the unqualified medical practitioner era.

By 1900, grants amounted to £80.0s.0d. while the Society subscription had contracted to £66.0s.0d. With the backing of the Chairman, Sir William Whitla, this figure of contribution was trebled in three years to £176.2s.6d.

A form of Royal recognition was announced in February of 1861. "The parent Society has now the honour of the prefix 'Royal' being permitted to be used. Her Majesty, who with the Prince Regent, presented a donation of £100 to the Society last year, having been graciously pleased to grant leave for this distinguished privilege being conceded to it".⁷

Many of the prominent physicians in Belfast have held the Chairmanship of the Society, including Sir William Whitla, Professor Symington, Doctors Walton Browne, Henry Whitaker and Henry S Ferguson. The circumstance of the beneficiaries mainly appear to be widows with young children, of doctors who

died unexpectedly early, and the widows of doctors who had survived to an advanced age, far outstripping what savings were left by their spouse.

Reading the early minutes of this Society brings forth one further thought. Education for doctors is based now on science and not on the arts. Something is thereby lost in accuracy of expression which was an integral part of the previous century's practitioners, even if it was not complimented with a precision of diagnosis. The use of words and the beautiful calligraphy are but a faded shadow of what once belonged to a more literate if less technically adept generation.

The Royal Medical Benevolent Fund of Ireland now exists throughout Ireland, and runs in parallel with the Royal Medical Benevolent Fund. The latter was founded in 1836 by the same group of doctors who founded the British Medical Association. The objectives of both Benevolent Funds are identical, the relief of medical practitioners and their families who are under severe and urgent distress occasioned by sickness, accident or other calamity.

By the nature of their work neither of the local organising secretaries are able to be specific, but it is possible to state that there are 10–12 beneficiaries in Northern Ireland at present who have received grants totalling some £20,000 from the Royal Medical Benevolent Fund and about £7,000 from the Royal Medical Benevolent Fund Society of Ireland. Subscriptions from Northern Ireland doctors to the two societies are much less than the benefit given — about £3,000 per year to the Royal Medical Benevolent Fund and about £1,000 to the Royal Medical Benevolent Fund Society of Ireland.

In this enlightened age one might expect to find that such organisations are superfluous. However, sadly this is not so. Calamity still visits medical families when financially vulnerable, leading to distress frequently involving younger members of the profession and their children. It should be no great sacrifice to most practising doctors to make an annual contribution to one or other Society by contacting either:

| | | |
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| Royal Medical Benevolent Fund c/o Mrs Lesley Donaldson 19 Cabinhill Gardens Belfast BT5 7AP. | or | Royal Medical Benevolent Fund Society of Ireland c/o Dr David McLean University Health Service Queen's University, 25 University Square Belfast BT7 1PB. |
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Case report

Central pontine myelinolysis without hyponatraemia

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Central pontine myelinolysis is an acquired demyelinating disorder of uncertain origin that predominantly affects the centre of the basilar portion of the pons. Several theories have been proposed to account for this condition, prominent among them being abnormalities in serum sodium concentration. We report on a patient who had central pontine myelinolysis with extrapontine lesions and Wernicke's encephalopathy but who maintained a normal or high serum sodium concentration throughout his hospital admission.

CASE REPORT. A 52-year-old man was admitted to hospital following two tonic-clonic seizures. There was a long history of excessive alcohol consumption of more than 100 units per week. One week prior to admission he had stopped drinking alcohol because he had become increasingly confused. He was on no regular medication. There was no history of trauma or epilepsy. On examination he was afebrile, anicteric and not clinically dehydrated. He scored 11 on the Glasgow Coma scale.¹ There were no abnormal neurological signs in the cranial nerves. Tone in the limbs was flaccid with hyporeflexia and flexor plantar responses. Sensation was intact. He became agitated and was commenced on multiple vitamin supplements and 40 mg chlordiazepoxide on a decreasing regimen. Initial investigations showed serum sodium 139 mmol/l, potassium 2.9 mmol/l, glucose 6.7 mmol/l and urea 6.9 mmol/l. Liver function tests were normal apart from serum gamma-glutamyl transferase 236 U/l (usual range 7–46).

Despite an adequate oral fluid intake he became more confused. He was disorientated in place and time and was unable to recognise his relatives. There was no asterixis or hepatic fetor. Serum ammonia was 57 μ mol/l (13–52). Electroencephalography showed a mild generalised abnormality of a non-specific nature. CT scan of the brain revealed no evidence of intracranial haemorrhage or central pontine myelinolysis. Magnetic resonance imaging was not performed.

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He remained confused and on the seventh hospital day developed a right basal pneumonia. Despite intravenous antibiotics he continued to deteriorate and died seven days later from cardiac arrest.

Throughout the 14 days his serum sodium concentration was measured daily or every second day and gradually rose from 139 mmol/l to 153 mmol/l. Liver enzymes remained approximately constant.

At necropsy the liver (1420 g) showed micronodular cirrhosis with fatty change and Mallory's hyaline deposits in the hepatocytes. Coronal slicing of the brain (1414 g) revealed no macroscopic haemorrhagic necrosis or atrophy of the mamillary bodies. Histologically the mamillary bodies and periventricular gray matter showed characteristic changes of Wernicke's encephalopathy with capillary prominence, ring haemorrhages, astrocytic gliosis and foamy macrophages. In the cerebellum there was atrophy of the anterior superior vermis with widespread Purkinje cell loss, proliferation of Bergman astrocytes, mild granular cell loss and isomorphic gliosis in the molecular layer. The pons on horizontal sectioning showed a roughly triangular area of granular softening disposed symmetrically in the centre of the basis pontis (Fig 1). Sections from this area showed clearly defined incomplete demyelination, mainly involving the transverse ponto-cerebellar fibres: there was a total loss of oligodendrocytes with both focal macrophage and reactive astrocyte infiltration. The tegmentum was not involved. Foci of myelinolysis were also seen throughout the subcortical white matter in the frontal, temporal and parietal areas and in the internal capsule. Electron microscopy of the edges of these subcortical lesions revealed many myelin sheaths undergoing vesicular-tubular myelinolysis (Fig 2) and acute necrosis of some accompanying oligodendroglia.



Fig 1 (above). Transverse section of midpons showing central area of demyelination.



Fig 2 (right). Electron micrograph (X 20,500) showing vesicular-tubular myelin breakdown in a subcortical white matter lesion.

DISCUSSION

This patient presented with clinical features of central pontine myelinolysis, namely, an acute confusional state, seizures, flaccid quadriplegia and a history

of chronic alcohol use. Wernicke's encephalopathy does not completely explain his mental confusion as the confusion progressed despite rapidly administered intravenous thiamine. Also seizures and flaccid quadriplegia are not features of Wernicke's encephalopathy.² The clinical spectrum of central pontine myelinolysis includes coma, the "locked-in" syndrome, seizures, facial weakness, pseudobulbar palsy, quadriplegia and behavioural changes without focal signs.^{3, 4, 5} The rapid neurological deterioration in this case is typical of central myelinolysis. In other cases reported most of the demyelinating lesions have evolved in two to four weeks.⁴

There is no diagnostic test for central pontine myelinolysis. The CT scan may be normal as in this case, or may show a non-enhancing hypodense lesion in the ventral pons.⁶ Magnetic resonance imaging is more sensitive than CT in visualising the pontine lesion but may also be normal early in the course of the disease.⁷ Few cases of central pontine myelinolysis are diagnosed in life probably because the clinical manifestations are obscured by other neurological abnormalities, or are absent if the lesion is small.^{3, 5}

The initial reports of central pontine myelinolysis coincided with the first widespread use of intravenous therapy to treat electrolyte abnormalities.⁸ In particular central pontine myelinolysis has been reported as developing when a low serum sodium concentration is increased at a rate greater than 12 mmol/l/day.⁹ However, other authors argue that rapid correction of hyponatraemia in itself is not dangerous, provided that hypernatraemia does not develop during the first 48 hours of therapy.¹⁰ Hyponatraemia alone has been blamed for producing the condition.¹¹ In animals pontine and extra-pontine myelinolysis can be induced more easily by rapid correction of chronic than of acute hyponatraemia.¹² The rapid correction of chronic hyponatraemia results in dehydration whereas rapid correction of acute hyponatraemia reduces to normal the levels of water in the brain. This rapid change in the levels of water in the brain is thought to be an essential component in the pathogenesis of central pontine myelinolysis.

Alteration in serum sodium concentration cannot be excluded as an aetiological factor in our patient, as it may have occurred prior to his hospital admission. However, many recently reported series of central pontine myelinolysis have included cases with normal serum sodium. McKee and colleagues found the characteristic pontine and extra-pontine lesions in ten patients who died from severe burns,⁵ and hyponatraemia was not present in any of these patients.

Central pontine myelinolysis is invariably associated with some other serious, often life-threatening disease. The condition was initially described in 1959 in alcoholic and malnourished individuals with liver disease.⁴ In more than half the cases it appeared in the late stages of chronic alcoholism, often co-existing with Wernicke's encephalopathy. Other frequently noted associations are patients with hepatic cirrhosis, Wilson's disease,¹³ chronic renal failure, severe burns,⁵ lymphoma, carcinoma, leukaemia,¹⁴ acute haemorrhagic pancreatitis and as a complication of renal¹⁵ or liver¹⁶ transplantation. These disorders may result in abnormalities of astrocyte metabolism with a reduced ability to generate new intracellular anions in response to osmotic change.¹⁷ Demyelination may then develop when a rapid change in serum osmolality results in osmotic endothelial injury and opening of the blood-brain barrier. This would allow entry of myelinotoxic factors from the bloodstream and also generate vasogenic cerebral oedema,

a known precipitant of demyelination. The rapid change in serum osmolality may be due to anions other than sodium, or to uraemia or hyperglycaemia. Individual variation in the reaction of central nervous tissue to similar degrees of osmotic stress may determine susceptibility. At present this is speculative but until these mechanisms are elucidated it will not be possible to predict who might develop central pontine myelinolysis.

It is important to consider the possibility of central pontine myelinolysis in any severely ill patient who develops an unexplained behavioural disorder with or without neurological signs. There is no specific treatment but major fluctuations in serum osmolality should be avoided as clinical and radiological recovery from central pontine myelinolysis is possible.^{7, 18}

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Case report

Prenatal diagnosis of cystic adenomatoid malformation of the lung in a twin pregnancy

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Most pregnant women in the United Kingdom will have at least one antenatal ultrasound examination¹ and many women will have several scans performed. This widespread use of diagnostic ultrasound during pregnancy and the continually improving quality of ultrasound equipment has resulted in the increased detection of unsuspected fetal malformations. In some cases, particularly when cysts in body cavities or minor degrees of dilatation of the urinary collecting system are seen, the exact diagnosis and prognosis may be uncertain. This often results in considerable anxiety of mothers and other family members and may lead to dilemmas about management. This report describes the management of a twin pregnancy in which a large intrathoracic cystic lesion was detected antenatally in one of the fetuses.

CASE REPORT. A 24-year-old woman, gravida three, para two, was found on routine ultrasound examination at her first antenatal visit to have a twin pregnancy of approximately 23 weeks gestation. No fetal abnormality was apparent on that occasion. At 27 weeks gestation, she complained of abdominal pain and ultrasound examination revealed polyhydramnios. Both fetuses were appropriately grown for gestational age, but a cystic lesion 4 × 4 × 3 cm was noted in the thorax of the second twin (Fig 1). The mass appeared unilocular, and was situated mainly in the right side of the chest, extending posterior and superior to the heart (Fig 2). The stomach was identified normally placed in the abdomen and the diaphragm appeared to be intact. These findings excluded a congenital diaphragmatic hernia and the most likely diagnosis was thought to be a cystic lesion of the lung. No other congenital malformation was detected.

The patient was admitted to hospital for rest. She was treated with oral ritodrine to relax the uterus and prevent preterm labour, and with weekly courses of intramuscular corticosteroid to improve fetal lung maturity. The problem was discussed with a variety of experts (perinatologist, geneticist, neonatal paediatricians and paediatric surgeon). It was decided not to perform amniocentesis or cordocentesis for chromosome studies because the pregnancy was multiple. It was thought that *in utero* aspiration of the lesion would be unwise as its exact nature was unknown. It was planned to deliver the patient by elective caesarean section in the Royal Maternity Hospital when appropriate.

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Fig 1. Longitudinal ultrasound scan of fetal thorax (Twin II) at 33 weeks gestation showing large sonolucent intrathoracic cyst.



Fig 2. Transverse ultrasound scan of fetal thorax (Twin II) at 33 weeks gestation showing cystic lesion posterior to the heart.

The patient rested in hospital until 34 weeks gestation when she had recurrent episodes of uterine contractions and was transferred to the Royal Maternity Hospital. Serial ultrasound examinations showed continuing polyhydramnios, satisfactory growth of both fetuses, and gradual enlargement of the cystic lesion to 4 × 4 × 7 cm. Elective caesarean section was performed at 35 weeks gestation, this time being chosen to avoid emergency delivery as there was increasing uterine irritability and maternal anxiety, and the fetuses were now reasonably mature.

Twin one was a healthy baby girl weighing 2240 grams who had no subsequent problems. Twin two, a baby girl weighing 2395 grams, did not establish spontaneous respiration until five minutes old and remained cyanosed despite 100% oxygen given by face mask. Intermittent positive pressure ventilation was commenced. Chest X-ray confirmed the presence of a cystic lesion in the right side of the chest with mediastinal shift towards the left but was otherwise normal. The baby was transferred to the Infant Surgical Unit and underwent thoracotomy when aged three days. The middle and lower lobes of the right lung were replaced by a cystic mass and were removed. The baby has made a good recovery. The diagnosis of cystic adenomatoid malformation was confirmed histologically.

DISCUSSION

Intrathoracic extracardiac malformations are usually extrinsic, such as congenital diaphragmatic hernia which occurs in approximately 1 in 2000 births.² Intrinsic intrathoracic lesions are relatively rare. They include bronchogenic cysts, congenital cystic adenomatoid malformation of the lung, bronchopulmonary sequestration, primary pulmonary hypoplasia, chylothorax, idiopathic hydrothorax and mediastinal masses. Accurate prenatal diagnosis may be difficult.³ A careful search should be made for the presence of other malformations which may be associated with some of the above conditions and consideration given to excluding possible chromosomal abnormalities.

Cystic adenomatoid malformation is a hamartomatous lesion of the lung. Stocker et al (1977) described three types,⁴ on several criteria but basically on the size of the cysts. All three are characterised by a proliferation of structures resembling terminal bronchioles, increased elastic tissue and polypoid columnar or cuboidal epithelial proliferation. Type I, found in the case reported here, consists of multiple large cysts up to 7 cm in diameter and has the best survival rate. Types II and III are composed of multiple small cysts and may appear solid and hyperechoic on ultrasound. Type II lesions may be found in association with other malformations and have a poor prognosis.⁴ Type III lesions are rare, often very large, and have a poor prognosis.

Detection of congenital malformations during prenatal life is important so that management may be planned to ensure the best possible outcome. Large intrathoracic masses may be associated with fetal hydrops secondary to obstruction of the venous return to the heart and pulmonary hypoplasia secondary to compression.⁵ At delivery, severe respiratory distress is likely because of pulmonary compression, pulmonary hypoplasia, prematurity, or any combination of these, and the baby is likely to require major surgery within a few hours or days of birth. For all of these reasons, an experienced paediatrician should be present at

the birth, and delivery should be in a unit with neonatal intensive care facilities, ideally close to an infant surgical unit. Discussion beforehand with everyone involved in the management of the patient allows delivery to be planned at the most suitable time. The method of delivery will depend on the individual circumstances. In the case described, caesarean section was performed because of breech presentation of the second, abnormal twin.

Space-occupying lesions in the fetal thorax, if large, are liable to cause respiratory embarrassment at delivery. Modern ultrasound equipment has made the antenatal detection of such lesions possible, allowing planning of the time, place, and mode of delivery to ensure the most favourable outcome. In the future, fetal surgery may become a realistic possibility.

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Case report

An aluminium foreign body in the oesophagus

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Impaction of an oesophageal foreign body is not an unusual problem and most commonly occurs in young children who are otherwise normal or in adults often with an underlying psychiatric history,¹ or benign oesophageal stricture. Early diagnosis is important and is usually not difficult. We report a case which outlines the problem with diagnosis of an aluminium foreign body.

CASE REPORT. A 22-year-old man was admitted as an emergency with a history suggestive of a foreign body lodged in the oesophagus. He had been drinking beer from a can. He had finished his second can and his girlfriend had opened the third can for him. He drank a few mouthfuls from this can quickly and then felt something sharp being swallowed. He immediately thought he had drunk the 'ring-pull' opener from the top of the can and when he asked his girlfriend she confirmed that she had put it into the can after opening it. He suffered a sharp retrosternal pain which over the next 30 – 60 minutes did not get better. When he presented at the accident and emergency department he was still able to swallow, and localised the pain to the mid-sternal level.

On examination he appeared well and not distressed. His pulse was 76/minute and of good volume. Blood pressure 120/70 mmHg. There was no clinical evidence of oesophageal perforation and abdominal examination was normal. Chest and abdominal X-rays failed to show any evidence of a foreign body or of oesophageal perforation. He was further questioned but he felt sure that the ring-pull was still stuck, and he was admitted to hospital with a diagnosis of mucosal tear or oesophagitis. He was commenced on an antacid preparation which produced some symptomatic improvement, and he was allowed oral fluids. He spent a comfortable night in hospital but the following day he was still adamant that there was something stuck in his oesophagus, although he was still able to swallow fluids. A lateral chest X-ray and further inspection of the original films did not reveal any foreign body. Conservative management was maintained in the belief that he simply had a small oesophageal tear.

The following day he still complained that there was something stuck. Because of the slight possibility of an oesophageal leak a swallow using water soluble contrast was ordered. This outlined a foreign body at the mid-oesophageal level consistent

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with a ring-pull. He was taken to theatre where the ring-pull was easily retrieved using the flexible gastroscope and biopsy forceps. He made a rapid recovery and was discharged the following day.

DISCUSSION

The early detection of an impacted oesophageal foreign body is important if the risk of oesophageal perforation is to be minimised.² Initial diagnosis depends upon whether the object is considered to be radio-opaque or not.³ Many people consider that all metals easily show up on an X-ray film and are radio-opaque objects — this case demonstrates that this is not true. As aluminium is a metal of low atomic number, it is less opaque to X-rays than most other metals, which are of higher atomic number. In fact, aluminium has a very similar atomic number to cortical bone.⁴ This proves a problem especially when X-raying the oesophagus, which on a plain chest X-ray lies over the spinal column. It might be thought that a lateral or oblique film would solve the problem, but because a chest X-ray requires a relatively high kilovoltage, and the higher the kilovoltage the less contrast there is between two different materials, this will also often fail to show up the foreign body.

The next step in diagnosis is to obtain a contrast radiograph, or the use of endoscopy.⁵ If perforation is considered to be a possibility then flexible endoscopy with the insufflation of air is not advisable and a water soluble contrast rather than barium should be used.⁶ A metal detector has been used to locate a foreign body which had passed unnoticed even after a water soluble contrast swallow.⁷

This case demonstrates the difficulties in detecting what is potentially quite a common oesophageal foreign body. Providing the limitations of plain X-rays are known the diagnosis should not be difficult. In order to avoid a potentially serious delay, examination by water soluble contrast medium should be performed at the earliest possible opportunity whenever the history suggests foreign body impaction which fails to be revealed by plain X-ray.

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Case report

The air-fluidised bed in the management of chronic varicose leg ulceration

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Air-fluidised bed therapy has been used for the prevention and treatment of bedsores. We report the successful use of this form of treatment for severe chronic varicose leg ulcers.

CASE REPORT. A 68-year-old woman presented to the geriatric medical unit with severe chronic varicose ulceration of the lower limbs. This had been present for over twenty years and had resulted in her spending two out of the past three years in hospital. She had been discharged from a dermatology unit less than three months earlier. Her ulcers were associated with marked oedema of the legs. She was grossly obese (weight 110 kgs) and had non-insulin-requiring diabetes mellitus. She lived in a sheltered dwelling with maximum home help support. A district nurse was dressing her legs twice daily. Although able to walk slowly with a Zimmer walking aid, she was reluctant to do so. She refused to elevate her legs and often slept in her chair. She also had been depressed and had exhibited antisocial behaviour.

Fungating ulceration and purulent discharge from the affected lesions were present at the time of her admission. There were three large purulent ulcers on the left leg, two of 5 × 5 cms and one of 14 × 8 cms. There was a 14 × 7 cms necrotic ulcer on the right leg. Maggots of the common house-fly were observed in the ulcer. Several toes were pre-gangrenous. She was in considerable pain and required slow-release morphine sulphate. Her sleep pattern was poor and she was demanding and unco-operative at times. Amputation was considered but was rejected by the patient.

Because of the severity of the ulceration and difficulties in nursing, therapy on an air-fluidised bed was commenced. A low calorie diet and daily wound dressing were instituted.

After eight days on the air-fluidised bed the oedema had greatly improved. The ulcers were cleaner and were beginning to heal and pain was less. Progress was maintained and after three weeks a similar but lower bed was used from which the patient could more easily be mobilised for physiotherapy. The air-fluidised bed was used for five weeks. At the end of this period two small ulcers (one of which

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measured 3 cms × 2 cms) remained on the left leg and another 3 cms × 2 cms ulcer remained on the right.

In spite of having been chair or bedfast for several weeks, the patient mobilised well. She was discharged to her sheltered dwelling two months after admission. Her weight fell to 93 kgs, her mood improved and she required only paracetamol for pain relief.

DISCUSSION

Air-fluidised bed therapy employs air bubbled through soda lime crystals coated with silicone. These microspheres look like a fine white powder. They act like a liquid and reduce pressure on the skin in contact with the bed to 11 mmHg (capillary closing pressure is 30 mmHg). This gives one of the lowest pressures of the methods available.^{1,2} The pH of 9 impedes bacterial growth.³ It has been used to nurse patients with burns and pressure sores,¹ but its use in severe varicose leg ulceration has not been described.

The bed can have a dehydrating effect by increasing insensible water loss. This may cause volume depletion⁴ which was beneficial in this patient's case as a considerable amount of her 17 kgs weight loss was achieved by an early reduction in leg oedema. However, this dehydrating effect can cause hypernatraemia which can be a serious problem in critically ill patients in whom fluid balance must be carefully monitored.⁵

The cost-effectiveness of such treatment is important. These beds are available only for hire at a cost of approximately £60 per day, including full 24-hour technical back-up. Before this patient commenced fluidised bed therapy, daily dressings were costing about £35 whereas none was subsequently needed. This patient had spent about two years in hospital at a cost of over £40,000 with marginal benefit to her leg ulcers. Community care involving daily dressings by a district nurse was also expensive. Factors such as the reduction in pain and depression are also important but are difficult to quantify.

Increased venous return, reduction in oedema and the bactericidal effect of the reduced pH may all have contributed to the improvement in this patient. Short term therapy with an air-fluidised bed may be clinically helpful and cost-effective in patients with severe refractory varicose leg ulceration when marked chronic oedema is present.

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Case report

Breast lymphoma: fine needle aspiration biopsy

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Non-Hodgkin's lymphoma of the breast is rare. This case confirms the importance of obtaining a pathological diagnosis for any clinically suspicious breast lump.

CASE REPORT. A 78-year-old woman presented with a five week history of a lump in her left breast and a four week history of a blocked right nostril. On examination, she had a hard, immobile mass in the breast and there was also obvious enlargement of her right nasal area, extending towards the eye and maxillary sinus. It was assumed that she had a carcinoma of her breast with a secondary metastasis in her nose. Because of her age she was commenced empirically on tamoxifen, but was admitted to hospital for further assessment. Detailed physical examination revealed no other abnormality. Routine blood tests, chest X-ray and ultrasound scan of the abdomen were normal. Computed tomography of the head revealed a large tumour involving the inside of the right nostril, extending posteriorly and superiorly, with distortion of the nasal bones.

A fine needle aspirate from the breast lesion indicated the diagnosis of malignant lymphoma. More extensive biopsies of the breast and nasal lesions were therefore performed, which confirmed the presence of a B-cell lymphoma of the centro-blastic type in both sites. Further computed tomography of the thorax and abdomen revealed no other abnormality. She subsequently underwent chemotherapy with cyclophosphamide, vincristine and prednisolone. At three month follow-up, after three courses of chemotherapy, the breast lesion was no longer clinically detectable and the nasal tumour had vanished.

DISCUSSION

About 20% of non-Hodgkin's lymphomas present with extra-nodal involvement; in one study only 0.44% involved the nasal cavity.¹ Most primary breast tumours are carcinomas; only one in a thousand is a lymphoma. Synchronous presentation of non-Hodgkin's lymphoma as a breast and nasal lesion is therefore very rare.

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Histological diagnosis is particularly important in the management of breast lumps.² The use of fine needle aspiration cytology is accurate,³ rapid and painless and once a diagnosis is made, unnecessary surgery may be avoided.⁴ If the diagnosis is lymphoma of the breast the prognosis overall is better than that for breast carcinoma, with a five year survival of 85% in one series.⁵ This case illustrates that fine needle aspiration cytology of all breast lumps should be obtained in order to reach accurate diagnosis and ensure correct management.

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Case report

Eosinophilic fasciitis presenting as psoriatic arthropathy

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Accepted 28 January 1992.

We describe a patient with eosinophilic fasciitis, a rare scleroderma-like steroid-responsive condition. Initial presentation suggested pauciarticular psoriatic arthritis.

CASE HISTORY. A 19-year-old student nurse presented with a four month history of pain and stiffness in her knees, ankles and elbows following a brief 'flu-like illness. Wrist and elbow movements were limited, with bilateral knee effusions and flexion deformities of approximately 10°. Evidence of psoriasis was limited to areas of hyperkeratosis on the elbows, and a few nail pits; there was a positive history in her maternal grandmother. Rheumatoid factor test was negative, serum C-reactive protein 19.5 mg/l (normal < 6), and erythrocyte sedimentation rate 39 mm/hr. X-rays of hands, elbows and knees were normal. A provisional diagnosis of psoriatic arthropathy was made and she was treated with the nonsteroidal anti-inflammatory drug nabumetone 1 gm daily, with improvement.

Five months later her condition deteriorated, her skin felt "very tight", and she had to walk on her toes because of tightness at the ankle. She described occasional difficulty with swallowing but gave no history of Raynaud's phenomenon. She now had thickened, shiny skin on the limbs and flexion deformity of her knees, wrists, and the small joints in her hands. Systemic sclerosis was considered to be the likely diagnosis, and she was commenced on d-penicillamine 100 mg/day and nifedipine 10 mg/tid.

Investigations showed eosinophilia of 8%; ESR 32 mm; C-reactive protein < 6 mg/l; serum IgM 2.72 g/l. Relevant autoantibody tests were negative (including antinuclear factor, anti-centromere antibody, anti SCL-70 and anti-neutrophil cytoplasmic antibody); complement components were normal and

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circulating immune complexes were not detected (Clq binding). Isotope bone scan revealed synovitis in both wrists, the metacarpophalangeal joints of both hands and ankles. *Borrelia burgdorferi* titres were negative. Barium swallow was normal.

Full thickness skin biopsy showed an intact epidermis, without hyperkeratosis or follicular plugging, and there was no vacuolar degeneration of the basal layer. There was loss of hair follicles, and the skin appendage structures were small and atrophic. The papillary dermis and upper reticular dermis were relatively normal but the deeper dermis showed thickened hyalinized eosinophilic collagen fibres (Figure). The dermis appeared thickened and there was extension of the collagen into the underlying subcutis. The fibrous septa within the subcutaneous fat were widened and within the fibrous tissue there were scattered chronic inflammatory cells at the periphery of the lobules of fat. The infiltrate was composed predominantly of lymphocytes and plasma cells with scattered histiocytes, but no eosinophils. The fascia was thickened, homogeneous and eosinophilic and was permeated by a chronic inflammatory cell infiltrate. A little underlying skeletal muscle was present in the biopsy and this showed myofibril degeneration and interstitial fibrosis. The overall appearances were those of eosinophilic fasciitis (Shulman's syndrome). Following histological diagnosis she was commenced on hydroxychloroquine sulphate 200 mg/day, prednisolone 40 mg/day and cimetidine 400 mg/twice daily; d-penicillamine was discontinued. She made a slow but gradual improvement, with return to normal gait.

It has been necessary to continue steroids at a relatively high dose.

DISCUSSION

Over 200 cases of eosinophilic fasciitis have been described since the initial description by Shulman.¹ It is a scleroderma-like disease characterised by symmetrical widespread inflammation and sclerosis of the deep fascia, subcutis and dermis: it has been termed *morphoea profunda* by some authors. It primarily involves the extremities, and is associated with peripheral blood and tissue eosinophilia, and often hypergammaglobulinaemia. Although well described, onset with polyarthritis may lead to initial diagnostic difficulty. Synovitis of large joints, hyperkeratosis and nail pits in conjunction with a family history of psoriasis, is a common mode of presentation of the pauciarticular type of psoriatic arthritis, and we believe this is the first report of eosinophilic fasciitis presenting in this manner. Synovitis had been present for at least four months prior to the insidious onset of sclerotic skin changes.



Figure. Thickened dermis and hyalinization of collagen bundles with chronic inflammatory cell infiltrate.

Eosinophilic fasciitis may be part of the spectrum of systemic sclerosis, as after one or two years a proportion of patients develop chronic fibrotic cutaneous features and some develop systemic manifestations, but there are some significant differences. These include the relative absence of Raynaud's phenomenon, normal nailfold capillary microscopy,² sparing of the epidermis and dermis, infrequent visceral involvement, absence of the serological features which characterise systemic sclerosis and the rare development of haematological complications.³ The male to female ratio is approximately equal, and in a recent review of 52 patients at the Mayo Clinic⁴ the mean age of onset was 47 years. In 46% of these cases, onset was related to unaccustomed strenuous exertion. Some authors mention a prodromal febrile illness as described by our patient, perhaps suggesting a viral trigger factor.⁵ Cutaneous manifestations are usual presenting features, and evolve through three stages: pitting oedema at onset, *peau d'orange* or dimpling, and finally induration. The most common sites are the arms and legs, with infrequent involvement of hands and feet. Isolated cases of oesophageal, cardiac or pulmonary disease have been described, but in contrast to systemic sclerosis these are uncommon features. In over 60% of cases eosinophilia is greater than 7% of the total white cell count, although peripheral eosinophilia can be transient, even in the absence of corticosteroid treatment. In our patient, eosinophilia was detected only once in her year-long illness.

To confirm the diagnosis a deep skin biopsy, including tissue from the epidermis down to the skeletal muscle through the deep fascia, must be performed. Changes range from inflammation with minimal connective tissue change to severe sclerosis. The deep fascia and septa of the subdermal fat are most extensively involved while the epidermis is normal or only slightly atrophic. Despite the name, the presence of eosinophils in the inflammatory infiltrate is not required for diagnosis. It is essentially a corticosteroid-responsive benign condition but patients with chronic ongoing disease have been described. In the larger proportion of cases resolution, which may be spontaneous, occurs within five years. Relapses, although unusual, do occur. Our patient displayed a characteristic clinical response to prednisolone. Cimetidine has also been reported to be effective,^{6,7} perhaps by involving the capacity of H₂ receptor antagonists to interfere with suppressor T-cell control.⁸

The etiology of eosinophilic fasciitis is unknown. One report of the condition developing in siblings not recently in contact within six months suggests the influence of genetic factors: they shared HLA A,B,C,DR, and DQ antigens.⁹ Hypersensitivity reactions to muscle tissue following exercise-induced damage and an association with cancer have also been demonstrated. An unusual syndrome characterised by incapacitating myalgia and peripheral eosinophilia in patients taking L-tryptophan has recently been reported. Some of these patients developed sclerotic cutaneous changes and the condition has been termed the eosinophilia myalgia syndrome. Overlapping clinical features suggested a relationship of both these syndromes with the toxic oil syndrome, a multi-system disease associated with ingestion of adulterated cooking oil in Spain in 1981. Although these three conditions display many features in common, Shulman¹⁰ described distinguishing clinical and laboratory features. Polyarthritides appeared to be equally prevalent in all three conditions. The recent suggestion that *Borrelia*

burgdorferi infections may be responsible for scleroderma variants including eosinophilic fasciitis remains speculative.¹¹

This case illustrates the need for precise diagnosis in patients who present with inflammatory polyarthritis and cutaneous sclerosis. Systemic sclerosis is unresponsive to steroids and the prognosis is often poor. In contrast, patients with eosinophilic fasciitis respond well to steroids or other potent immunosuppressive therapy and complete remission may occur. Appropriate full-thickness skin biopsy must be considered in all patients with clinically atypical systemic sclerosis.

We thank Mr C J F Russell, FRCS (Royal Victoria Hospital) for the tissue biopsy and Mrs M Maguire for typing the manuscript.

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Case report

Brain abscess ten years after penetrating glass injury to the skull

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Accepted 4 February 1992.

Delayed brain abscess formation has been reported in relation to retained fragments of bone, wood and metal following penetrating injuries to the skull. Intracranial glass fragments have been described on three previous occasions,¹⁻³ but we have been unable to find its association with brain abscess.

CASE REPORT. A 71-year-old man presented with a three week history of headache and progressive left-sided weakness followed by drowsiness and incontinence of urine over four days. Ten years previously, he had sustained an injury to his forehead and right eye by glass fragments from a broken windscreen in a road traffic accident. He did not lose consciousness following the accident. Multiple fragments of glass were removed from his right eye and forehead. He made a full recovery and ultimately returned to his occupation as a farmer. On admission, he was drowsy, confused and incontinent. Bilateral papilloedema was noted and he had a left hemiparesis. Cranial CT scan with intravenous contrast showed a 4 cm diameter ring lesion in the right frontal lobe with peripheral rim enhancement, surrounding oedema and contralateral midline shift. In addition, a high density shadow was seen on the medial margin of the enhancing ring (Figure). Since he was apyrexial with a normal peripheral white cell count, the provisional diagnosis was of a metastatic tumour. He was treated with dexamethasone 4 mg every six hours for three days following which his neurological condition improved.

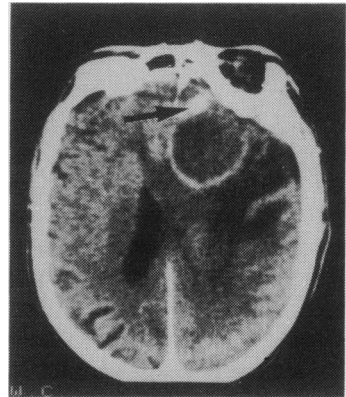


Figure. Contrast CT scan showing ring lesion with peripheral rim enhancement, surrounding oedema and midline shift. Hyperdense lesion (arrow) is presumably the larger fragment of glass.

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At subsequent craniotomy a coronal incision was fashioned in the usual way. Two holes were seen in the right frontal bone some distance from the frontal air sinus. Through these holes protruded granulation tissue which extended through the bone and the dura mater to reach the cerebral cortex. After opening the dura, aspiration through the granulation tissue yielded 35 ml of thick yellow pus. The abscess cavity was excised completely with the involved dura and bone. During dissection of the medial wall of the abscess capsule, two fragments of glass 12 × 5 and 3 × 2 mm (resembling typical windscreen fragments) were found within the brain substance. Examination of the anterior cranial fossa showed no abnormality in the roof of the orbit, ethmoid sinuses or frontal air sinuses. Culture of the pus yielded coliforms and enterococci after seven days incubation.

Following operation, he made a slow recovery, and treatment with mannitol followed by intravenous benzyl penicillin, metronidazole and chloramphenicol was given for seven days. CT scan 10 days after the operation revealed changes consistent with surgery, but the high density shadow seen in the preoperative scan was no longer visible, which must have been due to the larger glass fragment. Histopathological examination of the abscess capsule showed a narrow outer layer of fibrosis surrounded by a broad zone of gliosis with numerous gemistocyte cells and an inner necrotic zone surrounded by active inflammatory granulation tissue rich in polymorphs and plasma cells. Granulomata or fungal hyphae were not seen. The appearances were those of an abscess of three or four weeks age.

After discharge from hospital, oral antibiotic therapy was prescribed for four weeks and phenytoin for six months. At review six months later he was asymptomatic.

DISCUSSION

Abscesses occurring immediately or at an interval following penetrating head injury constitute only 3–12% of all intracranial abscesses.^{4,5} A brain abscess due to retained intracranial fragments usually occurs within a few weeks, but there is considerable variation and a delay of up to thirty-six years has been reported.⁶ The foreign bodies which provoke abscess formation are usually bone, wood or metal and the age distribution may be from 7–54 years.⁴

Wooden foreign bodies cause abscess formation in 48% of cases,⁷ and abscesses are ten times more common in the presence of bone fragments than in their absence.⁸ Metal fragments are often hot and sharp at penetration and perhaps produce a relatively sterile wound, which may explain the lower frequency of abscess formation (10–33%).⁵ Retained intracranial glass fragments have been reported but none of these patients developed brain abscess.^{1–3} As with other foreign bodies, the most common site of skull penetration for glass is the orbit. Penetration through the frontal bone has only been reported once previously, and these authors found at computed tomography that glass produces a hyperdense image.¹

Why are some of these intracranial abscesses delayed? The initial infective organism may be of low virulence or may be rendered so by an antibiotic administered at the time of trauma;⁸ reactivation may then be caused by a subsequent infection. Alternatively, the foreign body itself may decrease local tissue resistance (gliotic tissue being relatively avascular) and so produces an area vulnerable to any casual bacteraemia. One third of cases of delayed abscess have been

reported as presenting with a two stage illness.⁹ In the present case, the finding of a chronic granulomatous track at operation leading to a relatively acute abscess of the order of three weeks old is consistent with the second speculation.

Treatment of such injuries is always difficult. Adequate debridement of wounds should be carried out and such foreign bodies, as are readily accessible, removed. In the present case, it was clearly not possible to remove the intracranial foreign bodies, even had their presence been recognised.

Because of the high incidence of epilepsy following intracranial sepsis, it is common policy to give prophylactic anticonvulsant therapy such as phenytoin for a period of three to six months. There is no indication to give prophylactic anti-convulsants in the treatment of the primary injury.

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Case report

Pseudogout, chondrocalcinosis and the early recognition of haemochromatosis

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Accepted 21 January 1992.

Idiopathic haemochromatosis is a disorder which has a well-known association with chondrocalcinosis. We report arthritis associated with chondrocalcinosis as the first clinical manifestation of underlying haemochromatosis in two other-wise asymptomatic male patients. In one patient, pyrophosphate arthropathy presented with acute pseudogout. Better appreciation of this presentation will result in earlier diagnosis of haemochromatosis and the institution of appropriate treatment to prevent irreversible liver damage.

CASE 1. A 54-year-old man with no previous history of joint disease presented with an acutely swollen, painful left knee which had developed overnight. Plain radiographs of the knee demonstrated intra-articular calcification. Polarising microscopy of centrifuged deposit from the knee demonstrated typical calcium pyrophosphate dihydrate crystals. A diagnosis of pseudogout was made and indomethacin 25 mg three times daily prescribed. After six months there was persistence of the effusion in the left knee, and he also complained of episodic pains in his fingers, toes and both elbows. The metacarpophalangeal joints in both hands were enlarged with tender second and third metacarpal heads, lacking full flexion. Elbows had flexion deformities of 10 degrees but were not tender. All other joints including shoulders, cervical and lumbar spine were not tender, with normal range of movement. The liver was palpable below the costal margin, but the spleen was not palpable. Abnormal skin pigmentation was not present.

Radiographs of knees, wrists, pelvis and feet showed extensive cartilaginous calcification, including involvement of the second and third metacarpophalangeal joints of the hands (Fig 1). In the spine there was calcification within some intervertebral disc spaces and along the posterior spinal ligament. Fluid obtained from asymptomatic right and left knees contained typical calcium pyrophosphate dihydrate crystals.

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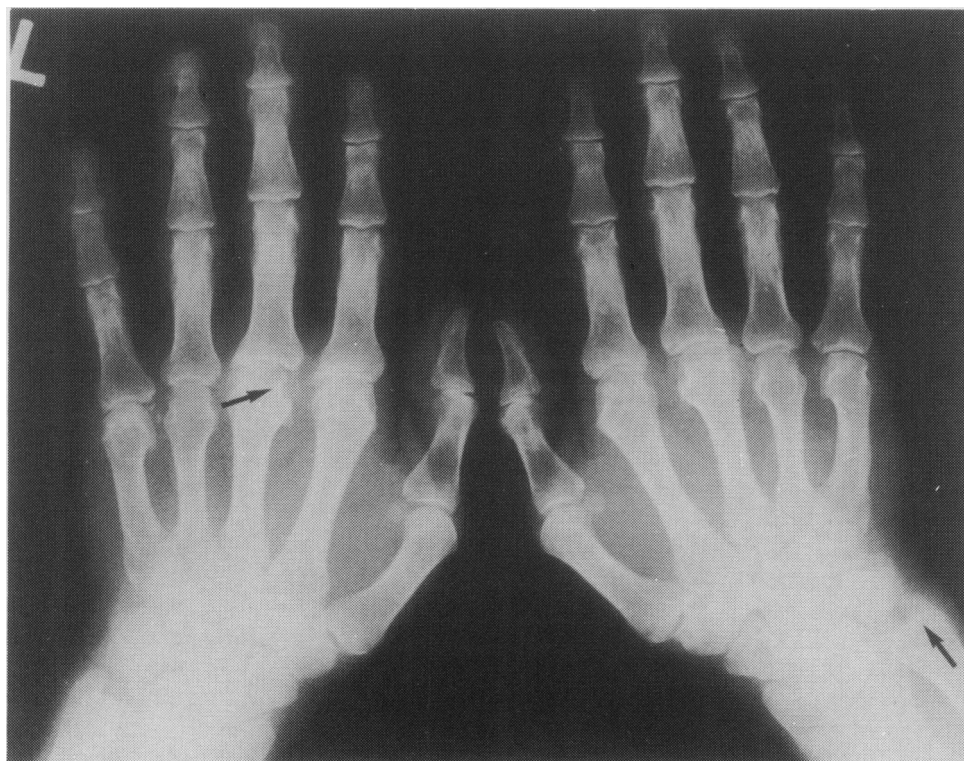


Fig 1. Case 1 — Calcification of wrist and third metacarpophalangeal joints.

Haemoglobin concentration was 15.5 g/dl, with a normal erythrocyte sedimentation rate and serum C-reactive protein. Plasma glucose was 8.2 mmol/l. There was moderate elevation of serum transaminases, a low plasma urea and a raised serum iron 68 $\mu\text{mol/l}$ (normal 14–29). Serum ferritin was also increased to 7920 $\mu\text{g/l}$ (normal 100–150). Transcutaneous liver biopsy showed extensive deposits of iron in hepatocytes by light microscopy with Prussian blue staining, and by electron microscopy. The concentration of iron by spectrophotometric analysis of liver tissue was 16.3 ng/kg dry weight (normal below 2.0). These results confirm the diagnosis of haemochromatosis.

His siblings (one male and three females) were screened, and plain radiographs, liver function tests, plasma glucose and full iron studies were entirely normal in all. The patient was venesected weekly, with a reduction in serum ferritin to 3080 mg/ml after 15 months. His joint symptoms have not returned although radiological calcification remains.

CASE 2. A 63-year-old male first presented in 1975 with pain and stiffness of the neck, shoulders, hands, hips and knees of two years duration. Examination showed painful limited movements of these joints. There was bony swelling of hands and knees with bilateral knee crepitus. Radiology showed cartilage loss and osteophytosis without chondrocalcinosis in hands, knees and feet. General examination was unremarkable apart from mild hypertension (170/100 mmHg).

He had not taken alcohol for 15 years. Haemoglobin was 15 g/dl with normal indices and erythrocyte sedimentation rate 4 mm in the first hour. Plasma glucose was 5.8 mmol/l. Serum aspartate transaminase was 74 μ /l (normal 16 – 51), alanine transaminase 144 μ /l (normal 10 – 45) and alkaline phosphatase 193 U/l (normal 35 – 106). Serum iron was elevated, 48 μ mol/l. Antinuclear antibodies, antimitochondrial antibody and anti-smooth muscle antibody were not detected in the serum. He was treated with indomethacin and salicylate for joint pain and cyclopentazide and potassium for hypertension.

On examination in 1985 the liver was 2 cm enlarged, smooth and non-tender without splenomegaly. He had painful limitation of movements of hips, knees, elbows, shoulders, wrists and fingers. There was a small effusion in the right knee and bilateral knee crepitus with bony enlargement of knees, wrists and finger joints. Radiographs of hips, knees, feet and lumbar spine showed widespread degenerative arthritis and chondrocalcinosis of the knee joints (Fig 2) but not the spine. Microscopy of synovial fluid from the right knee revealed typical calcium pyrophosphate dihydrate crystals. Haemoglobin was 15.7 g/dl and erythrocyte sedimentation rate 3 mm/hr. A glucose tolerance test showed mild diabetes mellitus. Serum alkaline phosphatase was still high at 387 U/l, but aminotransferases and gammaglutamyl transferase were normal. Serum iron was 46 μ mol/l,

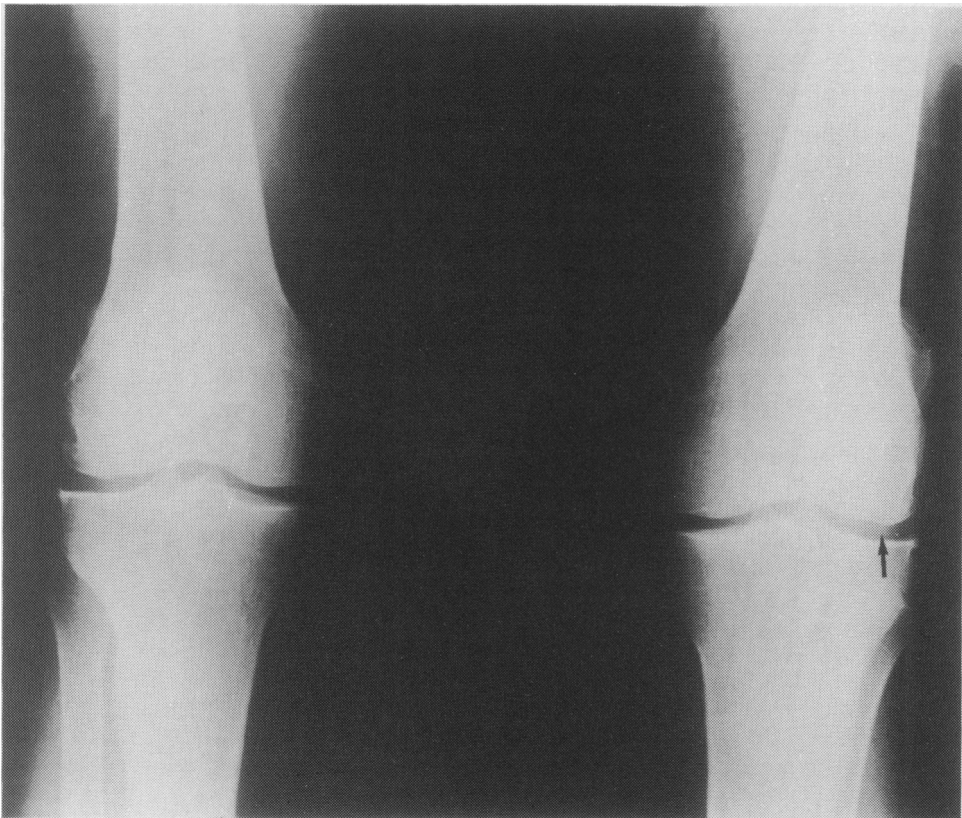


Fig 2. Case 2 — Chondrocalcinosis of the knees.

total iron binding capacity 54 $\mu\text{mol/l}$ (normal 46–72), with 76% saturation. Serum ferritin was 3423 $\mu\text{g/l}$. A desferrioxamine excretion test showed increased urinary iron elimination over 24 hours from 2 μmol to 19 μmol indicating considerable iron overload. Ultrasound scan of the liver showed increased echogenicity and decreased penetration consistent with fatty degeneration or early cirrhotic change. Computerised tomographic scan of the liver gave an attenuation value of 75 Hounsfield units (normal 60–70), just below a diagnostic range for haemochromatosis.¹ Needle biopsy showed extensive iron deposition in hepatocytes with normal liver architecture and membrane-bound haemosiderin deposits on electronmicroscopy. The concentration of iron per dry weight liver was 10.1 ng/kg confirming a diagnosis of haemochromatosis. Screening of the patient's siblings (two male and three female) was undertaken as in Case 1 and revealed no abnormalities. The patient was venesected weekly.

DISCUSSION

The relationship between chondrocalcinosis and calcium pyrophosphate dihydrate crystal deposition was first described by McCarty et al² and association with other diseases have been reported.^{3, 4} In a series of patients with haemochromatosis Schumaker described two patients with small and large joint arthropathy due to these crystals.⁵ The incidence of this arthropathy in haemochromatosis is now thought to be between 30 to 50% and of these over half have radiological evidence of chondrocalcinosis. The severity of the arthropathy, however, correlates poorly with the degree of iron overload and liver involvement and has even presented after prolonged venesection.^{6, 7}

Two previous reports have described four patients who subsequently had a diagnosis of haemochromatosis confirmed, where an arthropathy was the presenting feature. These patients had a history of arthritis present for two to 27 years prior to identification of haemochromatosis. Though the distribution of the arthropathy involving spinal and peripheral joints occurs in other diseases associated with calcium pyrophosphate crystal deposition, the changes at the metacarpophalangeal joints are characteristic and highly suggestive of the chondrocalcinosis associated with haemochromatosis.^{7–10} Our first patient presented with an acute episode of pseudogout which has not been previously described as the presenting feature of haemochromatosis.

In patients with chondrocalcinosis where the distribution of the disease is suggestive, including those who present with pseudogout, estimation of serum iron, ferritin and aminotransferases should be performed leading if necessary to a desferrioxamine test, CT scan and biopsy of the liver.

We thank Dr M E Callender for performing the liver biopsies, Dr D McMaster for the measurement of liver iron stores and Mrs M Loughran for typing the manuscript.

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Book reviews

Diabetes and atherosclerosis. By R W Stout. (pp 295. £79.50). Volume 125 of *Development in Cardiovascular Medicine*. Dordrecht, Netherlands: Kluwer Academic Publishers, 1992.

This edited book provides a very useful series of chapters relating to the development of arterial and heart disease in diabetics. Atherosclerosis is widely recognised to be the major killer in Type I and Type II diabetic patients, but there are few comprehensive reviews of the background data and this book fills a niche.

Robert Stout has been joined by colleagues in Belfast who cover the pathophysiological mechanisms. The editor provides an outline of the development of atheroma, the epidemiology of the increased incidence of atheroma-related diseases in diabetes and the known risk factors. Keith Buchanan covers hormonal control of insulin secretion, Patrick Bell the potential role of insulin resistance and Robert Stout the possibility that insulin is a proocative agent. Liz Trimble and Ian McDowell present the various lipid abnormalities in Type I and Type II diabetic subjects, and the problem of knowing when and how to treat. Brew Atkinson and Catherine Ritchie discuss the role of hypertension and the current therapeutic approaches. Laurence Kennedy indicates the potential pathological role of glycation of lipoproteins, coagulation proteins and structural proteins, and the development of advanced glycation end-products. Elizabeth Mayne covers the various haemostatic and endothelial abnormalities that are likely to be involved in the development of micro and macro-vascular disease, David McCance and David Hadden the relationship of proteinuria to both nephropathy and heart disease and Randal Hayes the pathophysiology and role of non-ischaemic heart disease.

The book is well referenced up to 1990 and the chapters provide succinct and readable background reading. The book reflects the high standing of clinical diabetes and related academic specialties in Ulster. I will find the book a useful source. I recommend the book to those who are interested in the all too common problem that it is atherosclerosis that most commonly leads to the clinical morbidity and early death of our diabetic patients.

RC TURNER

The future of medical journals. Edited by S Lock. (pp 217. £14.95). London: British Medical Journal, 1991.

In 1984 I established the present editorial position on the *Ulster Medical Journal* in the words of Francis Bacon "Reading maketh a full man, conference a ready man, and writing an exact man." The Editorial Board trust that your *Journal* will provide good medical reading and therefore stimulate conference, but most of all that the skills of medical writing will be inculcated in an increasing number of doctors.

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This little book which was produced in commemoration of 150 years of the British Medical Journal, records the views of most of the senior editors of the major medical journals in the English-speaking world. The Ulster Medical Journal does not even appear on the list of the major journals in regard to what is known as "Impact factor" or "Citation Index" but nonetheless we exist as a peer-reviewed general medical journal and intend to continue to do so. The articles in this booklet are chiefly concerned with the views of editors about themselves, and in some-what highminded views in how the journals improve medical knowledge. Perhaps all of us would benefit from the discipline of having to write down what we mean, and then submit it to our peers to allow them to re-order our phrases and even our thoughts in the belief that there is a final common pathway of expression which in some way is best. Dr Arnold Relman, past editor of the New England Journal of Medicine, states "perhaps the most important qualities of all in an editor are moral courage and a sense of fair play".

Whatever the future technology of bioinformatics — from papyrus to parchment to paper to pixels — I have a feeling that the Ulster Medical Journal will still be publishing papers from doctors in Ulster for many years to come, both to express the wisdom and experience of senior members of the profession and to encourage the first faltering footsteps of a scientifically trained but not very literate medical graduate who feels the need to write something down.

DR HADDEN

The case for preconception care of men and women. By M A Wynn and A Wynn. (pp 169. £29.50). AB Academic Publishers, PO Box 42, Bicester, Oxon OX6 7NW.

This book is written in a rather journalistic manner and therefore would be appreciated by those who prefer their source of knowledge presented in this way.

The concept of preconception care is important in obstetrics. However, the subject matter in this book concentrates mainly on aetiological factors affecting mutations of the parental germ cells, rather than on the preconception care that is really necessary to avoid risks to mother and fetus. For example, when the high incidence of fetal abnormalities is discussed in diabetes the importance of normalisation of their blood glucose prior to conception to reduce the possibility of fetal abnormalities is not mentioned. Similarly, when the harmful effects of drugs are being discussed, the management of patients on certain drugs and the need to change to another drug of similar effect which does not cross the placental barrier is not mentioned.

The information in this book is valuable but one is surprised that with this title the contents do not contain information on the preconceptional management of patients who need it most. Pregnancy in patients with hypertension, diabetes and heart disease is becoming more common each year, to say nothing of patients with disruption of their pelvic architecture following road traffic accidents.

JMG HARLEY

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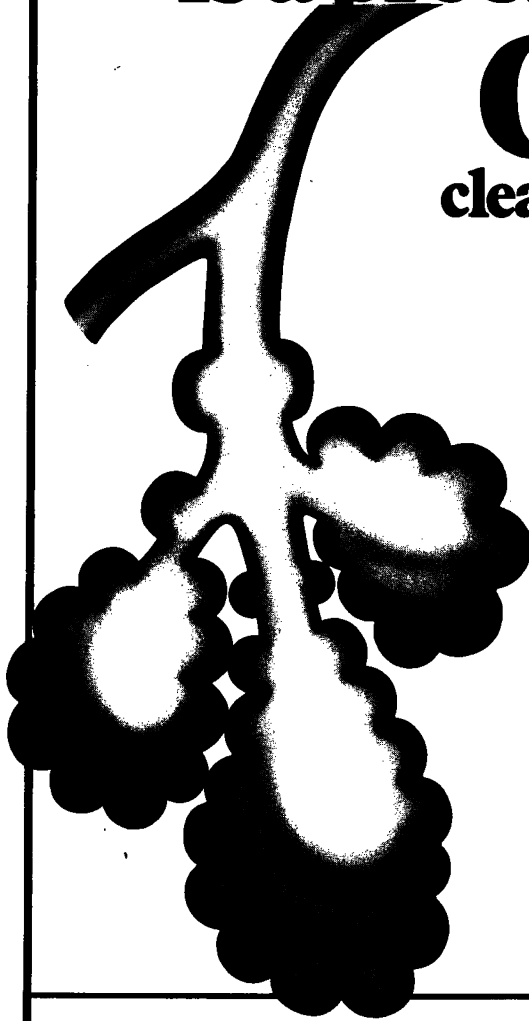
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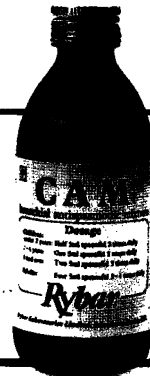


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