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[continued on back cover

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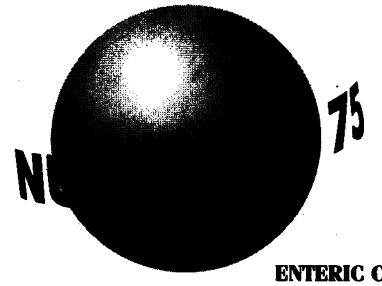

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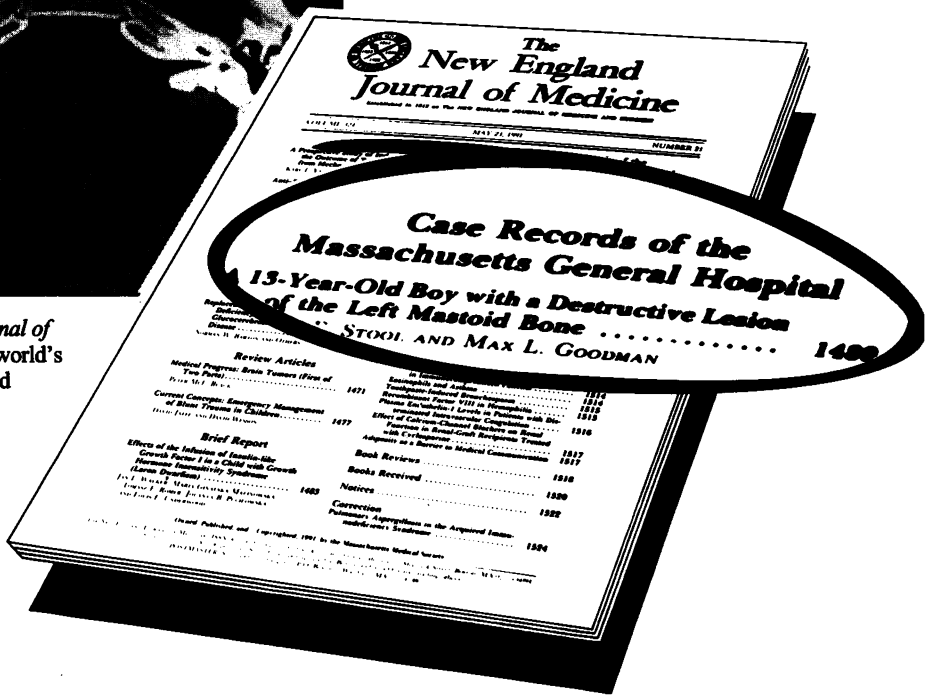

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The Ulster Medical Journal, Volume 63, No. 2, October 1994.

The Ulster Medical Journal

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1994

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The Ulster Medical Journal

The Journal of the Ulster Medical Society

Volume 63 No. 2 October 1994

Editorial

More change . . .

The editorial in the Spring 1994 issue dwelt on resistance to the concept of change which many of us in the medical profession, even in Ulster, would recognise. We want to be doctors, we are trained to be doctors, we can see changes which would benefit those people who come to us as patients – but we are resistant to politicians who think they can see a broader perspective.

But changes are happening and happening fast. The costs of providing X-rays, laboratory tests, day theatres, car parks, even offices, as much as the traditional costs of beds and nurses are driving those changes. At this time Dr McKenna and his group of wise people are deliberating on the future profile of the two largest and most centrally placed hospitals in Northern Ireland. One single administrative institution, on two or more sites, might not please the politicians who are looking for competition, but would certainly diminish individual doctors reluctance to see their personal provider profiles reapportioned by supposedly rival organizations.

Or will fund holding practices or their newly appointed practice managers, the second wave of whom are about to appear on the contracting scene, will they be the decision makers? Some well established practices are resisting this scenario and opting to join a non-fund holding group, hoping to retain more of the personal nature of their medical life with less insistence on financial considerations.

The desire of government to control the health care organization is not new, nor even unreasonable. But politicians are wary of their electoral base and this leads to electorally based decisions which are not necessarily best for the delivery of good health care. Sir Dennis Faulkner, a respected businessman, with a group of similarly experienced colleagues in positions of executive responsibility as chairmen of hospital trusts, could see the problem from an even wider perspective than our local politicians: but the business view that Northern Ireland warranted only one health care organization, not four, did not get political approval. Are we stuck in a "no-change" position for non-medical reasons?

There have been many changes for the better in the past few years: communication by dedicated telephones between general practitioner and hospital is now expected – but can the patient expect as much? Letters in both directions are more expeditious. We audit what we do and make suggestions on how to do better. We have better premises, particularly in family practice – some hospitals are still too old fashioned. But all of these changes could have been achieved without the radical reforming zeal of the government of the day: all simply needed to have money spent. Waiting lists for some surgical procedures cause much concern: the controlling point is when in the course of an illness the decision to enter the surgical scene is made – change in procedures, and increasing microsurgical and intravascular technology means a change in the general perception of when something should be done. Large clinics with waiting rooms full of exasperated but usually tolerant people waiting to see a hospital specialist should no longer exist: the very nature of a "consultation", and of a "specialist" is changing.

The brave new world of the health service reforms of the 1980's will not necessarily be applicable in the year 2000. There is no real consideration for office practice for specialists in current hospital planning: we count the beds, calculate the number of nurses required, and add on the outpatients as an addendum. The major reduction in expensive long term hospital geriatric care, which Dr Taylor and Dr McConnell refer to in their three articles in this issue, shows that that sort of calculation can get it very wrong. Will we move to specialist "office practice", with hospitals functioning only for acute emergencies and complex operations? That would be a change back to the situation 50 years ago when "Consultants" provided specialist opinions from their consulting rooms at home in University Square, and provided emergency services at the hospital largely as a grace and favour.

It is exactly 50 years since the then Colonel Ian Fraser, fresh from surgical destinction in the allied invasion of Sicily, found himself leading a mobile hospital to the D-Day beaches, armed with the newly available penicillin. It is 80 years since the young Thomas Houston set out to support the troops in the trenches of Northern Europe. The historical view may allow us to adjust our sights for the future. Will we be celebrating 50 years of the National Health Service in 1998? I hope we will.

D R HADDEN

The Ulster Medical Journal, Volume 63, No. 2, pp. 127 - 136, October 1994.

The changing face of oesophageal cancer treatment in Northern Ireland

K McManus, J McGuigan

Accepted 6 September 1994

SUMMARY

In the late 1970's the options for treatment of oesophageal cancer were limited. When cure was thought possible, resection was performed by the Ivor Lewis or oesophagogastrectomy techniques. Mortality was high, local recurrence rates disappointing, and long-term survival poor. For those patients whose tumours could not be resected, palliative intubation required open operation with high morbidity, and gave poor quality of life. In 1994, selective screening is diagnosing cancers early, more extensive resections are possible with lower mortality, and fewer local recurrences. Adjuvant therapy is increasing the operability rates. Gradually the facade of poor prognosis is being etched away, so that more patients are being given better quality of life, and cure is a distinct possibility. Palliation can be achieved endoscopically by dilatation, intubation or laser ablation combined with local external beam radiation. Mortality for palliative procedures is now considerably reduced.

INTRODUCTION

In 1987 a review of the treatment of oesophageal cancer over the preceding ten years was undertaken at the Royal Victoria Hospital. Of the 401 patients presenting to the hospital, exploratory surgery was performed on 247, and 221 underwent resection. Clear tumour margins were obtained in 112, 82 of whom survived their surgery. Thus only 20 per cent of those who presented for oesophageal surgery left hospital with their disease completely resected. The five-year survival following resection was 13.2% (Fig. 1).

A broad range of factors affecting survival following resection have been examined, and while many were found to be significant, the only independent predictors of survival were sex, involvement of proximal resection margins, curative resection and presence of nodal metastases

By way of palliation, 113 Celestin tubes were passed at open laparotomy or thoracotomy and 26 Atkinson tubes introduced endoscopically. In nine patients the tumours were bypassed, 21 patients were referred for primary radiotherapy, and 14 were unfit for surgical intervention.

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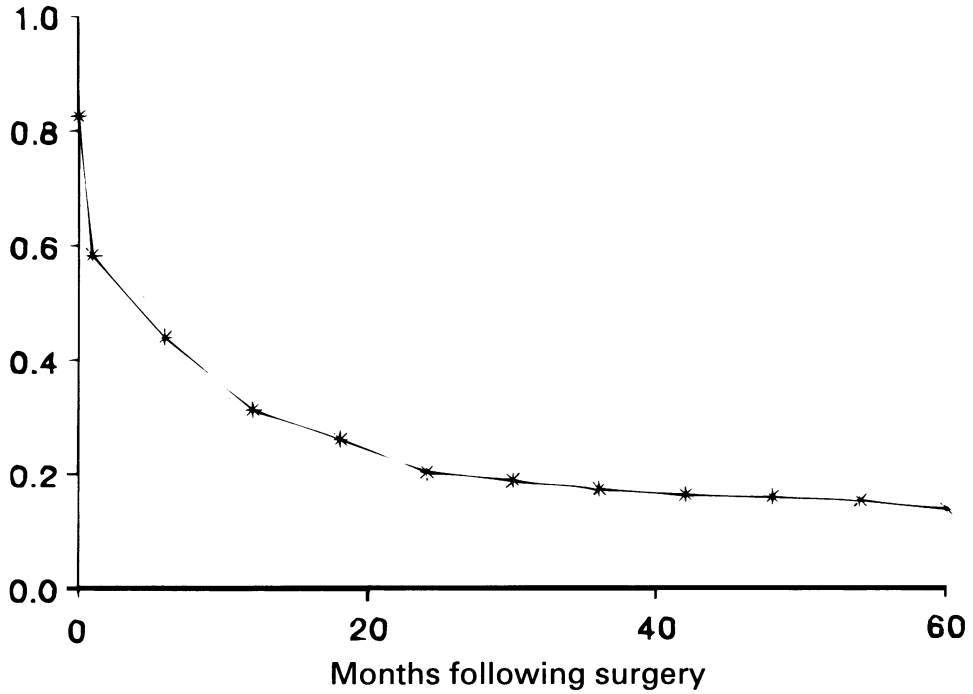

Fig 1. Probability of survival following oesophageal resection, RVH 1977-1986. All patients.

Following a transition period in 1986-1987, a number of changes have been made in the approach to oesophageal malignancies. Endoscopic surveillance has been introduced for patients with premalignant pathology. Laser ablation has become the treatment of choice for palliating inoperable tumours. For those in whom cure is thought possible, the more extensive total thoracic oesophagectomy operation has been introduced with the aim of resecting more of the proximal oesophagus, performing the anastomosis in the neck. Of the 139 oesophageal resections performed between 1988 and 1992, 73 have involved total oesophagectomy; 20 have been for benign or premalignant disease.

In this paper we describe the changes in our approach to oesophageal cancer, the results of these changes, and outline future plans for treating this difficult disease.

DIAGNOSIS AND SURVEILLANCE OF OESOPHAGEAL PREMALIGNANT CONDITIONS

It has become apparent that peptic disorders are associated with oesophageal cancer. In the 1977-86 series, 21.5% of patients had hiatal hernia, and a further 24.3% had other peptic disease. In particular, patients whose oesophagitis has advanced to the stage where the oesophageal lining has changed to columnar intestinal epithelium (Barrett's metaplasia), have a cancer risk 40 times that of the normal population.[1] In conjunction with our colleagues in gastro-enterology, it is our practice to maintain endoscopic surveillance for all patients with Barrett's metaplasia. When metaplastic columnar epithelium is found in the

true oesophagus above the oesophagogastric junction, yearly surveillance is recommended. When dysplasia is reported, more frequent and more intense surveillance is required. If any biopsy is reported as containing high grade dysplasia, resection is recommended.

Thirty-three of 112 cancers in the latter series had metaplastic epithelium present in the oesophagus adjacent to the tumour. Six of these had been under surveillance, and the five Stage I tumours have a good chance of long-term survival. In addition, 12 resections were performed for Barrett's metaplasia without cancer.

More recently we have turned our attention to molecular biology in an attempt to determine which patients are likely to develop tumours. Oncogenes, genetic mutations associated with tumour development, have been identified in oesophageal tumours from a tissue bank set up at the Royal Victoria Hospital (A Ritchie). Mutations of the p53 tumour suppressor gene, which render it inactive, have also been demonstrated in tumour tissue from this bank (K Gleeson, Department of Medical Genetics, The Queen's University of Belfast). It is hoped that assaying for these mutant genes or their protein products will help indicate premalignant change in patients under surveillance. Oncogenes, being unique to malignant and premalignant cells, would be suitable targets for anti-tumour antibodies, and this treatment modality may be available to us in the future.

RESECTIONS FOR OESOPHAGEAL CANCER

The standard oesophagogastrectomy procedure is usually performed via a left thoraco-abdominal incision (Fig 2). The proximal limit of resection is judged by the surgeon to be a point 5-7 cm proximal to the palpable tumour. The anastomosis is made below the aortic arch within the chest. When the tumour encroaches significantly onto the stomach the resection is extended to include a total gastrectomy. The Ivor Lewis oesophagogastrectomy devised for more proximal tumours, requires an upper midline laparotomy for mobilization of the stomach and a right thoracotomy to resect the tumour, again with a 7 cm margin, the anastomosis being above the level of the azygos vein. The decision as to whether the oesophagogastrectomy or Ivor Lewis approach was used depended on the barium and endoscopic findings.

Total thoracic oesophagectomy is performed using the technique described by Matthews[2] using a left thoraco-abdominal incision, resembling that for the oesophago-gastrectomy but with further dissection of the oesophagus above the aortic arch into the neck from below. The anastomosis is performed via a small cervical incision. This operation is performed whenever there is enough stomach available after adequate tumour resection to reach comfortably to the neck. It is therefore not used for true gastric fundal tumours where an oesophago-gastrectomy or total gastrectomy is performed. The decision on the extent of resection does not need to be made until the tumour is mobilised and is fully assessible.

Mention must be made of the 'transhiatal' oesophagectomy approach which is performed via a laparotomy with blunt dissection of the oesophageal tumour and anastomosis in the neck. This operation does fulfill the requirements of total thoracic oesophageal resection with the reduced morbidity of the cervical

anastomosis but avoiding some of the complications of thoracotomy. While recognising its place in the treatment of benign conditions such as advanced achalasia and Barrett's metaplasia, we have reservations about its use in malignant conditions.

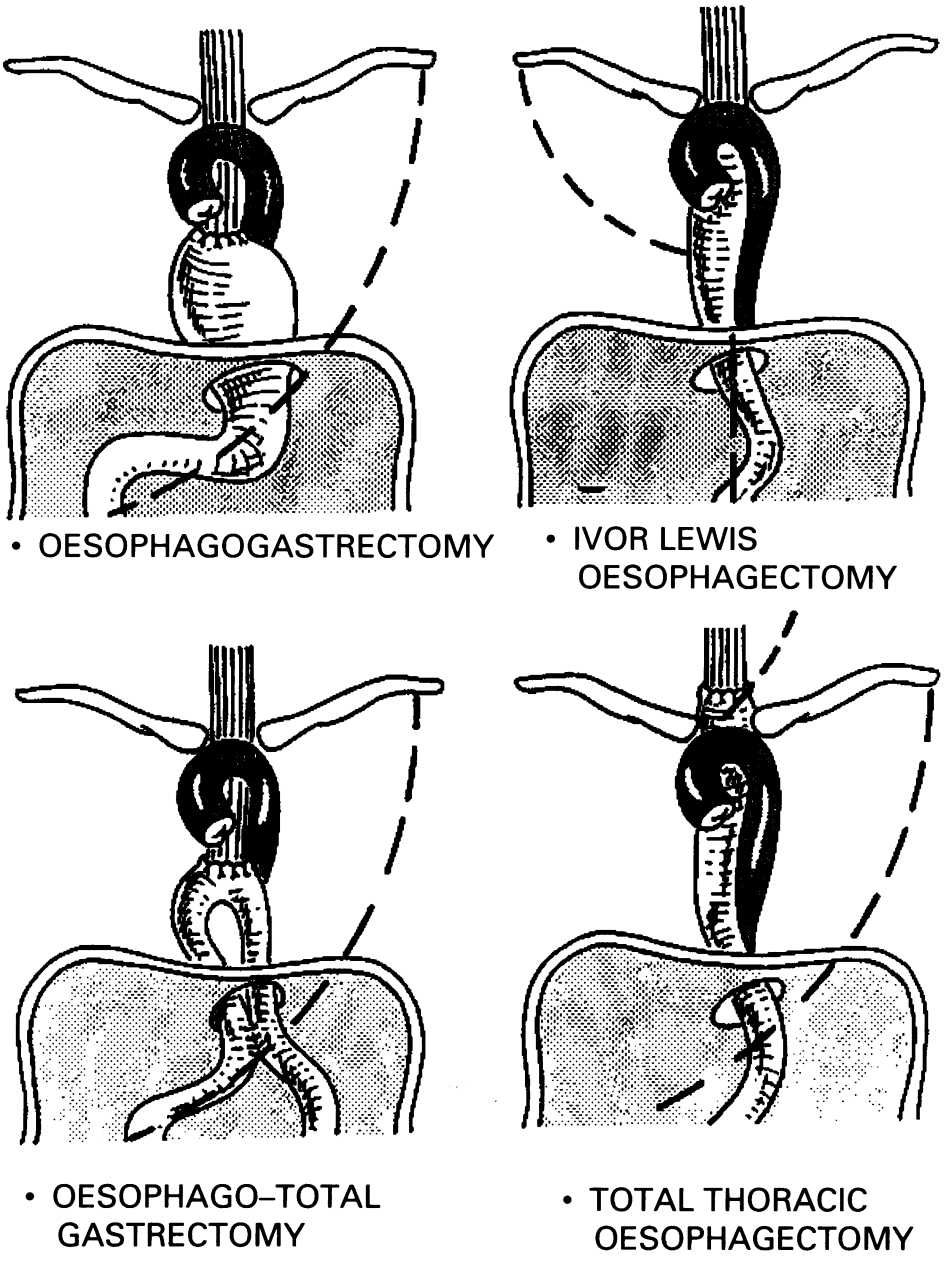

Fig 2. Operative approaches to oesophageal cancer.

MORBIDITY/MORTALITY

The National Confidential Enquiry into Postoperative Deaths (NCEPOD)[3] for 1992 recognised oesophagectomy as a procedure with risks "worse than that of transplantation and cardiac surgery". This is especially the case in the hands of the occasional oesophageal surgeon.[4] Our mortality for oesophagectomy improved from 39% in 1977 to 11% in 1985. This improvement was maintained with the introduction of the more extensive procedures. The mean mortality for total thoracic oesophagectomy over the first five years was 10.9%, with only one death in the last two years. The mortality for the oesophagogastrectomy procedures was 13.5% in the same period. Much of the improvement over recent years has been a result of better patient selection on the basis of our earlier experience, and also from improvements in postoperative intensive care. An important contribution has been the decrease in anastomotic leaks.

Intrathoracic anastomotic leak has a mortality of over 50%. The incidence has been lowered by the use of mechanical staplers.[5] Cervical anastomotic leak has occurred in two patients (4.3%), with no deaths. Leaks in the neck usually drain via the local incision without mediastinal or pleural contamination. The resulting fistulae drain well and close spontaneously. Non-fatal complications occurred in 23%, being comparable for all procedures.

TABLE

Factors favourably affecting survival following resection for oesophageal cancer 1977-1986 (univariate analysis)

Covariate	p value
Female sex	0.003
Curative resection	<0.001
Nodes free of metastases	<0.001
Clear proximal margin	<0.001
Tumour confined to oesophageal muscle	0.003
Ivor Lewis operation	0.004
Favourable differentiation	0.02
Clear distal margin	0.057
Short symptom duration	0.041
Squamous histology	0.07
Site of tumour (middle third/lower third/cardia)	0.14
Weight loss	0.16
Year of operation	0.57
Age	0.57
Surgeon grade	0.89

SURVIVAL FOLLOWING RESECTION OF OESOPHAGEAL CANCER

The factors affecting survival after resection and discharge from hospital for the 1977-1986 series are summarised in Table I. A multivariate analysis showed that only female sex, curative resection, absence of nodal metastases and clear proximal resection margins were independent predictors of long-term survival. Nodal metastases, the most significant predictor of survival, are indicative of systemic dissemination of tumour. This is reflected by the fact that regardless of the type of operation there is substantial tumour-related mortality in the early months after surgery due to undetected metastatic disease. What is clear is that the future treatment of carcinoma of the oesophagus must include systemic therapy. Many surgeons have now become interested in chemotherapy, in conjunction with surgery. Until recent years, no chemotherapeutic regimes had satisfactory effects on solid tumours, though 5-fluorouracil had some effect in gastrointestinal tract adenocarcinoma.

Success in treating squamous carcinoma of the larynx and lung with cisplatinum logically progressed to trials on oesophageal cancer. [6, 7, 8] The problems of toxicity and patient compliance are now better controlled, and regimes of rehydration, diuretics, sedation and anti-emetics are being standardised. Pilot studies in Belfast have shown the feasibility of such therapy, and patients are being entered in a number of trials of preoperative chemotherapy.

The other main cause of early death is tumour recurrence at the anastomosis. Recurrence is related to the type of operation and particularly the presence of tumour in the vicinity of the proximal resection margin. This is reflected in the survival curves for these two factors (Figs. 3 and 4).

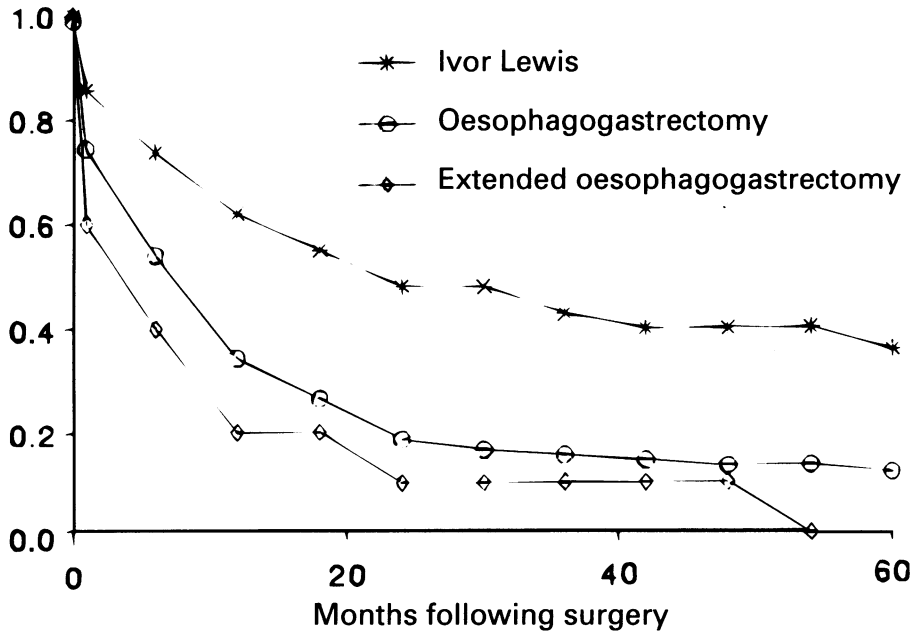

Fig 3. Probability of survival following oesophageal resection, RVH 1977-1986. (p = 0.004) Operation type. (Operative deaths excluded).

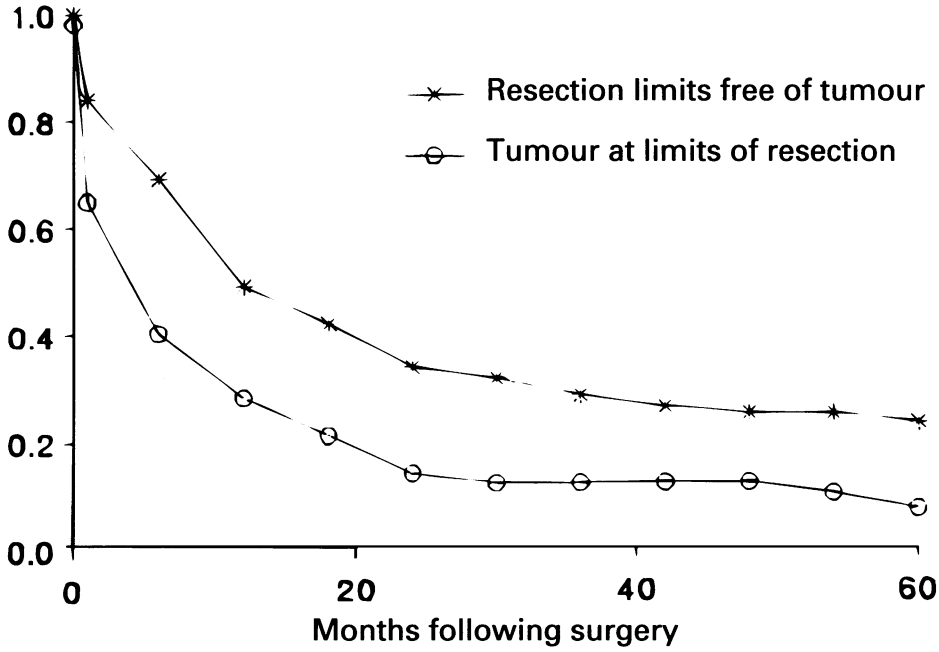

Fig 4. Probability of survival following oesophageal resection, RVH 1977-1986. (p = 0.0006) Proximal margin. (Operative deaths excluded).

With the introduction of total thoracic oesophagectomy the incidence of proximal limit involvement and anastomotic recurrence has dropped to zero. Despite attempts to gain a greater proximal margin in both Ivor Lewis and oesophagogastrectomy resections, the recurrence rates remain high for these operations (38.5% for Ivor Lewis, 15.8% for oesophagogastrectomy). The distal clearance is similar between all groups though a number of tumours have recurred on the lesser curve of the stomach. The short and medium term survival in the total thoracic oesophagectomy group reflects the improvement in proximal tumour clearance (Fig. 5).

PALLIATION OF UNRESECTABLE OESOPHAGEAL CANCER

Thirty eight per cent of oesophageal cancers present to this hospital in an inoperable state. Palliation, therefore, is a major part of the treatment of oesophageal cancer. Inability to swallow ones' own saliva is such an unpleasant end, that aggressive measures are usually employed to restore at least a liquid diet, despite the considerable risks of some of the procedures.

Resection is a major debilitating operation with high risk and prolonged recovery. It is not indicated when metastatic disease is detected during preoperative staging or open exploration, nor when resection is likely to be incomplete and the prognosis poor. Tumour bypass is advocated by some authorities, especially in the case of tracheo-oesophageal fistula.[9] The operation is as extensive a procedure as resection with an anastomosis in the chest. The experience in our 1977-1986 series showed very poor results for bypass.

Intubation of the tumour can maintain a lumen adequate to allow a pureéd diet. An Atkinson tube may be placed at endoscopy, or a Celestin tube at open operation. As the mortality for open insertion was 26% in the 1977-1986, this method is now generally avoided. Mortality from endoscopic placement is low, but the tubes are sometimes poorly tolerated and blockage is not an infrequent complication. Expandible metal mesh stents are now available and claims of lower complication rates have been made.[10] We find that that these stents are relatively easily placed under radiological control but their expense limits their widespread use. They may be over-used in patients who should be offered a chance of a curative resection.

Laser ablation overcomes some of the disadvantages of intubation in that tumours high in the oesophagus can be treated, and a degree of flexibility and motility is maintained in the oesophagus to the extent that a relatively normal diet is tolerated by many patients. It is now the treatment of choice in palliating tumours of the true oesophagus. It is less suitable for tumours of the oesophago-gastric junction or for tumours which cannot be dilated to allow introduction of the endoscope carrying the laser. For undilatable tumours one must resort to traction intubation via laparotomy or, in dire circumstances, a jejunostomy.

At present, chemotherapy has no place in the routine palliation of oesophageal carcinoma. Current therapy is not aimed at palliation but rather at preoperative tumour shrinkage as described above, or for patients with a high probability of micrometastatic disease suggested by extra-oesophageal invasion, nodal metastases or residual disease after resection.

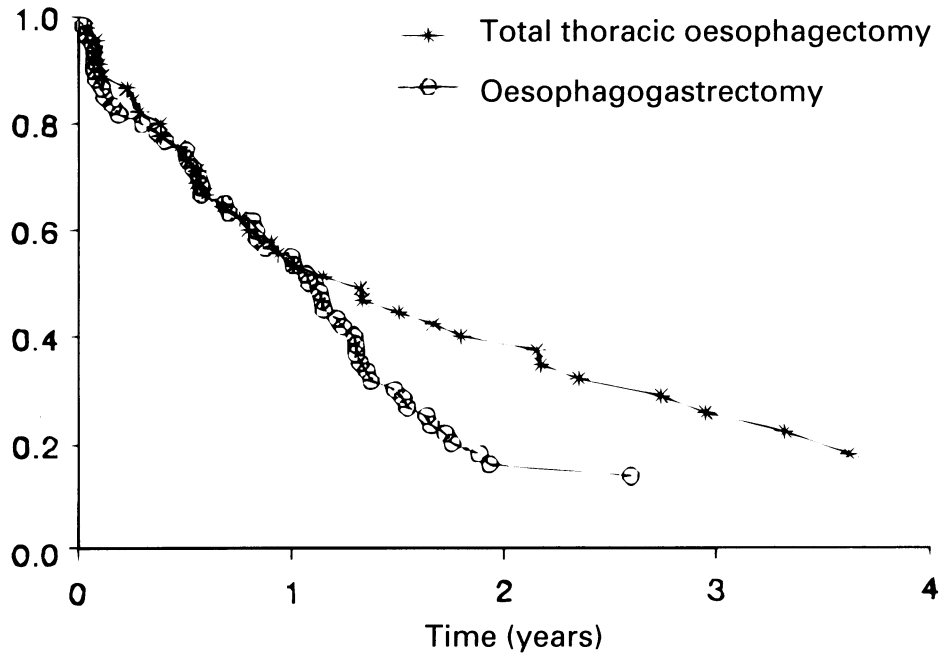

Fig. 5 Probability of survival following total thoracic oesophagectomy compared to oesophagogastrectomy (Ivor Lewis and standard) 1988-1992.

Fluid and nutritional balance can be maintained by insertion of a jejunostomy and sometimes a narrow bore nasogastric feeding tube. Neither method relieves the complete dysphagia but has the effect of prolonging life of the patient with the distressing symptom of 'drowning in his own saliva'. The limited uses of these techniques have been as a bridge to surgery, to allow a patient time to arrange his affairs or for specific social reasons. Laparotomy and jejunostomy has a high mortality rate in this setting.

Radiotherapy currently has a role in obtaining local control of tumour, both squamous and adenocarcinoma, in patients who are unfit for surgery. It also has a role in the pain control of metastatic lesions. Unfortunately, when used as palliation for dysphagia in patients with advanced disease, it is less useful. Our 1977-1986 review included a number of such patients referred for dilatation after radiotherapy. The perforation rate was 44% and mortality almost as high. The trend continues in our current practice. We would prefer to palliate dysphagia with laser ablation and then use an endo-oesophageal stent to maintain a lumen. When the disease is regarded as local, radiotherapy may then be used with curative intent.

CONCLUSIONS

Changes made in the approach to oesophageal cancer in the late 1980's have increased the chances of survival. Neoadjuvant chemotherapy and radiotherapy are on trial in an attempt to improve operability and survival. The future direction of the science of oncology will clearly involve tumour manipulation at the molecular biological level. The Royal Victoria Hospital in association with The Queen's University of Belfast is at the forefront of research into the use of these new technologies in relation to oesophageal cancer. Clinical benefits can be expected in the not too distant future. The surgeon's role is changing from being the sole hope for patients with oesophageal cancer to being one arm of a multidisciplinary approach.

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REFERENCES

1. Pera M, Trastek V F, Pairolero P C, Cardesa A, Allen M S, Deschamps C. Barrett's disease: Pathophysiology of metaplasia and adenocarcinoma. *Ann Thorac Surg* 1993; **56**: 1191-7.
2. Matthews H R, Steel A. Left-sided subtotal oesophagectomy for carcinoma. *Br J Surg* 1987; **74**: 1115-7.
3. National Confidential Enquiry into Perioperative Deaths, Royal College of Surgeons of England, London 1993.
4. Matthews H R, Powell D J, McConkey C C. Effect of surgical experience on the results of resection for oesophageal carcinoma. *Br J Surg* 1986; **73**: 621-3.
5. McManus K G, Ritchie A J, McGuigan J, Stevenson H M, Gibbons J R P. Sutures, staplers, leaks and strictures, a review of anastomoses in oesophageal resection at the Royal Victoria Hospital, Belfast, 1977-86. *Eur J Cardio-Thorac Surg* 1990; **4**: 97-100.
6. Orringer M B, Forastiere A A, Perez-Tamayo C, Urba S, Takasugi B J, Bromberg J. Chemotherapy and radiation therapy before transhiatal esophagectomy for esophageal carcinoma. *Ann Thorac Surg* 1990; **49**: 354-5.

7. Hildenberg A D, Carey R W, Wilkins E W, Choi N C, Mathisen D J, Grillo H C. Preoperative chemotherapy, surgical resection, and selective postoperative therapy for squamous cell carcinoma of the esophagus. *Ann Thorac Surg* 1988; **45**: 357-63.

8. Matthews H, Walker S, Steel A, Cullen M H. Mitomycin, ifosfamide and cisplatin: An effective, preoperative treatment for oesophageal carcinoma. *Br J Cancer* 1990; **62**: 497-8.

9. Weaver R M, Matthews H R. Palliation and survival in malignant oesophago-respiratory fistula. *Br J Surg* 1980; **67**: 539-42

10. Knyrim K, Wagner H-J, Bethge N, Keymling M, Vakil N. A controlled trial of an expansile metal stent for palliation of esophageal obstruction due to inoperable cancer. *New Engl J Med* 1993; **329**: 1302-7.

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Chemical lumbar sympathectomy in patients with severe lower limb ischaemia

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SUMMARY

Over a 13 year period, 544 chemical lumbar sympathetic blocks with phenol in 489 patients were performed by the author with the aid of X-ray image intensification. There was objective and subjective improvement in the signs and symptoms of limb ischaemia in 72%, judged by relief of rest pain, improvement in skin blood flow or healing of ischaemic ulcers. Of patients treated in the years 1990-1993, 44% had suffered either death or major amputation within two years of their treatment. Three serious and probably avoidable complications are described.

INTRODUCTION

Perhaps no procedure has generated more controversy in the treatment of peripheral vascular disease than lumbar sympathectomy. Surgical section of the lumbar sympathetic chain was first performed in 1923 to alleviate muscular rigidity associated with spastic paralysis in war wounded. The obvious effect on the lower limb vasculature was immediately apparent, and the following year an operation was carried out in the Mayo clinic for Raynaud's phenomenon. At about the same time chemical lumbar sympathectomy was being carried out in Vienna and the vascular aspect of this work was soon evident. Reid and colleagues in 1970 reported the long term follow-up results of a large series of cases with ischaemic rest pain treated with phenol sympathetic block.[1]

In the normal subject a complete sympathetic blockade leads to visibly dilated veins or increased blood flow, seen clinically in reduced capillary refilling time. Vasoconstrictor reflexes such as the cold or "ice" response are abolished. The blood flow increase is largely restricted to the skin, with an increase in skin temperature and feeling of warmth in the extremity. Skin capillary oxygen tension and venous oxygen tension are increased. Muscle blood flow is usually unaffected so that intermittent claudication may not be helped. It is not always possible to predict the effect of sympathetic blockade in a patient with a diseased vascular system as a "steal" phenomenon may occur although this is rare. The main indication for sympathetic blockade is rest pain and ischaemic cutaneous ulcers in a limb not amenable to direct arterial reconstruction. Associated coronary artery disease, pulmonary disease and other physical conditions in the aged make some patients poor candidates for surgery. The mechanism whereby the sympathetic nervous system is involved in peripheral

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pain has not been fully elucidated. The cutaneous pain threshold via a negative feedback loop is influenced by sympathetic efferents, and some afferent pathways involved in ischaemic pain are believed to pass through the sympathetic nervous system.

This paper presents a review of 544 neurolytic lumbar sympathetic blocks performed for peripheral vascular disease in the 13 year period ending December 1993. All the procedures were carried out by the author using radiological control.

METHODS

The technique used is a modification of the classical method.[2] The patient lies in the lateral position with the side to be treated uppermost, on an X-ray translucent operating table, and is lightly sedated with intravenous ketamine 20-50 mg and Diazemuls 5-10 mg. Adequate monitoring is provided by pulse oximetry. A skin wheal is raised with local anaesthetic about 10 cm lateral to the midline of the back at the level of L3 vertebra. A 22 gauge 150 mm needle is directed to strike the body of L3 and then manipulated to slide off the antero-lateral surface of the vertebral body so that the tip lies near the anterior surface of the vertebrae. A second needle is directed cephalad to lie in a similar position in relation to L2. Correct position of the needles is confirmed using a C-arm image intensifier and careful aspiration performed to ensure that the needle tip is not in a blood vessel. A small volume of 10% phenol in Conray – 420 contrast medium is injected. If X-ray confirms that the solution is spreading correctly in a thin linear fashion that conforms to the anterior edge of the vertebrae, a maximum of 2 ml of the phenol in contrast medium is injected through each needle. A thermal print-out hard copy confirming correct spread of contrast is obtained, and 1-2 ml air injected through each needle to clear the neurolytic agent before withdrawal. Patients remain in the lateral position for 20-30 minutes to limit spread of solution laterally towards the genito-femoral nerve or posteriorly towards the somatic nerves. Patients were interviewed within 24 hours and a clinical assessment made of the success of the procedure. No special tests were performed. Results were arbitrarily graded as good, improved or unchanged depending on the adequacy of relief of rest pain and the clinical appearance of increased skin blood flow and warmth in the foot.

RESULTS

Five hundred and forty-four blocks were performed on 489 patients; 55 patients had bilateral blocks, on separate occasions The male to female ratio was 1/1.05 and the average age 72 years (range 14-95). Twenty-eight patients were less than 50 years-of-age and five of these were females having bilateral treatments for severe vasospastic disease (Raynaud's phenomenon or chilblains). All patients were referred for treatment by one of four vascular surgeons. Patients were interviewed 24 hours after the block had been performed and a clinical evaluation made. Where the foot was obviously warmer, with hyperaemia and complete relief of rest pain, the block was graded as good (40%). Many of these patients spontaneously volunteered that they had had the first good nights sleep for weeks. Patients who had objective or subjective evidence of lesser degrees of warming of the foot or incomplete relief of rest pain were graded as improved (32%). Thus a favourable response was obtained in 72% of all patients. No detectable change was seen in the remaining 28% and they were

graded as unchanged or poor. Thirty-seven patients were diabetic and a favourable response to the block was obtained in 54%.

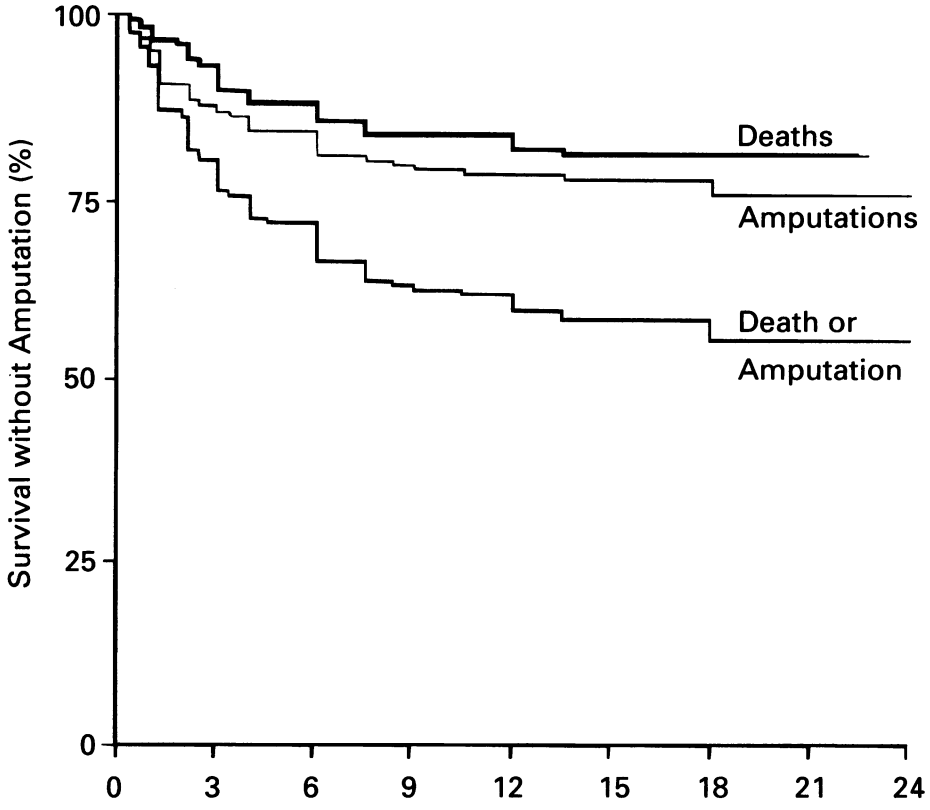

Fig. 1 Proportion of the subgroup of 148 patients remaining alive (upper line, 20% died), remaining amputation free (middle line, 24% had above or below knee amputation) or suffering neither death or amputation (lower line). Four patients who died within 6 months of amputation are not included in the deaths.

In an effort to establish the long-term fate of these patients, a chart review was undertaken of the 148 patients treated during the period January 1990 to December 1993. The mean follow-up period was 8 months (range 3 days -42 months). Twenty patients could not be traced. Thirty patients (20%) are known to have died within two years of their treatment and an additional 34 (24%) suffered a major (above or below knee) amputation. Thus 44% of this subgroup of patients suffered a major complication (death or amputation) within two years (Figure 1). Four had died within six months of amputation and these deaths are not included in Figure 1. Six patients became bilateral amputees. Fifty-two (35%) of this subgroup had good long-term improvement in the condition of the treated limb with healing of ischaemic ulcers and relief of rest pain. In five there was return of peripheral pulses which had been absent and in one only the claudication distance improved. Twelve of these 148 patients

were diabetic, of whom two were dead within a year and five had undergone a below-knee amputation within six months giving an unfavourable outcome in 58%. Three of the diabetic patients had satisfactory circulation in the treated limb at one year.

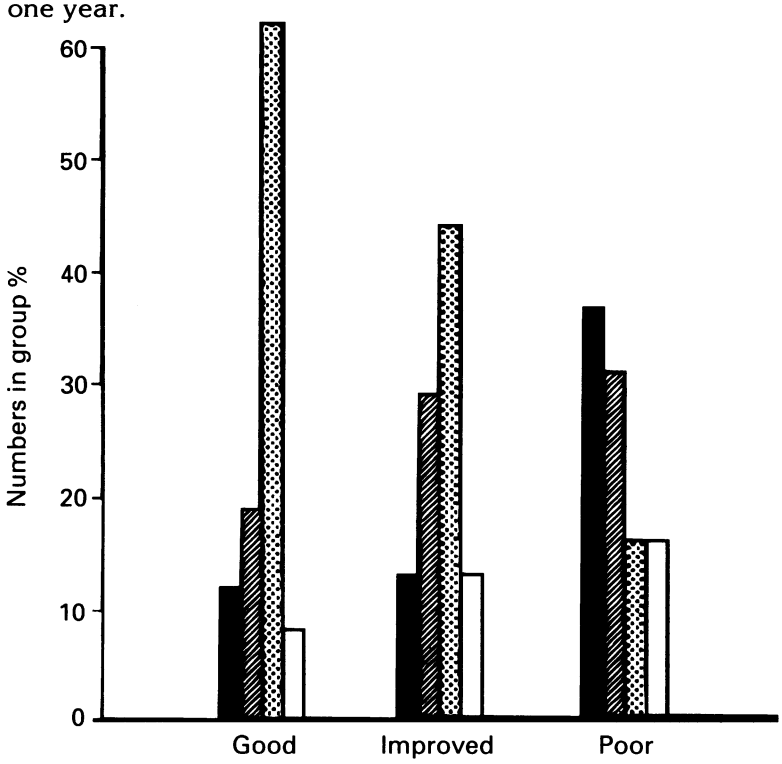

Fig. 2 The response to sympathetic block was graded as good (27), improved (52) or poor (49). Solid bars represent deaths, hatched bars amputations, stippled bars survival with satisfactory circulation, plain bars survival with continuing circulatory problems; all expressed as a percentage for each group.

Grading the success of the block as good, improved or poor according to the clinical evaluation was somewhat arbitrary, and Figure 2 is designed to justify this classification. The figure shows that in the good responders, deaths and amputations were less likely, and the chance of survival with satisfactory circulation in the treated limb high (62%). In the poor responders, death or amputation occurred in 68%. Those patients graded as improved occupied an intermediate position. The incidence of minor amputations (toes) without loss of the limb was 12%

Complications

Minor complications were more commonly observed in the earlier treatments. In the first 200 patients, nine minor adverse effects were seen, and these included hypotension (2), convulsions (3) or severe nausea and vomiting (2). Larger doses of phenol were used in these patients, and the unwanted effects were presumably caused either by accidental intravascular injection or by the systemic toxic effects of phenol.

Genito-femoral nerve irritation

Genito-femoral nerve neuritis or neuralgia is said to occur in 5-7% of patients following chemical sympathectomy and is conventionally regarded as being due to the neurolytic solution spreading to involve the genito-femoral nerve as it lies on the anterior surface of the psoas muscle where it may be close to the sympathetic chain. A similar complication can occur after surgical sympathectomy, and the numbness, burning and hyperasthesia in the groin may last for several weeks. There is a well recognised anatomical anomaly where the genito-femoral nerve is fused with the sympathetic trunk for part of its course, so some of these complications may be unavoidable.

Case 1 A 25-year-old female suffering from severe chilblains had a successful neurolytic block performed. She developed a severe and apparently permanent genito-femoral nerve lesion. Ten years later the area of hypersensitivity and tenderness is still present although less acute. Permanent genito-femoral nerve irritation has not previously been described after chemical sympathectomy.

Psoas haematoma

Case 2 A 60-year-old male diabetic patient who had had a recent myocardial infarct had an apparently technically satisfactory block performed. It was not obvious in the medical notes that he was being treated with warfarin. He subsequently complained of severe back and leg pain and a CT scan showed a large haematoma in the psoas muscle. He suffered a fatal cardiac arrest five days later.

Spinal paralysis

Case 3 A 76-year-old male diabetic patient developed a complete flaccid paralysis below the waist 12 hours after a phenol sympathetic block. There had been technical problems in getting a satisfactory spread of solution at the L3 level and it was suspected that a small amount of phenol solution was injected intra arterially. He died suddenly four months later, without any sign of neurological recovery. Autopsy revealed a recent occlusive thrombus in a severely diseased coronary artery system, infarction of the lower lumbar spinal cord, and a patent anterior spinal artery. It was suspected, but not confirmed, that this individual was one of the few (1.4%) in whom the great radicular artery of Adamkiewicz is supplied from the lumbar artery at the L3 level, and that injection of phenol into or near this dominant artery caused a direct toxic or ischaemic effect on the lower spinal cord.

DISCUSSION

Many studies have indicated that patients with rest pain, ulceration or limited gangrene are likely to derive benefit from lumbar sympathectomy. Chemical sympathectomy is a simple procedure to perform and in our hands the success rate compares favourably with that claimed using computed tomography control (72% versus 76%).[4] It can be repeated if necessary although this only occurred once in this series. We have not found special investigations particularly helpful although younger patients would usually have a local anaesthetic block performed with bupivacaine (Marcain) so that the effect of a permanent block can be assessed. Because the number of patients with vascular disease is large, complicated and time consuming physiological studies are not always feasible.

It has generally been accepted that the ankle-brachial systolic pressure ratio or index measured with a Doppler probe is important; if less than 0.35 the response to sympathectomy is likely to be poor although rest pain may be relieved. A recent study suggests that orthostatic responses to changes in limb position as measured by a duplex ultrasound flowmeter may be used to predict the clinical outcome to sympathectomy.[5]

In a recent review Cotton and Cross concluded that sympathectomy was a good procedure in end-stage disease when no other option was open;[6] there was little to support the addition of sympathectomy to a synchronous vascular reconstruction as a way of improving graft patency; sympathectomy probably does not postpone amputation or influence the level at which this will be required. In general, the most beneficial effect of sympathectomy was relief of rest pain, with a somewhat lesser effect of improved healing of ulcers. Increased walking tolerance was of benefit in a small group of patients with intermittent claudication. Occasional dramatic deterioration has been described but is sufficiently rare not to prevent sympathectomy from being widely used in the treatment of ischaemic pain. Their general conclusion was that the use of chemical sympathectomy in the control of ischaemic rest pain and pregangrene in the lower limb is at least as effective as open operation and the high morbidity and mortality of the latter is considerably reduced.[6]

The use of sympathectomy in diabetic patients has been questioned by some workers. Although diabetic ischaemic disease increased the risk of failure of sympathectomy, 54% of diabetics in the present series responded favourably initially and in those patients who were followed up for a longer period, 25% had a good long term result. Our numbers are small but a 58% incidence of major complication (death or amputation) within a year as against 44% for the group as a whole would suggest that diabetics do less well, although some worthwhile results can be obtained. Two recent publications take the view that diabetic status has no prognostic value, and should be ignored.[7,8] Of equal concern are the increasing numbers of renal transplant and chronic dialysis patients who are being referred with critical ischaemia.

I feel that chemical lumbar sympathectomy is useful in the treatment of patients with severe lower limb ischaemia manifest as rest pain, ulceration or toe gangrene, where arterial reconstruction is not feasible for technical or medical reasons. It is of doubtful value in patients with massive tissue necrosis, deep infection or forefoot gangrene, and the inability to salvage an effective walking surface of the foot will ensure that the procedure is not successful. It can be done as a day procedure, although our preference would be that the patient stays in hospital the night following the block. The very serious complications seen in three patients could probably have been avoided. In many patients critical limb ischaemia is an end-stage phenomenon, and almost 50% will suffer major amputation or death within two years. Some patients are unable or unwilling to stop smoking, and progression of their disease is inevitable.

ACKNOWLEDGEMENT

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REFERENCES

1. Reid W, Watt J K, Gray T C. Phenol injection of the sympathetic chain. *Br J Surg* 1970; **57**: 45-50.
2. Cousins M J, Reeve T S, Glynn C J, Walsh J A, Cherry D A. Neurolytic lumbar sympathetic blockade; duration of denervation and relief of rest pain. *Anaesth Intens Care* 1979; **7**: 121-35.
3. Cross F W, Cotton L T. Chemical lumbar sympathectomy for ischaemic rest pain. *Am J Surg* 1985; **150**: 341-5.
4. Dondelinger R, Kurdziel J C. Percutaneous phenol neurolysis of the lumbar sympathetic chain with computed tomography control. *Ann Radiol* 1984; **27**: 376-9.
5. Morgan P H, Psaila J V, Stone J, Carolan G, Woodcock J P. Postural changes in femoral artery blood flow in normal subjects, patients with peripheral vascular occlusive disease and patients undergoing lumbar sympathectomy, measured by duplex ultrasound flowmetry. *Eur J Vasc Surg* 1992; **6**: 408-15.
6. Cotton L T, Cross F W. Lumbar sympathectomy for arterial disease. *Br J Surg* 1985; **72**: 678-83.
7. Aburahma A F, Robinson P A. Clinical parameters for predicting response to lumbar sympathectomy in patients with severe lower limb ischaemia. *J Cardiovasc Surg* 1990; **31**: 101-6.
8. Baker D M, Lamerton A J. Operative lumbar sympathectomy for severe lower limb ischaemia: still a valuable treatment option. *Ann R Coll Surg Engl* 1994; **76**: 50-3.

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Complications associated with central venous catheters in a haematology unit

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SUMMARY

The use of central venous catheters in patients suffering from haematological disorders has brought enormous benefits, but has been associated with an increase in septicaemia. We have reviewed septic and other complications in 43 patients who received one of three different forms of central venous catheters (type A – Hickman®, type B – Portacath®, type C – Pasport®) during 1991. All complications were reviewed up to 18 months following insertion. The total complication rate was 31% (0.97 per 100 catheter days), and the total sepsis complication rate was 18.8% (0.49 per 100 catheter days). Type A catheters had the greatest sepsis complication rate of 29.5% (0.84 per 100 catheter days), with type B 15% (0.39 per 100 catheter days) and type C 9.9% (0.32 per 100 catheter days). Prophylactic antibiotics on the day of catheter insertion did not reduce the sepsis rate or prolong catheter survival.

INTRODUCTION

Many of the patients suffering from haematological malignancies benefit from the insertion of indwelling central venous lines as these provide reliable, pain free access for intravenous therapy (chemotherapy, intravenous fluids, blood transfusions etc), venous sampling and total parenteral nutrition.[1, 2] Three different central venous catheters are inserted in our unit. Type A (Hickman®) lines are hollow silicone-rubber catheters which have central venous access through the internal jugular vein and exit, after a subcutaneous course, through the anterior chest area. Type B (Portacath®) catheters also gain central venous access through the internal jugular vein but, instead of coming out through the skin, end with a subcutaneous reservoir in the lower anterior chest area. This requires to be "plumbed in" using a specially designed needle prior to use. Type C (Pasport®) catheters also have a subcutaneous reservoir but this lies in the forearm and the line gains venous access through the antecubital route. Type A lines are generally inserted for those patients about to embark on intensive chemotherapy, including autologous bone marrow transplantation, while type B and C lines are reserved for those receiving intermittent chemotherapy or rarely in those who are judged incapable of looking after a type A catheter. Patients find these catheters of great benefit and the vast majority master the techniques of long term catheter care in the case of the type A line. Both the

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type B and C catheters have resulted in increasing numbers of patients being able to have chemotherapy at home via a controlled pump infusion. However, use of the catheters has been associated with an increased incidence of septicaemic episodes.[3]

The aim of our study was to evaluate the complication rate (both septic and otherwise) in the three different central lines inserted in haematology patients during the year of 1991, following all these patients over an 18 month period from the date of insertion.

PATIENTS AND METHODS

Patient Population

This was a retrospective study reviewing the medical records of all the patients who had Type A, B or C line insertions during the year 1991. The patients were followed for a maximum period of 18 months from the date of line insertion. Forty-three patients had line insertions during the year– their clinical diagnoses are listed in Table 1. There were 21 males and 22 females, mean age 41.7 ± 15.3 years (1 SD). It became the policy of the unit to give routine antibiotic prophylaxis to all patients undergoing central catheter insertions in May 1991– this consisted of teicoplanin 200mg iv 2 hours prior to surgery and a further 200mg iv at induction of anaesthesia. The basis for this regime was to attempt to achieve maximal plasma and tissue concentrations of teicoplanin at the time of line insertion. Two groups of patients resulted – those who received antibiotic prophylaxis and those who did not prior to the establishment of this policy.

TABLE 1

Clinical diagnoses of patients in study.

Diagnosis	*Number of patients*
Acute myeloid leukaemia	2
Acute lymphatic leukaemia	6
Hodgkins disease	3
Non-Hodgkins lymphoma	9
Multiple myeloma	19
Plasma cell leukaemia	1
Synovial cell sarcoma	1
Myelodysplasia	1
Osteosarcoma	1

Catheter insertion

There was no formal policy for the type of catheter inserted but, in general, those who were to receive intensive chemotherapy, including autologous marrow transplantation, had a type A catheter inserted. Type C catheters were reserved for those with reasonable venous access in the arms, especially young females where the cosmetic acceptability of this type of line was beneficial. The technique of catheter insertion was not specified by a predetermined protocol but all were inserted by the same surgical team using a strict aseptic technique similar to that described by Heimbach and Ivey.[4] All the patients had general anaesthesia with the exception of 3 patients who had local anaesthesia.

Patients with low platelet counts (<50 x 10^9/L) were given platelet transfusions before and during the procedure.

Following the procedure wound care was the responsibility of the nursing staff in the ward. Type B and C catheters did not require intensive wound management and clips were generally removed after 7 days. However with type A catheters management involved cleansing of the exit site on a daily basis with chlorhexidine and povidone iodine solutions. The catheter was flushed daily with 10 mls of heparinised saline when the line was not in use. All of the patients were given supervised training by nursing staff with regard to this procedure, and were subsequently able to perform this themselves at home.

Review of Complications

Septic and other complications (i.e. thrombosis, extravasation of chemotherapy, malposition of catheter tip, accidental withdrawal and haemorrhage) were reviewed over an 18 month period (maximum) from the date of insertion of the catheter. For this study all septic episodes were classified into three separate categories. 1. Catheter related septicaemia: an episode of microbiologically proven septicaemia in culture from the catheter or peripheral blood, or bacterial growth from the catheter tip. 2. External exit site infection: indicated by either erythema, tenderness, palpable thrombosed vein or pus around the site. This was subsequently confirmed by positive swab culture. 3. Clinically suspicious catheter infection: fever and malaise with no apparent clinical signs of line infection such as erythema or tenderness, no localising signs of other infection and no positive blood cultures, but responding to either teicoplanin or vancomycin.

Cumulative catheter survival was calculated using Kaplan-Meier survival plots, and the log-rank test was used to compare differences in survival between the three lines. Deaths occurring unrelated to line complications, patients with lines in situ at 18 months or with lines electively removed because treatment had finished were "censored" in the analysis. The chi-squared (χ^2) test, with Yates correction, was used to assess for statistical differences between the occurrence of septic episodes in those who had received prophylactic antibiotics and in those who had not.

RESULTS

Thirteen patients received a type A (Hickman®) catheter, 20 patients a type B (Portacath®) catheter and ten patients a type C (Pasport®) catheter. Four patients received a second type A catheter following removal of the initial catheter and 1 patient required insertion of a second type C catheter following removal of the first, giving a total of 48 catheter insertions. The following results refer to the complications in catheters as opposed to patient numbers. Overall the cumulative catheter survival was 73.1% at six months and 66.9% at both 12 and 18 months. There was a cumulative total of 12,734 catheter days, a total complication rate of 31% or 0.97 per 100 catheter days and a total septic complication rate of 18.8% or 0.49 per 100 catheter days. There were no statistically significant differences in the cumulative survival of the three different lines up to six months or between type B and C catheters up to 18 months (log rank test). Results for the individual catheters are summarised in Table 2.

TABLE 2

Summary of results for three different lines.

	Type A	*Type B*	*Type C*
Number inserted	17	20	11
Cumulative total days	2757	6857	3120
Complication free	10 (59%)	15 (75%)	8 (73%)
Sepsis complication	5 (29.5%)	3 (15%)	1 (9%)
(per 100 catheter days)	0.84	0.39	0.32
Non-Sepsis complication	2 (11.5%)	2 (10%)	2 (18%)
Cumulative catheter survival			
6 months	63.7%	78.6%	79.5%
12 months	—	78.6%	66.3%
18 months	—	78.6%	66.3%

With type A catheters, four patients developed catheter related septicaemia, necessitating removal of the lines following failure of antibiotic therapy. These were subsequently replaced and no further complications occurred with the new lines. One other patient had a clinically suspicious catheter infection that responded to teicoplanin. All of the patients suffering from acute leukaemia (n=8) received type A catheters and it was four of these patients who developed the catheter related septicaemia. The other type A catheters were inserted in patients with relapsed non-Hodgkins lymphoma (n=4) and Hodgkins disease (n=1) who required marrow ablative chemotherapy. Three patients died with their lines *in situ* from causes unrelated to catheter sepsis. In the remaining patients catheters were electively removed following treatment, with all of the lines having been removed (or the patient having died) after six months. The only non-septic complications seen were two lines that were accidentally withdrawn.

With type B catheters, three patients developed catheter related septicaemia but all responded to intravenous antibiotics. Eight patients died with their lines *in situ* from causes unrelated to catheter sepsis, the others surviving the 18 month study period. The non-septic complications were one catheter induced subclavian vein thrombosis and one extravasation of chemotherapy.

With type C catheters, there was no catheter related septicaemia, but one episode of exit site infection occurred that responded to antibiotics. Four of the patients died with their lines *in situ* from causes unrelated to catheter sepsis, the others surviving at 18 months. The non-septic complications included one catheter tip lying in the wrong position and another of extravasation of chemotherapy.

Out of a total of 48 catheter insertions, 18 were carried out without prophylactic antibiotic cover (seven type A, seven type B, four type C). There was no statistical difference in cumulative catheter survival between those who had received prophylactic antibiotics (n=30) and those who had not (n=18) [log-rank test, $p>0.5$]. Similarly, there was no statistical difference between the number of septic episodes in the two subgroups, either in the initial four weeks following the insertion (three episodes in the prophylactic antibiotic group as opposed to two in the non-prophylactic group, $\chi^2=0.22$, $p>0.5$) or overall (five episodes compared to four $\chi^2=0.73$, $p>0.25$). Comparison of the individual lines was not possible because of the small numbers involved.

There were seven episodes of catheter related septicaemia caused by *Staphylococcus epidermidis*, one episode of exit site infection caused by *Staphylococcus aureus* and one episode of clinically suspicious line infection. All the episodes of septicaemia secondary to catheter infection were caused by coagulase negative staphylococci and despite treatment with the most effective antibiotics against these organisms (teicoplanin and vancomycin), four of the catheters had to be removed. The one episode of exit site infection related to *Staphylococcus aureus* responded rapidly to appropriate antibiotics.

Non-septic complications accounted for about 50% of the total complications. The one episode of subclavian vein thrombosis required treatment with thrombolytic agents and subsequent removal of the line, with delay in delivering the next course of chemotherapy. The two cases of extravasation probably occurred due to the needle either not being correctly positioned in the subcutaneous reservoir or having slipped out from the reservoir during the infusion. Both these patients were very ill and died within a short period of time from other causes unrelated to the extravasation. The two type A lines that were accidentally withdrawn did not require replacement and the type C catheter, whose tip lay in the internal mammary vein, had to be removed and replaced before treatment could commence.

DISCUSSION

Central venous catheters have increased in popularity due to the improved quality of life associated with them. Patients suffering from haematological malignancies require frequent venepuncture both to monitor therapy and to administer chemotherapeutic agents. Many of these agents cause phlebitis and there is a high risk of extravasation of cytotoxic agents that may cause considerable discomfort and even tissue necrosis. Because of these factors central venous access is seen almost as an essential step before the patient undergoes treatment. All the catheters are made of silicone rubber which is less thrombogenic than other materials,[5] they can be inserted with relative ease surgically and many agents and substances can be safely given through them.[1] Patients find them acceptable, they are involved in the care of the lines in the ward and at home and their quality of life is improved to a great extent, which is an important factor for many of those whose long term outlook is not good.

The total number of complications at first sight appears to be high but represents only 0.97 complications per 100 catheter days overall. Sepsis is the most common complication (0.49 per 100 catheter days) but there is considerable variation in septic complications with the type of line inserted; the type B and C being considerably lower than the type A catheters. Our results

are reasonably comparable with those reviewed by Press et al[6] and others.[3, 7, 8] The type A catheter is inserted in patients who are more unwell to start with and undergoing intensive chemotherapy. It is also more susceptible to infection because it is exposed externally and requires greater care and attention from the patient. Many of the patients undergoing line insertions are very ill and may have low blood counts. They can develop episodes of pyrexia, on the basis of presumed infection, but blood cultures are often negative and there is no other microbiological proof of sepsis other than response to empirically prescribed antibiotics. The catheter may well be the source of sepsis in some of these cases.

Most instances of catheter infection are caused by coagulase negative *Staphylococcus epidermidis*,[6, 9, 10] with at least half the strains producing an extra-cellular "glue" substance which causes adherence of the organisms to the catheter wall and protects them from the action of antibiotics. Even the most effective antibiotics against these strains are only effectual in up to one third of cases. Once a central line is infected it may well have to be removed as antibiotic treatment alone is not always successful — four out of 8 (50%) in this study. A study by Larson et al[3] reported septicaemia in 38.3% of patients with acute leukaemia who received type A lines and Hickman reports similar figures in marrow transplant patients[11] — these results are similar to our own. There is some evidence that prophylactic antibiotics given at the time of insertion have a role in preventing later catheter related sepsis and in prolonging catheter life.[3, 9, 12] In our study we were unable to demonstrate any difference in cumulative survival or septic complication rates following the introduction of prophylactic teicoplanin but numbers are small and a larger randomised study would be required.

Although sepsis represents the majority of complications seen with these central catheters and is an important cause of morbidity, mortality appears to be very small and indeed, none of the patients died from septicaemia secondary to catheter infection. Other non-septic complications seen include damage to the lines, venous thrombosis and blocking of the catheters. There are thrombolytic agents available (urokinase) to unblock catheters and repair kits are available for the repair of type A catheters.[13] Non-septic complications accounted for about 50% of the total complications overall, and played an increasing role in the lines less susceptible to septic complications. All complications are associated with extra cost to the hospital in terms of additional medical and nursing care, and therefore all possible precautions should be taken to attempt to decrease the occurrence of these episodes. The non-septic complications may be more amenable to prevention than the septic ones. The risk of extravasation of chemotherapeutic agents can be minimized by carefully checking that the needle is lying in the subcutaneous reservoir and subsequently securing this firmly to prevent it from slipping out. Radiological assessment, with the use of radio-contrast dye, can be used if doubt exists and this will also reveal the position of the catheter tip. The risk of accidental withdrawal of type A catheters can be decreased by good patient education and ensuring that the line is well secured.

In conclusion these catheters improve quality of life although septic complications are a significant problem. Fortunately, the vast majority of these episodes can be satisfactorily controlled with antibiotics. Increasing experience in the use of

these catheters, both by patients and by medical/nursing staff, and further improvement in the design and materials used in manufacture may lead to reduction in septic complications in the future. The role of prophylactic antibiotics requires further intensive investigation.

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REFERENCES

1. Ivey M F, Adam S M, Hickman R O, Gibson D L. Right atrial indwelling catheter for patients requiring long term intravenous therapy. *Am J Hosp Pharm* 1978; **35**: 1525-7.
2. Mahan J D, Mauer S M, Nevins T E. The Hickman catheter: a new haemodialysis access device for infants and small children. *Kidney Int* 1983; **24**: 694-7.
3. Larson E B, Wooding M, Hickman R O. Infectious complications of right atrial catheters used for venous access in patients receiving intensive chemotherapy. *Surg Gynecol Obstet* 1981; **153**: 369-73.
4. Heimbach D M, Ivey T D. Technique for placement of a permanent home hyperalimentation catheter. *Surg Gynecol Obstet* 1976; **143**: 634-6.
5. Boros S J, Thompson T R, Reynolds J W, Jarvis C M, Williams H J. Reduced thrombus formation with silicone elastomer (silastic) umbilical artery catheters. *Paediatrics* 1975; **56**: 981-6.
6. Press O W, Ramsey P G, Larson E B, Fefer A, Hickman R O. Hickman catheter infections in patients with malignancies. *Medicine* 1984; **63**: 189-200.
7. Thomas M. The use of the Hickman catheter in the management of patients with leukaemia and other malignancies. *Br J Surg* 1979; **66**: 673-4.
8. Reed W P, Newman K A, de Jongh C et al. Prolonged venous access for chemotherapy by means of the Hickman catheter. *Cancer* 1983; **52**: 185-92.
9. Mackinnon S, Garden O J, Gribben J G, Burns H J G, Baird D, Burnett A K. The use of Hickman catheters in marrow transplant recipients. *Int Ther Clin Monit* 1987; **4**: 122-6.
10. Blacklock H A, Hill R S, Clarke A G, Pillai M V, Matthews J R D, Wade J F. Use of modified subcutaneous right-atrial catheter for venous access in leukaemic patients. *Lancet* 1980; **1**: 993-4.
11. Hickman R O, Buckner D, Clift R A, Sanders J E, Stewart P, Thomas E D. A modified right atrial catheter for access to the venous system in marrow transplant recipients. *Surg Gynecol Obstet* 1979; **148**: 871-5.
12. Mullan F J, Hood J M, Barros D'Sa A A B. Use of the Hickman catheter for central venous access in patients with haematological disorders. *Br J Clin Prac* 1992; **46**: 167-70.
13. Bjeletich J. Repairing the Hickman catheter. *Am J Nurs* 1982; **82**: 274.

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Myotonic dystrophy: relative sensitivity of symptoms signs and abnormal investigations

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SUMMARY

Twenty-five symptoms, signs, and abnormal investigations were looked for in 20 patients with clinically-definite myotonic dystrophy. Weakness of facial muscles, neck flexors, and arm external rotators was found in all patients (sensitivity=100%). Arm external rotation has not been reported as a frequently involved muscle in previous clinical studies on myotonic dystrophy. Careful examination of muscle strength may therefore predict which patients may or may not carry the abnormal gene for myotonic dystrophy.

INTRODUCTION

Myotonic dystrophy is the commonest inherited neuromuscular disease as well as the one most varied in its clinical expression.[1] The myotonic dystrophy gene has recently been localised to the q13.3 region on the long arm of chromosome 19 where there are increased numbers of cytidine-thymidine-guanidine trinucleotide repeats.[2] This has enabled the diagnosis to be made on DNA extracted from peripheral blood.

Before the availability of a blood test there were numerous clinical evaluations to determine the sensitivity and specificity of various symptoms, signs, and investigations in myotonic dystrophy. These have become no less relevant now since not everyone with weakness or cataract is likely to have a gene test without some preliminary screening. Furthermore, with the existence of a gene test, there is now a "gold-standard" against which other measures can be judged. We were impressed that the frequency of muscle weakness on careful neuromuscular examination of patients with myotonic dystrophy was higher than that reported in the literature. We carried out the following study to test this hypothesis before the gene test was available.

PATIENTS AND METHODS

Twenty patients attending the Northern Ireland Muscle Clinic were studied. All patients had weakness, myotonia on either clinical examination or electromyography, and a family history compatible with autosomal dominant inheritance. Males and females were represented equally and ages ranged from 17 to 61 years with a mean of 34 years. Each patient was examined by M A who

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was experienced in the clinical evaluation of patients with neuromuscular disease. The symptoms, signs, and investigations set out in the table were recorded as present or absent; the investigations were generally available from the patients' records, but symptoms and signs were determined by clinical evaluation.

Symptoms	Falls	Hypersomnolence
	Dysphagia	
Signs	Cataracts	Testicular atrophy
Weakness	Eyelids	Proximal arms
	Eye muscles	Finger abduction
	Dysarthria	Hip flexion
	Face	Knee flexion
	Neck flexion	Knee extension
	Neck extension	Ankle dorsiflexion
	Arm external rotation	Ankle plantarflexion
Myotonia	Action	Percussion
Investigations	Raised CK	Myopathic EMG
	Abnormal ECG	Myotonic EMG
	Abnormal biopsy	

Muscle strength was recorded manually using the MRC scale and classified as either normal (grade 5) or abnormal (grade<5). Action myotonia was assessed by handgrip and percussion myotonia from thenar eminence and finger extensors. Electromyographic studies were carried out with concentric needle electrodes usually from deltoid, biceps, abductor digiti minimi, quadriceps and tibialis anterior. Muscle biopsies fom quadriceps were taken using a UCH needle and frozen in isopentane cooled in liquid nitrogen. Cryostat sections were stained with haematoxylin and eosin, Gomori's trichrome, acid phosphatase, NADH-tetrazolium reductase and ATP-ase pre-incubated at pH 9.4, 4.6, and 4.3.

RESULTS

The frequency of weakness in each muscle group is shown in figure 1 and that of the remaining symptoms, signs, and investigations in figure 2. Face, neck flexors, and arm external rotators were universally weak whereas knee flexion and neck extension were rarely involved. Myotonia was by definition present but was more reliably obtained by percussion than by handgrip. Cataract was the commonest somatic feature but was much less prevalent than weakness and myotonia.

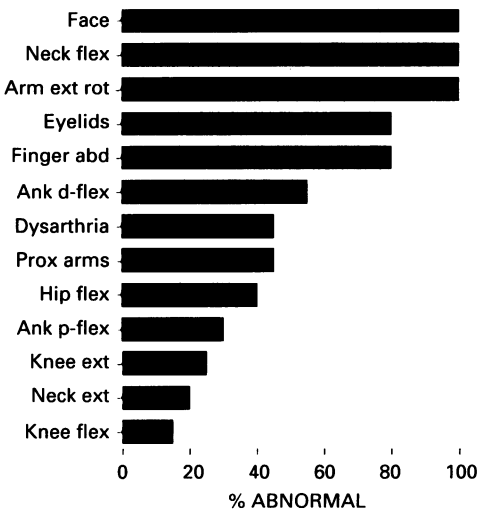

Fig. 1 Frequency of muscle weakness.

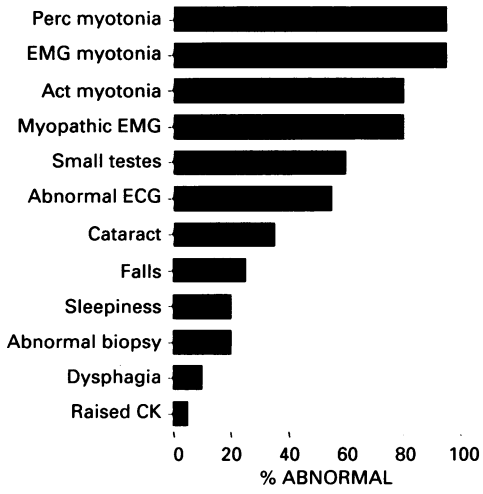

Fig. 2 Frequency of other abnormalities.

DISCUSSION

The measures which we studied differed greatly in their frequency of occurrence in patients with myotonic dystrophy. Weakness of the face, neck flexors, and arm external rotators achieved a sensitivity of 100%. Whereas the first two are well-known to be involved in this disease, weakness of the external rotators of the arm does not seem to have been commented on elsewhere as a frequent occurrence. This movement is carried out by relatively small muscles, principally infraspinatus, and the examiner tests these using pressure on the wrist with the elbow at 90°. The forearm then acts as a lever which generates a considerable torque. Lesser degrees of weakness may therefore be apparent than when a larger muscle with a smaller moment such as deltoid, is tested.

Apart from myotonia which was a criterion for inclusion the other variables showed relatively low sensitivity. The low frequency of cataracts was probably related to use of an ophthalmoscope with a +20D lens rather than a slit-lamp. Results on sensitivity of cataracts in myotonic dystrophy have varied but in a recent study this was 86% for all types of cataract compared with 61% for orbicularis oculi weakness.[3] Muscle biopsy was relatively insensitive with only 20% of biopsies showing the so-called "characteristic" appearances of myotonic dystrophy – increased central nuclei and type 1 fibre atrophy.

The finding of a group of abnormalities with 100% sensitivity would enable the diagnosis to be excluded in their absence and thus would be a useful screening procedure prior to gene testing. This would still be necessary as weakness of face, neck flexion, and arm external rotation is certainly not specific to myotonic dystrophy. This high sensitivity was obtained on an obviously affected group of patients, albeit relatively young. It remains to be seen whether they will be as sensitive in at-risk first degree relatives of myotonic dystrophy patients. The advent of a specific gene test will thus enable the value of careful clinical examination to be tested definitively.

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REFERENCES

1. Harper P S. Myotonic dystrophy. London: *Saunders*, 1989.
2. Harley H G, Rundle S A, Reardon W, et al. Unstable DNA sequence in myotonic dystrophy. *Lancet*, 1992: **339**: 1125-28.
3. Ashizawa T, Hejtmancik J F, Liu J, Perryman M B, Epstein H F, Koch D D. Diagnostic value of ophthalmologic findings in myotonic dystrophy: comparison with risks calculated by haplotype analysis of closely related restriction fragment length polymorphisms. *Am J Med Genet*, 1992; **42**: 55-60.

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Changes in the pattern of deliberate self-poisoning presenting at Craigavon Area Hospital: 1976, 1986 and 1991

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SUMMARY

Deliberate self-poisoning presenting at Craigavon Area Hospital in 1991 was examined and compared to the years 1976 and 1986. Self-poisoning has not declined over the 15 year period 1976-1991. The reduction in the use of benzodiazepines, and increase in paracetamol, previously reported, continues. Possible reasons for this are examined, in relation to local and national drug prescribing.

INTRODUCTION

Deliberate self-poisoning is defined as a deliberate, non-fatal drug overdose, done in the knowledge that it is potentially harmful and that the amount taken is excessive.[1] It is a major public health problem, being the second most common cause of emergency medical admissions of men, and the most common cause of female admissions.[2]

Monitoring of deliberate self-poisoning is important, as after a suicide attempt there is a considerable risk of eventual suicide. During the year after a suicide attempt, about 1% of adults die by suicide, and the risk remains increased several years later. [3,4] If prevention of suicide is to be achieved, this high-risk group will require targeting, and interventions which reduce the lethality of their chosen methods of self-harm should be considered. In Northern Ireland 18% of suicides occur by self-poisoning with drugs.[5]

Inter-regional variations in patterns of self-poisoning are known to exist[6,7] as are changes in pattern over time and in age and sex distribution.[8,9,10] An ongoing assessment of self-poisoning presenting at this hospital in 1976 and 1986 has been reported previously,[11] and this study examines the phenomenon in the same area in 1991 and compares it with the previous years. Changes in frequency of presentation to hospital may be due to a true fluctuation in incidence or to altered referral patterns by general practitioners. We have identified general practitioner management of deliberate self-poisoning in this

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area. It has also been assumed that drugs used for deliberate self-poisoning reflect those currently available,[12] and that successful suicides are often related to the lethality of the drug. We have related our experience of deliberate self-poisoning to both local and national drug use, where such data exists.

METHODS

Craigavon Area Hospital is the only hospital serving three local district council areas which have a stable, mixed urban/rural population which has grown from 146,000 to 160,000 between the years 1976 - 1991.

The accident and emergency department records for 1991 were examined retrospectively. All cases of self-poisoning were identified and relevant medical and psychiatric notes surveyed. The following categories were excluded: age under ten, the use of alcohol alone, and accidental self-poisoning (as defined by the relevant medical officer). 289 cases of deliberate self-poisoning by 252 individuals were identified, and notes were available in all cases. Repeat presentations (two or more episodes by three individuals during 1991 were not excluded, to allow for comparison with the 1976 and 1986 figures.

The 95 general practitioners whose catchment areas are served by this hospital were surveyed by postal questionnaire to ascertain whether their tendency to refer cases of self-poisoning had changed over the last 15 years.

Quantities of the relevant drugs prescribed, or sold over the counter, in the Southern Health and Social Services Board area (where such data existed) were compared with their frequency of use in overdosage in the years studied. This data was provided by the Drug Utilization and Research Unit, Department of Therapeutics, The Queen's University of Belfast, by courtesy of Dr Hugh McGavock and Miss Elaine Milligan.

Analysis was by the SPSS (Statistical Package for the Social Sciences) computer package, using chi-squared tests where appropriate.

RESULTS

Demographic data

There were 289 recorded episodes of deliberate self-poisoning in 1991 (1.81/1,000 of population), compared to 228 (1.48/1000) in 1986, and 265 (1.82/1,000) in 1976. These figures allow for the local population increase 1976-1991; there was no statistically significant difference in the years in question. The sex ratio remained unchanged at approximately 1.5 female: 1 male in each of the three years studied.

While the 1986 results showed a reduction in modal age of those self-poisoning compared with 1976 (from 30-40 years to 10-20 years of age), this trend was not continued in 1991 (modal age 20-30 years).

Drug and alcohol use

The drugs taken in deliberate self-poisoning are shown in Table 1. The use of benzodiazepines alone has shown a marked fall, from 40.8% in 1976 to 14.5% in 1991 (X^2=48.25, $p<0.001$.), as have the total number of cases where benzodiazepines were used either alone or in combination, from 169 in 1976 to 68 in 1991 (X^2=76.12, $p<0.001$.). Paracetamol has replaced the

benzodiazepines as the single most commonly used drug, both alone and in combination with other drugs. Alone, its use has increased from 0.8% of cases in 1976 to 17.3% in 1991 (X^2=44.72 p < 0.001.). When combinations of drugs including paracetamol are added, the number of cases has increased from 31 in 1976 to 93 in 1991 (X^2=33.54, p<0.001.).

TABLE 1

Drugs used for deliberate self-poisoning

	1976 n=265	*1986 n=228*	*1991 n=289*
Benzodiazepines	108 (40.8%)	61 (26.8%)	42 (14.5%)*
Paracetamol	2 (0.8%)	21 (9.2%)	50 (17.3%)*
Aspirin	17 (6.4%)	9 (3.9%)	18 (6.2%)
Neuroleptics	5 (1.9%)	2 (0.9%)	10 (3.5%)
Antidepressants	17 (6.4%)	9 (3.9%)	18 (6.2%)
Anti convulsants	5 (1.9%)	3 (1.3%)	4 (1.4%)
Anti inflammatory	2 (0.8%)	12 (5.3%)	13 (4.5%)
Combination without psychotropic drugs	7 (2.6%)	32 (14.0%)	43 (14.9%)
Combination with psychotropic drugs	53 (20.0%)	50 (21.9%)	50 (17.3%)
Unknown, others	49 (18.4%)	29 (12.8%)	33 (11.4%)

* p<0.001

No statistically significant difference was noted in the use of anti-depressants, salicylates, or neuroleptic drugs from 1976 to 1991. There had been a statistically significant increase in the use of non-steroidal anti-inflammatory drugs from 1976 to 1986, alone and in combination (X^2=20, P<0.001), but there was no further increase in 1991. Alcohol had also been ingested by a significant, unchanging (36%-38%) minority of patients in each of the three years.

Outcome

The number of patients discharged without follow-up has increased from 17% to 48% (X^2=58.52, p<0.001); concurrently the number admitted to the psychiatric unit fell from 45% in 1976 to 18% in 1991 (X^2=71.99, p<0.001.) There was little change in the number of patients followed up in the out-patient department, or of those who left the hospital contrary to advice.

TABLE 2

Outcome of patients

	1976 n=265	*1986 n=228*	*1991 n=289*
Discharged/no follow-up	46 (17.4%)	86 (35.5%)	140 (48.4%)
Out patients	43 (16.2%)	48 (21.1%)	53 (18.3%)
Admitted psychiatric unit	120 (45.3%)	36 (15.8%)	52 (18.0%)
Left contrary to advice	31 (11.7%)	50 (22.0%)	41 (14.2%)
Unknown	25 (9.4%)	8 (3.5%)	3 (1.0%)

General practitioner referral patterns

79 replies (83% response rate) were received to the postal questionnaire. Of these, 85% said there had been no changes in the referral pattern, 13% said they were more likely to refer and 3% less likely to refer cases to hospital. Several practitioners commented that referrals were always made, because of the difficulty in ascertaining quantities and types of drugs ingested.

Drug prescribing patterns

In view of the suggestions that patterns of self harm alter with availability of method,[12] general practice prescribing figures for benzodiazepine drugs were obtained from the Northern Ireland Drug Utilisation Research Unit (table 3). The data is for the Southern Health and Social Services Board area (which includes the area served by Craigavon Area Hospital) and is for 1981 and 1991 only; no comparable data exists for 1976.

TABLE 3

General practice prescriptions of hypnotics, and of sedatives/tranquillisers containing benzodiazepines in the Southern Health and Social Services Board area (data from the Drug Utilization Research Unit QUB)

Preparations containing benzodiazepines	*1981*	*1991*	*% change*
Hypnotics	83,951	103,444	+23.2%
Sedatives/ tranquillisers	146,298	105,356	-28%^
TOTAL:	230,249	208,800	-9.3%

There has been an overall fall of 9% in benzodiazepine prescriptions. These figures cannot be directly compared with the figures of self-poisoning at Craigavon Area Hospital as they come from different populations, albeit largely overlapping. They do however provide an indication that the decline in use of benzodiazepines to self-poison appears greater than the decline in their availability.

DISCUSSION

This study is the second in a planned series[11] to look at changes in the pattern of deliberate self poisoning at five yearly intervals in a stable population. The data confirm earlier studies which identified a decrease in the rate of self poisoning in the first half of 1980's,[6,9] followed by a reversal of the trend in the second half of the decade.[14] There has been no significant trend in the age of those who self-poison, nor any change in the sex ratio, which remains at approximately 1:1.5, male: female, although other studies have noted a narrowing of this difference.[15]

General practitioner referral patterns could significantly affect the detected incidence of deliberate self-harm,[16] but our postal survey suggests that this is not the case in this area.

The use of paracetamol as a self poisoning agent has continued to increase, and that of benzodiazepine has fallen.[6,17] Changes in the usage of drugs might reflect changes in their availability — this was thought to be the primary reason for the fall in barbiturate-induced fatalities in the 1960's and 1970's.[18] The reported death rate from paracetamol poisoning has remained fairly constant in Northern Ireland, varying from two to five deaths per year from 1984 - 1989.[5] This may not record the deaths of a few very severely ill patients transferred from Northern Ireland to tertiary referral centres in England. The death rate in England and Wales from paracetamol poisoning alone rose from 105 to 210 per year between 1975 and 1991.[19] This data did not include those deaths caused by a combination which included paracetamol, because of the difficulty in attributing death in these cases to poisoning by a single agent.

Paracetamol and paracetamol-containing compound tablets in circulation in England and Wales have increased from 2.85 billion in 1973-74 to 4.8 billion in 1991-92[13] – equivalent to a 4% annual increase. Comparable figures for Northern Ireland do not exist. The use of paracetamol in overdosage has increased much more rapidly — by 86%, for example, in the period 1986 to 1991. Clearly, while increased availability of paracetamol may play a part in its use, other influences exist. The present trend may also be a consequence of a decrease in ready availability of other drugs, such as benzodiazepines.[20] Fashions in patterns of self poisoning may also be significant – perhaps influenced in part by media representation of self poisoning,[22,23] though this has been disputed.[24]

Total benzodiazepine drug prescriptions in this area fell by 9% from 1981-1991; during 1986-1991, there was a 31% fall in benzodiazepine use in self-poisoning. The increased numbers of prescriptions of benzodiazepine-containing hypnotic drugs may reflect the increase in the elderly population (to whom they are frequently prescribed) who are less likely to self poison. The number of benzodiazepine-containing sedative/tranquilliser prescriptions has fallen by

over one quarter. Although there are increasingly negative attitudes to benzodiazepines by both doctors and patients,[20,21] we do not know if the number of tablets per benzodiazepine prescription has changed. While the frequency of deliberate self-poisoning with either paracetamol or benzodiazepines does appear to increase or decrease in relation to the availability of these drugs, the rate of change of self-poisoning appears to be much greater, suggesting that other factors are involved.

The outcome of these patients has also changed. There is an increasing tendency to discharge patients without follow-up , perhaps mirroring the changes in diagnoses by junior staff – away from classifying cases as neurotic illness and towards identifying situational crises. Because of the circumspection required in interpreting diagnoses, recorded principally by junior staff, we have not analysed this data in detail.

The principal reservation of this study is its design – by retrospective case note review. Nevertheless all notes were available, and it is unlikely that the quality of some of the information (for example, the drug used as the poisoning agent) would be greatly improved by a prospective study. The central findings of this study remain valid, that the decline in self poisoning seen in the early 1980's has not continued, and that there has been an increase in the use of a more toxic agent-paracetamol. A potentially less hazardous paracetamol-based analgesic, containing methionine, is commercially available. Continued monitoring of the epidemiology of deliberate self-poisoning and suicide, and action to reduce the availability of the more lethal compounds, may help to achieve the government's stated aim of a reduction in the suicide rate by at least 15% by the year 2000.[25]

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REFERENCES

1. Morgan H G, Burns-Cox C, Pocock H, Pottle S. Deliberate self-harm: clinical and socio-economic characteristics of 368 patients. *Br J Psychiat* 1975; **127**: 564-74.
2. Gelder M, Cath G, and Mayou R. Oxford Textbook of Psychiatry, 2nd Edition, 1989; p 493.
3. Kreitman N. (ed) Parasuicide, London: Wiley 1977.
4. Hawton K, Fagg J. Suicide and other causes of death following attempted suicide. *Br J Psychiat* 1988; **152**: 359-66.
5. Darragh P. Epidemiology of suicide in Northern Ireland 1984-1989. *Irish J Med Sci* 1991; **160**: 354-7
6. Platt S, Hawton K, Kreitman N, Fagg J, Foster J. Recent clinical and epidemiological trends in parasuicide in Edinburgh and Oxford: a tale of two cities. *Psychol Med* 1988; **18**: 405-18.
7. Platt S. A subculture of parasuicide ? *Human Relations* 1985; **38**: 257-97.
8. Jones D I R. Self poisoning with drugs: the past 20 years in Sheffield. *Br Med J* 1977; i: 28-9.
9. Brewer C, Farmer R. Self poisoning in 1984: a prediction that didn't come true. *Br Med J* 1985; **290**: 391.

10. McMurray J J, Northridge D B, Abernethy V A, Lawson A A H. Trends in analgesic self-poisoning in West Fife 1971-1985. *Quart J Med* 1987; **65**: (246) 835-43.

11. Kelly C, Galloway R. Deliberate self poisoning presenting at Craigavon Area Hospital: 1976 and 1986. *Ulster Med J* 1992; **61**: 12-18.

12. Wells N. Suicide and deliberate self-harm 1981; London: Office of Health Economics.

13. Brandon G. (Personal Communication), Paracetamol Information Centre, Suite 201a Butler's Wharf, 45 Curlew St, London SE1 2ND.

14. Hawton K, Fagg J. Trends in deliberate self poisoning and self injury in Oxford 1976-90. *Br Med J* 1992; **304**: 1409-11.

15. Fuller G N, Rea A J, Payne J F, Lant A F. Parasuicide in central London 1984-1988. *J Roy Soc Med* 1989; **82**: 653-56.

16. Kennedy P, Kreitman N. An epidemiological survey of parasuicide ("attempted suicide") in general practice. *Br J Psych* 1973; **123**: 23-34.

17. Smith A F, Botton D A, Irving J B. Review of drug overdosage in West Lothian. *Health Bull 1983*; **41**: 84-93.

18. Johns M W. Self poisoning with barbiturates in England and Wales during 1959-1974. *Br Med J* 1977; **1**: 1128-30.

19. Deaths from poisoning by solid or liquid substances: accidental, suicidal or undetermined whether accidentally or purposely inflicted. England and Wales. Table 10, 1973-1991. Office of Population Censuses and Surveys, St Catherine's House, 10 Kingsway London WC2B 6JP.

20. Chaplin S. Benzodiazepine prescribing. *Lancet 1988*; **i**: 120-1.

21. Petursson H, Lader M H. Withdrawal from long-term benzodiazepine treatment. *Br Med J* 1981; **283**: 643-45.

22. Bray G P. Liver failure induced by paracetamol. *Br Med J* 1993; **306**: 457-8.

23. Collins S. Health prevention messages may have paradoxical effect. *Br Med J* 1993; **306**: 926.

24. Platt S. The aftermath of Angie's overdose: is soap (opera) damaging to your health? *Br Med J* 1987; **294**: 954-7.

25. Department of Health. The Health of the Nation: Mental Illness. 1993; London Her Majesty's Stationary Office.

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Geriatric medicine: the anatomy of change

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SUMMARY

We have studied workloads and patterns of care in geriatric medicine from 1982 to 1993 in the Ulster Hospital. There was a 137% rise in admissions, a 16% reduction in domiciliary visits and a 31% increase in ward assessments. The continuing care waiting list fell to zero in 1993. The number of new outpatients rose by a factor of 8.6 between 1987 and 1993. Between 1990 and 1993 there was an increased admission rate from nursing homes and of patients suffering from respiratory system diseases. Mortality rates fell from 27.8% in 1982 to 19.3% in 1990 and to 12.1% in 1993. Mean age and sex ratios remained unchanged over the years while the average length of stay halved from 43.3 to 22.6 days between 1990 and 1993. 81% of admissions in 1993 were emergencies. Care of the elderly in hospital and the interface with general medicine are changing.

INTRODUCTION

The specialty of Geriatric Medicine was introduced to Northern Ireland in 1948 through the work of Professor George Adams in the Belfast City Hospital, later consolidated and developed by Professor Robert Stout. From dealing primarily with continuing care and rehabilitation the specialty has changed rapidly, with a greater emphasis on acute care, investigation and rehabilitation.[1] This change has occurred at a time of movement from hospital to community based continuing care.[2] The Department of Health Care for the Elderly in the Ulster Hospital, which serves a population of 16,000 over the age of 65 years, adapted to these changes by reducing bed numbers and providing an acute continuous take-in service for the elderly (age related) from 1991, an orthogeriatric unit in 1992-3 and an acute stroke unit in 1994.[3]

The acute take-in service for the elderly at this hospital has been planned in co-operation with our general medical colleagues. Patients aged 65 years or over are accepted directly from the general practitioner and those aged 75 years or over are accepted from the accident and emergency department. At least half of general medical inpatients in Eastern Health and Social Services Board hospitals are aged 65 years or older, and more than one quarter are aged 75 years or older.[4] With two services providing medical care for the elderly, greater clarity, in respect of policies for the interface between care of the elderly and general medicine, is required.[4] We studied the changes in the pattern and delivery of care in a hospital department of Health Care for the Elderly over the past decade, to understand the present changes and to try to suggest how general and geriatric medicine might best relate to each other.

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METHODS

Data was collected retrospectively on admissions, ward assessments, domiciliary visits and day hospital and outpatient attendances in the Department of Health Care for the Elderly from 1982 to 1993. Sources used included ward admission and discharge cards and books, medical case notes, monthly and yearly statistical summaries, ward assessment and domiciliary visit books. Full data was available for the years 1990 to 1993 and these two years formed the main comparison groups in the study. Pre–1990, data for the years 1982, 1985 and 1987 was used as it was the most complete. Data considered to be incomplete in these years was excluded from the study. Admission, discharge and domiciliary visit numbers were complete for all years.

For 1990 and 1993, during preparation for a separate study, more detailed information was collected from ward discharge letters and case notes. Age, sex, postal code, source of admission, type of admission, length of stay, main diagnosis, outcome of the admission and cause of death were recorded.[5] Main diagnoses were classified into systems.[6] An admission was classified as acute if the patient was admitted via the accident and emergency department, or urgently to the ward via the general practitioner, outpatients, day hospital or a domiciliary visit. Data was collected only on patients admitted under the care of the consultants (ICT & JGMcC) looking after the geriatric catchment area of East Belfast and Castlereagh, to enable comparison over the years. Patients were followed up to discharge from the ward or death. Analysis was by STATVIEW on an Apple Macintosh computer using Chi-squared and unpaired t-tests.

RESULTS

Ward admissions to the Department of Health care for the Elderly.

The proportion of admissions from home fell in 1993 (n=732, 79.4%) compared with 1990 (n=510, 85.4%) while that from private nursing homes rose (1993, n=106, 11.5% compared with 1990, n=26, 4.3%). 747 (81%) of the 1993 admissions were emergency. (Table 1)

TABLE 1

Domicile at the time of admission in 1900 and 1993 to the Department of Health Care for the Elderly.

Year	*Total*	*Male*	*Home*	*Private nursing home***	*Residental* accommo-dation*	*Sheltered dwelling*
		%	%	%	%	%
1990	597	31.5	85.4[a]	4.3[c]	7.2	3.1
1993	922	30.4	79.4[b]	11.5[d]	5.7	3.4

* Private, voluntary and Board residential accommodation.

** including elderly mentally infirm.

ab $X^2 = 8.85$, $p = 0.0029$

cd $X^2 = 23.29$, $p < 0.0001$

There was a significant decrease in mortality rates between 1987 (n=115, 26.1%) and 1990 (n=115, 19.3%) and between 1990 and 1993 (n=112, 12.1%) and an increased rate of discharge to private nursing home care between 1990 and 1993. There was a marked fall in the proportion of patients discharged home between 1987 and 1990, but numbers rose again in 1993 (n=541, 58.7%), compared with 1990 (n=307, 51.4%) (Table 2).

TABLE 2

Discharge outcome of patients admitted in 1982-1993.

Year	*Total*	*Male*	*Died in hospital*	*Home*	*Private nursing home*	*Residential accommo-dation*	*Sheltered dwelling*	*To other ward or hospital***
		%	*%*	*%*	*%*	*%*	*%*	*%*
1982	389	38.0	27.8	64.0	0.0	8.2		
1985	346	31.8						
1987	440	30.5	26.1[a]	65.2	0.9*	7.7		
1990	597	31.5	19.3[b]	51.4[d]	13.4[f]	7.7	3.4	4.7
1993	922	30.4	12.1[c]	58.7[e]	16.5[g]	5.6	3.3	3.7

* 1987 was the first year patients were discharged to nursing homes.

** including hospice care.

ab $X^2 = 6.93$, $p = 0.0085$

bc $X^2 = 14.43$, $p < 0.0001$

de $X^2 = 2.66$, $p = 0.12$

fg $X^2 = 9.106$, $p = 0.0025$

The mean age of patients in 1990 was 81.6 years (95% CI 81.0 to 82.2 years) compared with 81.1 years (95% CI 80.7 to 81.4 years) in 1993 ($p = 0.17$, t-test). The ratio of males to females did not differ significantly over the years. Around 80% of all admissions in 1990 and 1993 were of people aged 75 years and over (Table 3).

TABLE 3

Age bands of patients admitted in 1982, 1990 and 1993.

Age band	*1982*	*1990*	*1993*
Number	389	597	922
	%	*%*	*%*
< 65	3.3	0.5	0.8
65-74	21.4	15.1	15.6
75-84	50.6	43.5	51.4
> 84	24.7	40.9	32.2

In 1990 1.8% of admissions were from other hospitals compared with 1.2% in 1993. In 1993, 351 (38.1%) of admissions were casualty, 30.8% from general practitioners, 13.2% from orthopaedics, 10.2% from domiciliary visits and 7.7% other. There was a marked increase in admissions from the Ards Hospital geriatric catchment area in 1993, 205 (22.2%) compared with 69 (11.6%) in 1990, through increased emergency and orthogeriatric admissions (Table 4).

TABLE 4

Admissions by geriatric catchment areas in 1990 and 1993.

Geriatric catchment areas	*1990*	*1993*
Number	597	922
Ulster Hospital	498	626
Ards Hospital	69	205
Belfast City Hospital	4	47
Royal Victoria Hospital	1	0
Other	25	44

Between 1990 and 1993 there was a greater than expected rise in admissions due to locomotor system disease, 16.4% (n=98) of 1990 admissions, compared with 22.9% (n=211) of 1993 admissions ($X^2 = 9.36$, $p = 0.0022$) and respiratory system disease, 11.7% (n=70) of 1990 admission, compared with 20.3% (n=187) of 1993 admissions ($X^2 = 18.875$, $p < 0.0001$). In the other systems observed figures were close to expected.

Between 1990 and 1993 there was a 32% decrease in domiciliary visits by the consultant geriatricians. Ordinary ward assessments (in general medical, orthopaedic and surgical wards) fell from 84 to 17 but this was more than offset by 87 'Assessment and Care Management' assessments in these wards. The number of new outpatients quadrupled from 1990 to 1993. The figure for the first four months of 1994 is 136. The number of new day hospital patients (30 place day hospital) remained steady at 450 to 550 per year (Table 5).

TABLE 5

Assessment prior to hospital admission: domiciliary visits, ward assessments, continuing care waiting list and new outpatients, 1982 to 1993.

Year	*Domiciliary visits**	*Assessments in other wards*	*Continuing care waiting list*	*New outpatients*
	%			
1982			178	
1984			147	
1985	100.00	79		
1987	112.7	117	66	21
1990	122.6	84	15	47
1993	83.8	104	0	182

* 1985 – baseline level taken as 100%

Respite care was moved from the Ulster Hospital to Forster Green Hospital in 1991, where admissions rose from 172 in 1990 to 343 in 1993, largely due to this move. The number of beds available to the Ulster Hospital geriatric catchment area fell from 196 in 1990 to 124 in 1993, which included 52 beds in Forster Green Hospital for respite, continuing care and slow stream rehabilitation. Fifty of the 124 beds were designated as continuing care in 1992, and this figure is now only 31, 25 of which are occupied. Nineteen of 72 beds in the Department of Health Care for the Elderly are presently occupied by assessment and care management patients awaiting placement. Lengths of stay decreased between 1990 and 1993 (Table 6).

TABLE 6

Bed usage in the Department of Health Care for the Elderly in the Ulster Hospital. Lengths of stay in 1990 and 1993.

Length of stay	*1990*	*1993*
Number	597	922
	%	*%*
0-14 days	36.7	51.7
15-31days	30.5	27.6
32 days or more	32.8	*20.7*

DISCUSSION

The fall in the continuing care waiting list for the Department of Health Care for the Elderly in the Ulster Hospital was due to the increased availability of private nursing home beds from 1987 onwards. The number of nursing home beds in Northern Ireland rose from 347 in 1982 to 1564 in 1987,[7] but the number of continuing care patients in our wards did not fall markedly until a more active policy of discharge to private nursing homes was instituted in 1990. That year also marked the start of increasing numbers of admissions of patients from private nursing homes to the Department of Health Care for the Elderly.

At the end of 1991 a more active policy of acute admissions from the accident and emergency department and from general practitioners was pursued. (Patients with a suspected myocardial infarction or acute gastrointestinal bleeding were admitted directly to the cardiology and general medical units as the Department of Health Care for the Elderly is separate from the main hospital site). This amounted to a continuous acute take-in system of patients over the age of 65 years from general practitioners and over 75 years from the accident and emergency department, resulting in a 54% increase in admissions between 1990 and 1993.

With patients no longer remaining in hospital for continuing care until death and more patients being admitted for rehabilitation and acute treatment, death rates and length of stay fell from 1990 onwards. 51.7% of patients stayed less than 14 days in 1993 and 27.6% stayed between 15 and 31 days. In a study in Canterbury equivalent figures of 70% and 19.7% were reported.[8] While there

was a significant fall in death rate between 1990 and 1993 the difference between the number of deaths plus nursing home discharges was not significant (195 versus 264 respectively, $X^2 = 2.719$, $p = 0.0948$). Further inroads need to be made into reducing lengths of stay through weekend discharges, more rapid placement of patients who have been 'care managed' and increased use of innovative schemes such as the 'Home from Hospital' scheme.

The rise in locomotor system admissions in 1993 was largely due to the opening of orthogeriatric beds. Of 211 such admissions, 119 came from the orthopaedic wards, of whom 8.4% had been admitted from nursing homes: 57% were subsequently discharged home, 20.1% were discharged to nursing homes and 8.4% died. The rise in respiratory system disorders between 1990 and 1993 is a reflection of the increasing numbers of acutely ill elderly people admitted. Pneumonia in the over 75's is 15-20 times more frequent than in younger adults[9] and usually occurs in the setting of chronic chest disease.[10]

The increased in-patient admissions was mirrored by a modest but significant rise in new out-patient numbers through general practitioners becoming more aware of short waiting times (1-2 weeks) and that a consultant geriatrician saw all patients and determined the need for review.[11] In the first third of 1994 new out-patient figures reached 75% of the total for 1993. Transport, space and staffing limitations will affect further increases.

While the number of continuing care beds dropped from 50 to 31, these were 'replaced' by 19 patients currently awaiting placement in nursing homes. In effect these are 'continuing care' patients in transit and underline the need to keep sufficient continuing care beds in hospital.

Although day hospital figures remained steady throughout the years and we believe that it performs a useful service, facilitating earlier ward discharge and maintaining elderly people at home, the function, effectiveness and costeffectiveness of day hospitals has been called into question by researchers from Cardiff.[12]

The fall in ward assessments in the general medical, surgical and orthopaedic wards, carried out in the past for continuing care and rehabilitation transfers to the Department of Health Care for the Elderly, has been more than matched by assessments for nursing home and residential placement. There is concern, in view of bed losses, nursing and medical staffing limitations and the increasing demands of the changing service, that fewer patients are being considered for transfer for rehabilitation and trials of rehabilitation, especially from the medical wards. This is an area which needs further investigation with our general medical colleagues since the need for rehabilitation for the elderly should increase. [13, 8]

The fall in domiciliary visits by the consultant geriatricians was achieved by general practitioners being able to admit patients directly and urgently from home by telephoning, by providing an active out-patient service with urgent referrals being seen within 24 hours and by providing immediate access to the day hospital. Domiciliary visits remain important in management of the elderly as it is only in the home situation that one can gain full insight into the social circumstances influencing illness and disability and the patient's ability to cope.

Geriatrician's input into 'orthogeriatric' care is now standard practice, with many patients in our unit requiring extended rehabilitation and care management. Such care can be provided in an orthogeriatric unit [14] or through an orthogeriatric liaison service. [15] While acute care for the elderly on an age related basis has proved a success in terms of numbers, consideration must be given to those elderly patients admitted to medical wards whose rehabilitation needs would be best met in a geriatric medical ward [4,8] and the early recognition and referral of those patients requiring assessment and care management.

We believe that a 'mixed economy' of patients is better than selecting out specific elderly patients for admission to geriatric medical units, although there is evidence in Belfast that the casualty officer and general practitioner pre-select prior to referral.[6] The geriatrician should have access to beds on the main acute hospital site[1] as this would greatly help integrated care of the elderly and early assessment for rehabilitation or care management. Geriatricians must be closely involved with general physicians in caring for the elderly.[6]

In the midst of these changes it is a matter of concern that 'geriatric medicine' does not become 'general medicine for the elderly' but that due care and attention are paid to those needing more complex social and functional rehabilitation or continuing care in hospital or the community. Our clinical experience is that nursing homes have difficulty coping with patients with severe pressure sores or swallowing difficulties. The ability of nursing homes to cope with those in a high dependency category has been questioned.[16] Services and training of staff in nursing homes will have to be improved to contend with increasing numbers of such patients. Private nursing home care may then turn out to be as expensive as care in National Health Service nursing homes or continuing care hospital units.[17]

Geriatric medical units must retain, improve and impart expertise which has not yet been developed in nursing homes. Such units should provide a continuing care hospital service for patients with complex medical and nursing needs and should train medical students, doctors and nurses in their care. In the new NHS 'Trust' and 'Fundholding' efficiency savings climate elderly people should not be denied proper access to medical care.[18]

REFERENCES

1. Care of the Elderly: present provision and future developments. Geriatric sub-committee, Eastern Health and Social Services Board, January 1992.
2. Bernard J. Whither community care? *Care of the Elderly* 1993; **1**: 23-25.
3. Taylor I C, McConnell J G. Philosophy and operational policy of the Department of Health Care for the Elderly, Ulster North Down and Ards Hospitals Trust, 1993.
4. Report of the working group of the medical sub-committee of the area medical advisory committee on acute general medical admissions. Eastern Health and Social Services Board, 21 March 1994.
5. Taylor I C, McConnell J G. Admission of Nursing Home patients to Geriatric Medical Wards. Ulster Med J, 1994; **63**: In press.
6. Differences between "geriatric" and "medical" patients aged 75 and over. Todd M, Crawford V, Stout R W. *Ulster Med J* 1993; **62**: 4-10.

7. Consultative paper on the registration and inspection of private and voluntary nursing homes. Department of Health and Social Services. November 1988.
8. Stevens R S, Potter J M, Hildick Smith M. Effect on a geriatric service of opening a 25-bed ward on the district general hospital site: an audit. *J Roy Coll Physns Lond* 1990; **24**: 107- 111.
9. Woodhead M. Pneumonia. *Care of the Elderly* 1994; **6**: 62-70.
10. Marrie T J, Haldane E V, Faulkner R S, Durant S, Kuram C. Community acquired pneumonia requiring hospitalisation. Is it different in the elderly? *J Am Geriat. Soc* 1985; **33**: 671-80.
11. Donald I P, Berman P. Geriatric outpatient clinics: an audit of clinical action, transport and general practitioners' views. *Age Ageing* 1989; **18**: 253-7.
12. Vetter N J, Smith A. Geriatric day hospitals. *Age Ageing* 1989; **18**: 361-3.
13. Tallis R. Rehabilitation of the elderly in the 21st century. *J Roy Coll Physns Lond* 1992; **26**: 413-22.
14. Murphy P J, Rai G S, Lowy M, Bielawska C. The beneficial effects of joint orthopaedic-geriatric rehabilitation. *Age Ageing* 1987; **16**: 273-8.
15. Harrington M G, Brennan M, Hodkinson H M. The first year of a geriatric–orthopaedic liaison service: an alternative to 'orthogeriatric ' units? *Age Ageing* 1988; **17**: 129-33.
16. Campbell H, Crawford V, Stout R W. The impact of private residential and nursing care on statutory residential and hospital care of elderly people in South Belfast. *Age Ageing* 1990; **19**: 318-24.
17. Donaldson C, Bond J. Cost of continuing care facilities in the evaluation of experimental National Health Service nursing homes. *Age Ageing* 1991; **20**: 160-8.
18. Grimley Evans J. Dangers of ageism. *Care of the Elderly* 1993; **5**: 217.

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Admission of nursing home patients to geriatric medical wards

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SUMMARY

Comparison was made between patients admitted from a nursing home and all other patients admitted to a geriatric medical unit in 1990 and 1993. The number of nursing home patient admissions rose from 26 in 1990 to 106 in 1993. Nursing home patients were frailer both physically and mentally with a dementia rate of 78% (in those who survived, 1993) and a mortality rate of 19.8% (1993), compared with a dementia rate of 19% and a mortality rate of 11.3% in all other admissions in 1993. Male patients admitted from a nursing home were more likely to die than females (33% versus 14.5%, 1993). Lengths of stay of nursing home patients were shorter, largely due to the availability of a 'safe environment' when discharged, but also related to shorter survival times. 61% of patient admissions from nursing homes in 1993 were considered 'unnecessary' and could have been avoided if specialist advice had been available before admission.

INTRODUCTION

Implementation of the Community Care Act 1990 on the first of April 1993, to assess need and maintain choice[1] occurred in a time of decreasing hospital based and increasing community based continuing care provision for elderly people. One of the main aims of the Act is to assess the need for admission to various forms of institutional care, including hospital and private and voluntary nursing homes.[2]

The number of hospital based continuing care beds for elderly patients has been decreasing since 1990 while nursing home beds in the Eastern Health and Social Services Board area have increased from 2182 in 1990 to 3671 in 1993.[3]

Has this move away from hospital based continuing care led to increasing numbers of nursing home patient admissions to geriatric medical units? If so, what are the characteristics of such patients and are such admissions necessary? The present study addresses these questions and suggests possible changes in nursing home care for elderly people.

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METHODS

The Department of Health Care for the Elderly in the Ulster Hospital is on continuous take-in for elderly patients. All patients admitted from nursing homes to the Department of Health Care for the Elderly in the Ulster Hospital in 1990 and 1993 who were under the care of the authors were studied. Patients who had been admitted to other wards or hospitals and transferred to the Department of Health Care for the Elderly were included. Data was also collected on all other admissions to the Department during 1990 and 1993 for comparison with the nursing home groups. The admission policy to the Department and its policy for transfer from other hospitals did not change during the study period.

Information was collected retrospectively from ward discharge letters with further recourse to the full case notes as required (nursing home patients especially), and entered directly on to a computer database. Age, sex, postal code, source of admission (accident and emergency, direct to the ward via the general practitioner), type of admission (emergency, booked), length of stay, abbreviated mental test score[4] of survivors (1993 only), reason for referral, main diagnosis, presence of dehydration, treatment given, outcome or cause of death were recorded. Main diagnoses were divided into systems (respiratory, locomotor, vascular, cardiovascular).[5] If no underlying cause was found the diagnoses of falls and poor mobility were recorded under the locomotor system. If there was no precipitating cause for confusion (such as a urinary tract infection) it was classified under 'psychiatric'.

An admission was classified as acute if the patient was admitted via the accident and emergency department or directly to the ward via the general practitioner (within hours), or directly from outpatients or the day hospital urgently, or within 24 hours of a domiciliary visit by one of the consultant geriatricians.

An estimate was made of the appropriateness of admission by considering admissions which might have been avoided if specialist advice had been available in the nursing home prior to admission. Patients who required intravenous fluids, blood transfusions or intravenous medication or who presented with an acute collapse, acute onset of symptoms, dehydration, abdominal pain (with suspicion of obstruction or a mass or pyrexia), those with need of further tests (other than routine blood tests, chest X-ray or simple bone and joint X-ray) or urgent referral to other specialties were classified as requiring admission. If, on reading the notes none of these conditions was met but the assessor (ICT) felt that any patient still warranted admission, they were also classified as requiring admission. Conversely, if the above conditions were met but the assessor felt that the patient's needs would have been better met in the nursing home (some patients with a stroke, those requiring terminal care or who were moribund on admission, patients whose families did not wish them to be moved) they were classified as not requiring admission. If the patient's family insisted that the elderly person should be treated in hospital they were classified as needing admission.

The abbreviated mental test score was not routinely recorded in the 1990 data or for patients who died, and was only entered for 1993 patients who survived. A score of less than 7 out of 10 was classified as dementia.[4] Note of functional

ability was entered to determine if nursing home patients were appropriately placed.

Data were analyzed using STATVIEW on an Apple Macintosh computer using Chi-squared and unpaired t-tests.

RESULTS

There were 26 nursing home admissions included in 597 ward admissions in 1990 compared with 106 out of 922 ward admissions in 1993 (X^2 = 23.29, $p < 0.0001$) . There were 6 males in 1990 and 30 in 1993 (NS). The average length of stay in 1990 was 32.7 days (95% confidence interval (CI) 10.3 to 55.2 days) compared with 15.2 days in 1993 (95% CI 11.7 to 18.7 days, $p = 0.007$, t-test). Six 1990 patients died compared with 20 in 1993 (NS). The mean age of 1990 patients was 84.6 years (95% CI 81.9 to 87.2 years) compared with 83.8 years (95% CI 82.5 to 85.1 years) in 1993 (NS). Because of the small number of patients in 1990 comparison of admission types or diagnoses between the two groups was not made. All nursing home patients were dependent for activities of daily living; there was no evidence of inappropriate placement.

1993 nursing home patients, admission data.

96 of 106 admissions were classified as emergency. Of these, 42 patients were admitted directly to the ward by the general practitioner, 41 were admitted directly from the accident and emergency department, 12 were admitted from domiciliary visits and one was admitted from the day hospital. Seven of the 10 non-emergency admissions were from orthopaedic wards.

Non-diagnostic reasons for referral from the general practitioners' letters were as follows :- general deterioration (n=16, 15%), confusion (n=12, 11%), falls (n=10, 9.4%), acute collapse (n=3) and immobility (n=2). Twenty-one patients needed intravenous fluids, 19 were dehydrated, 15 required intravenous antibiotics and three needed intravenous diuretics. Details of the main diagnosis on admission are given in the table.

TABLE

Classification of the main diagnosis on admission for 106 patients admitted in 1993 from a nursing home

System	*Number*	*Details*
Respiratory	20	Chest infection (15), chronic obstructive airways disease (3), lung carcinoma (2)
Locomotor	17	Fractured neck of femur (7), falls (5), osteoporosis (2), poor mobility (2), osteoarthrosis (1)
Gastrointestinal	9	Cholecystitis(2),pseudo-obstruction (2), colonic carcinoma (1), dysphagia (1), abdominal pain (2), diarrhoea (1)

System	Number	Details
Cardiovascular	9	Congestive heart failure (6), left ventricular failure (2), postural hypotension (1)
Genitourinary	9	Urinary tract infection (5), drug induced acute renal failure (3), prostatic carcinoma (1)
Vascular	4	Gangrene (2), pressure sores (2)
Psychiatric	2	Dementia (2)
Skin	1	Herpes zoster (1)
Metabolic	1	Hyperosmolar non-ketotic coma (1)
Other	34	Anaemia (8), dehydration (5), overmedicated (4), septicaemia (3), lymphoma (1), postural leg oedema (1) multisystem disorder (12)

1993 nursing home patients, assessment of need for admission to hospital.

65 of the 106 patients were assessed as not requiring admission to hospital because the problem could probably have been dealt with in the nursing home following specialist advice. Fourteen of these 65 died compared with seven of the 41 classified as requiring admission (NS). Of the 14 who died, three required terminal care, two were moribund, three had a severe stroke, three were extremely frail and three had severe dementia. Thirty of the 65 not requiring admission were admitted directly from the general practitioner, 33 from the accident and emergency department and two following a domiciliary visit.

There was no significant difference in the age or sex of those assessed as requiring admission compared with those not requiring a admission. In general those not requiring admission were less ill but diagnostic categories were similar between the two groups. These 106 patients used 1613 (7.8%) out of a total of 20,649 occupied hospital bed-days. The 41 assessed as requiring admission used 652 bed-days.

1993 nursing home patients, compared with all other patients admitted in 1993.

Of the nursing home patients, ten of the 30 men died compared with 11 of the 76 women ($X^2 = 6.10$, $p = 0.013$). Overall 19.8% of the nursing home patients died compared with 11.3% of all other patients ($X^2 = 5.86$, $p = 0.013$). There was no significant difference in the mean age of the nursing home patients who survived and those who died.

There were 922 admissions to the Department of Health Care for the Elderly in 1993. 78% of the 85 survivors of those admitted from a nursing home had dementia (abbreviated mental test score of < 7/10), compared with 19 % of the survivors of all other patients ($X^2 = 143.6$, $p < 0.001$). The mean age of the nursing home patients was 83.8 years (95% CI 82.5 to 85.1 years) compared

with 80.7 years (95% CI 79.3 to 81.2 years) for all other patients ($p < 0.0001$, t-test). The mean length of stay of nursing home patients was 15.2 days (95% CI 11.7 to 18.8 days) compared with 23.5 days (95% CI 21.7 to 25.4 days) for all other patients ($p = 0.002$, t-test). The mean length of stay (survival) of nursing home patients who died was 14.7 days compared with 19.9 days for all other patients (NS). The equivalent figures for survivors were 16.2 days (nursing home) and 23.9 days (all others) ($P = 0.005$, t-test). There was no significant difference in the ratio of males to females between the two groups.

Of the 106 nursing home patients 32 were admitted in the first quartile of 1993, 29 in the second, and 22 and 23 in the third and fourth quartiles. Community care assessments were introduced on the first of April 1993.

DISCUSSION

This study shows that between 1990 and 1993 there was a 400% increase in the number of nursing home patients admitted to the Department of Health Care for the Elderly, while the number of nursing home places in the Eastern Health and Social Services Board rose by 68% and the total number of admissions to the Department by 74%. The rise in total admissions is a reflection of the change in Hospital Care of the Elderly from continuing care and rehabilitation to acute care and rehabilitation.

The marked rise in nursing home patient admissions may also reflect an increase in the number and frailty of elderly patients admitted to nursing homes, although the 1993 quartile admissions did not increase following the introduction of the more rigorous assessment for prospective nursing home patients on April 1st 1993. Mortality rates of nursing home patients did not differ significantly between 1990 and 1993, although care must be taken in interpreting this because of the small number of 1990 admissions. There is clear evidence from this study that male patients admitted from a nursing home are more likely to die than female, but all nursing home patients are more likely to die than patients admitted from home or residential care.

Nursing home patients admitted to geriatric medical units are an 'at risk' group, as shown by their higher death rates, poorer functional ability and increased rate of dementia compared with all other patients. The shorter length of stay of nursing home patients compared with other patients is related to the availability of a safe environment when discharged, although the shorter survival times for nursing home patients who die, compared with other patients who die (average 5 days less), also contributes.

It is a difficult decision for the casualty officer to send a frail elderly person, whom he has never seen before, home. There are and will continue to be inappropriate admissions to general and geriatric medical wards. While one cannot judge precisely the number of 'unnecessary' admissions the present study suggests that a significant saving in bed-days, perhaps of up to 1,000 in each average geriatric medical unit catchment area, might be possible if specialist advice and help were more readily available to general practitioners. This would come most appropriately from a hospital or community based geriatrician with access to acute beds. It would have to be a 24 hour, seven days a week service, although there is evidence from this study and others [5] that there is a slower onset of illness in patients admitted to geriatric medical units rather

than general medical units. There may also be a delay in referrals, since 18% of nursing home (1993) patients were dehydrated on admission. Two patients were admitted unnecessarily following a domiciliary visit by a consultant geriatrician, emphasising the need for a critical appraisal of admissions of patients from nursing homes by all grades of medical staff.

Nursing homes provide a protected environment and recommended nursing levels [6] are not dissimilar to those in geriatric medical units. They lack the medical cover and equipment available in hospital (such as specialized beds). If simple equipment improvements and treatment, in the form of intravenous fluids and antibiotics (first dose given by the general practitioner), were available in nursing homes it is likely that hospital bed-days could be saved while maintaining a better quality of life for the patient. Input from professions allied to medicine may also be required. The concept of a 'hospital wing' of one to two beds is not a new one; it already exists in the voluntary sector.

The type of treatment and the decision to treat should not be dependent on a patient's age. However there are patients who have been admitted inappropriately from nursing home to hospital care who could have been more appropriately and comfortably cared for in the nursing home, provided specialist advice or assessment had been sought. We would recommend a pilot scheme, based on one nursing home, to assess the feasibility of the geriatrician and general practitioner working in closer harmony to try to maintain elderly people in nursing homes rather than admitting them to hospital.

'Effectiveness of treatment must be combined with the least disruptive solution for the elderly person in social, psychological, medical and functional terms.'[7]

REFERENCES

1. Bernard J. Whither community care? *Care of the Elderly* 1993; **1**: 23-5.
2. Assessment and Care Management. Implementation working group. Eastern Health and Social Services Board Social Services Directorate, January 1993.
3. Strategy for future institutional care for elderly people. Eastern Health and Social Services Board Planning Department, 1993.
4. Qureshi K N, Hodkinson H M. Evaluation of a ten-question mental test in the institutionalised elderly. *Age Ageing* 1974; **3**: 152-7.
5. Todd M, Crawford V, Stout R W. Differences between "geriatric" and "medical" patients aged 75 and over. *Ulster Med J* 1993; **62**: 4-10.
6. Guide to good practice for residential and nursing homes. Eastern Health and Social Services Board Social Services Directorate, 1993.
7. Horrocks P. The components of a comprehensive district health service for elderly people – a personal view. *Age Ageing* 1986; **15**: 321-42.

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The role of the general practitioner hospital in inpatient care

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SUMMARY

The rationale of the general practitioner hospital continues to be questioned. A study of the services and case-mix of two of the four remaining general practitioner hospitals in Northern Ireland was undertaken to determine whether the nature and cost of inpatient care in these hospitals was comparable to the available alternatives. The case-notes of all non-maternity admissions (n=509) were reviewed. The two hospitals provide acute medical care for a wide range of patients. The majority of patients appeared to require hospitalisation. It is likely that the beds at the two hospitals were mainly a substitute for district general hospital care. The general practitioner hospitals were estimated to be less costly than alternative forms of care, although it was doubtful whether they fulfilled all the structural criteria of quality generally regarded as important for hospitals of this type.

INTRODUCTION

There continues to be debate concerning the role of the general practitioner hospital in modern health care. On the one hand, these hospitals are viewed as inessential anachronisms. Concerns have been expressed about their relative isolation, structures (equipment, or existence of admission and discharge policies) and outcome (quality and efficiency of care), and many have been threatened with closure. Criticisms of unnecessary admissions and uneconomical use of beds have also been made.[1] Proponents, on the other hand, stress their strengths, such as continuity of care in accessible, informal surroundings, avoiding admission to the more expensive district general hospital, shorter waiting times[2] and a unique type of intermediate care linking primary and secondary care[3].

These conflicting views, in part, can be explained by the exceptional variety of roles which general practitioner hospitals seem to play throughout the UK. They are particularly well suited to the care of the elderly,[4] respite care or holiday

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relief as well as for patients requiring short periods of rehabilitation or terminal care.[5] Such diversity has hindered attempts to produce or evaluate data, for example on the standard and outcome of care. Most available studies have been carried out by highly motivated general practitioners who, in analysing and documenting their own experiences, have tended to present a largely favourable view of their hospitals often with little objective support. Nonetheless, these studies are important since they show that care in these hospitals can be beneficial, and sometimes invaluable for particular types of patients. More evidence is needed about these hospitals to determine whether the balance of favour swings toward the sceptics or the enthusiasts.

There have been very few comparative studies on the standards or appropriateness of care, outcomes and costs for similar patients treated in a general practitioner hospital and a district general hospital. A commonly cited but dated, cost-effectiveness study from the 1970s by Rickard[6] demonstrated that community hospitals with fewer than 35 beds had higher costs than the district general hospital. Other studies from the early 1970s have compared the effectiveness of the two types of hospitals,[1, 7] but more up-to-date studies are needed.

This study was commissioned by the Northern Health and Social Services Board in order to clarify the role of its two remaining general practitioner hospitals pending the rationalisation of hospital services. The two hospitals are situated in predominantly rural areas, although one (The Robinson Memorial Hospital) is within a short distance of two district general hospitals. The other (Dalriada Hospital) is more peripheral and located in the most isolated part of the Board area, serving a smaller and more sparsely distributed catchment population considerably more remote from the nearest district general hospital. Table 1 provides summary information on the hospitals and their respective patient populations.

The principal objectives of the study were to describe retrospectively the non-maternity case-load of the two hospitals over a 12 month period, to describe resource use at the two hospitals from routine hospital activity data and to obtain some idea of the costs of treatment at these hospitals in comparison to alternative forms of care.

METHODS

The retrospective survey was based on data extracted by a medically trained member of the research team (GMcE) from the case-notes of all non-maternity inpatients admitted at both hospitals (n=509) during the period 1 October 1990 to 30 September 1991. A form was devised for each inpatient episode and was supplemented by a brief form completed by the general practitioner responsible for the admission. The general practitioner form provided a partial validation of the case-note data, as well as providing additional information on the possible alternatives to admission if the bed had not been available (assuming that only currently available local resources could be used) and the reasons for using the hospital bed rather than an alternative form of care.

A crude costing exercise was undertaken to compare costs to the NHS of general practitioner hospital care in comparison to alternative care (mainly district general hospital but including domiciliary care). The alternatives to

general practitioner hospital care suitable for each patient were specified by the admitting general practitioners. Patients who would have been admitted to hospitals farther afield (44/509, 9%), or for whom there were insufficient data (17/509, 3%) were costed as if they had been admitted to one of the two local district general hospitals. Specialty cost data for general medicine and geriatrics (the most appropriate specialties for comparison) were used to determine the average costs of patients at the two district general hospitals and for one of the general practitioner hospitals. The medical staff, pharmacy and diagnostic components of the specialty costs were allocated according to whether or not the patient made use of that part of the service while in the general practitioner hospital. For example, pharmacy costs were attached only to those patients who were on drug treatments. Nursing staff and general services costs were apportioned equally across specialties. All inpatients were assigned to a specialty on the basis of their primary diagnostic grouping. Overall running costs were used for the general practitioner hospital for which no specialty costs were available (Dalriada Hospital).

TABLE 1

The two general practitioner hospitals: 1990/91 data.

Characteristic	*The Robinson Memorial Hospital*	*Dalriada Hospital*
Catchment population	24,000 (15 mile radius)	18,000 (10 mile radius)
Distance to district general hospital(s)	9 miles and 1 mile	19 miles
No. of non-maternity beds	24	22
Inpatients per year sex distribution mean age	320 63% female 70 years	200-250 53% female 69 years
Percentage occupancy	80%	85%
Average length of stay	23 days	18 days
Consultant outpatient clinics	None – all at local hospital 1 mile away	11 per month

It was necessary to assume that patients admitted to the general practitioner hospital were as severely ill, on average, as patients in the relevant specialties in the district general hospitals; that outcome and length of stay (or the care episode) in the district general hospital would be the same as in the general practitioner hospital across all care alternatives; and that length of stay was the principal factor affecting total costs. As so few patients were deemed suitable by the general practitioner for either nursing home or domiciliary care, rather crude cost approximations were used. Nursing home provision for 31 patients was estimated using the average cost per residential week for an old people's home in the vicinity (£30.00 per day). Due to the unavailability of suitable

information, the cost of home-based care for 27 patients was strictly nominal at £10.00 per day based on two hours of home-help. Although other forms of home care were available, a very modest level of domiciliary support was assumed since the general practitioners would only have been prepared to see the most able patients managed at home. Furthermore, 78% of this group were living with relatives and would have had direct family support (a non-NHS cost).

RESULTS

During the study period, 82% (417/509) of the total admissions at both general practitioner hospitals could be identified from the case-notes; 220/268 from Dalriada Hospital and 289/351 from the Robinson Memorial Hospital. Most cases (86%) had only one admission during the study period. The mean age of both hospital populations was 69.5 years and only 21% were aged under 60. For both hospitals, the mean length of stay was 20.5 days with a range up to 396 days. Eight per cent (39/509) stayed over two months and 57% for less than two weeks. The distribution of age and length of stay for each hospital is shown in the figure.

The primary reasons for admission are shown in Table 2. Almost two-thirds of the patients at each hospital were admitted primarily for the management and/or diagnosis of a medical and/or surgical problem, convalescence being the second most common reason. The primary reasons for admission of 39 patients who stayed over two months were analysed separately; 23 of these were admitted for the management and/or diagnosis of a medical problem and ten for convalescence or social/respite care. Only two of the 22 patients admitted for terminal care were in this long-stay group. Sixty-seven patients (13%) died in hospital.

A wide variety of primary diagnoses was recorded at each hospital. The most commonly recorded diagnostic group was disease of the circulatory system (79/509, 15%), approximately half of which involved heart disease. Other diagnostic categories included supplementary classification (59/509, 12%), ill defined symptoms and signs (56/509), and respiratory disease (54/509, 11%). On average, 18% of admissions at both hospitals were seen by a visiting consultant and for all but seven there was some evidence in the notes of a management plan and/or objectives for admission.

An average of 69% (351/509) of patients admitted at both hospitals required investigations, particularly blood chemistry/haematology (88%), bacteriological tests (39%) and x-rays (37%). For almost two-thirds (62%) of patients at both hospitals, the main objective was the introduction of a new therapy or drug treatment. A few were admitted primarily for education, but as this is not generally regarded as a specific treatment it was not often recorded in the notes. The remainder were admitted either for the adjustment or stabilisation of their existing treatment regimen (93/509, 18%), or for nursing care alone (83/509, 16%). Although one of the hospitals had an operating theatre, no surgery was performed.

According to the general practitioners' own assessments, 77% (391/509) of all patients would otherwise have been admitted to one of the local district general hospitals. They regarded only 5% (27/509) as suitable for management at home with appropriate family support and a similar proportion would have been

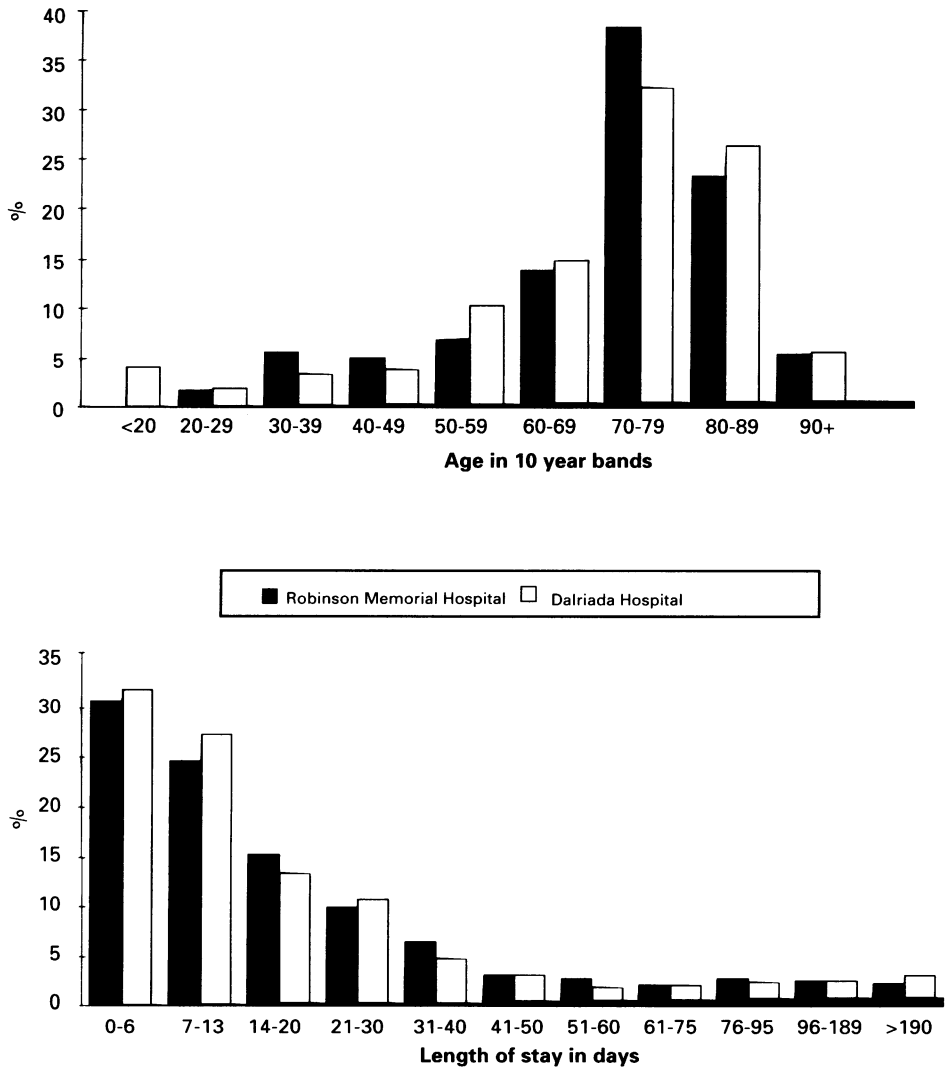

Figure. Age distribution and length of stay for patients admitted to the two general practitioner hospitals

admitted to a residential or nursing home. Continuity of care (254/509, 50%) and patient convenience and/or accessibility (196/509, 39%) were the two most common reasons for admission to the general practitioner hospital rather than one of the two local acute hospitals. Other reasons included the unavailability of beds elsewhere (17/509, 3%) and for a small number of psychiatric diagnoses, avoiding the stigma of admission to a psychiatric hospital (2%).

The total costs of care for the 289 admissions at the Robinson Memorial Hospital and 220 admissions at Dalriada hospital (during the study period) were estimated at £476,376 and £485,011 respectively, compared to £773,795

and £532,484 estimated for the specified alternative forms of care including nursing home provision and home-based care. The higher district general hospital average costs were accounted for by higher levels of nursing and medical staff costs and greater overhead (fixed) costs including diagnostic costs.

TABLE 2

Primary reasons for admission

	The Robinson Memorial Hospital		*Dalriada Hospital*	
	No.	*%*	*No.*	*%*
Management/diagnosis of medical/surgical problem	181	62.6	135	61.4
Convalescence	24	8.3	24	10.9
Investigation	17	5.9	24	10.9
Respite care/holiday relief	18	6.2	13	5.9
Terminal care	14	4.8	8	3.6
Observation	9	3.1	9	4.1
Other social reasons	13	4.5	4	1.8
Rehabilitation	11	3.8	1	0.5
Other	1	0.4	2	0.9
Insufficient data	1	0.4	–	–
TOTAL	289	100.0	220	100.0

DISCUSSION

The objectives of the study were to obtain some indication of the nature and costs of care of the two general practitioner hospitals relative to alternative forms of provision. Both hospitals appeared to be providing a mainly acute 'general medical' service to elderly patients over a wide range of diagnoses. Although the range of diagnoses was similar to those of other studies with similar sized samples in a number of different locations,[8, 9] there was comparatively little emphasis on some of the roles typically attributed to general practitioner hospitals such as terminal care and rehabilitation.[5] In view of this rather atypical emphasis on acute general medical care, we compared the general practitioner hospitals with the general medicine and geriatric specialties in the district general hospital.

Although there was no direct evidence on outcome or appropriateness of care, these preliminary findings did indicate that these hospitals were fulfilling a useful role; patients improved sufficiently to be discharged within a reasonable period of time after the acute episode. There was no evidence of bed-blocking

and it would appear that neither hospital was becoming a long-stay geriatric facility, a criticism often levelled at general practitioner hospitals.[10] The general practitioners appeared systematic in the treatment of their patients and there was evidence of a management plan for all but 1% (7/509) of admissions at both hospitals. There were very few transfers elsewhere, which would suggest that admissions were for the most part appropriate and patients could be managed successfully without further specialist help. However, this does not preclude the possibility that patients could have been better managed at home or in other settings.

Hospital activity rates at the two hospitals demonstrated relatively economical use of inpatient resources and this was borne out by the crude estimate that general practitioner hospital provision was, on average, less costly to the NHS than an alternative pattern of care. The mean length of stay (20 days) and occupancy (80%) during the study period compared favourably with those found in other studies,[2] as well as those for general medicine at one of the two local district general hospitals (47 days, 68% occupancy) and for geriatric medicine at the other (63 days, 80% occupancy). However, both general practitioner hospitals failed to meet certain structural standards of quality recently cited as important for the effective functioning of such hospitals.[11] In particular, at the time of the survey, neither operated a system of clinical audit and one had no formal written admission/discharge policy.

There were a number of limitations in the design of this study. It was not possible to compare directly the relative quality and cost-effectiveness of general practitioner hospital and district general hospital provision in similar groups of patients. Two obvious limitations of the cost evaluation were the reliance on total and average costs and the assumption that the general practitioner hospital patients were as severely ill as those treated in the district general hospitals. (An inspection of individual diagnoses demonstrated that this was not the case). The higher average costs at the two district general hospitals were due to higher staffing levels justified by the more complex total caseload and the provision of facilities which would not normally be used by all patients such as an intensive care unit. Individual patient costs could not be calculated from the available data. The costs of domiciliary and nursing home care were also crude, but since they applied to only 11% of patients, are unlikely to affect the comparison.

In reality, the relative costs of district general hospital and general practitioner hospital care may depend on the ability of the former to absorb cases treated at the general practitioner hospital into its workload without additional facilities or staff. However, it was not possible with the routine data to carry out a marginal cost analysis to determine whether or not this would be the case. Occupancy levels at the two nearest district general hospitals would suggest that there was relatively little spare capacity for cases admitted to the general practitioner hospitals and therefore average costs were broadly appropriate. It was not possible in this study to look at the hospital utilisation rates of the general practitioner hospital catchment population. If total admission rates are higher as a result of the presence of these hospitals (due to increased accessibility), this could erode the cost advantage shown in the crude comparison. In addition, the cost comparison was confined to NHS costs with no allowance for patients and relatives travel.

In the absence of an independent measure of severity, there was no means of corroborating the general practitioners' professional judgements on whether hospitalisation was appropriate in the first place or whether the alternatives which they suggested were appropriate. In a more rigorous study, some form of independent review panel could assess the suitability of cases for hospitalisation. Patient selection effects may be present in these hospitals and general practitioners may tend to admit older patients with straightforward or clearly established diagnoses. It was not feasible to include a description of patients from the general practitioner hospital catchment areas who were primarily admitted to the district general hospitals in order to determine how they compared in terms of severity of illness.

It is likely, on the basis of the available data, that general practitioner hospital provision is both a partial 'add-on' (it may increase utilisation levels in the vicinity) and a less costly substitute for district general hospital services in this rural area. There is little evidence that it is a substitute for domiciliary care.

On the whole, these hospitals do appear to play an important, though somewhat poorly defined role in inpatient care. However, without further comparative research on costs and outcomes in similar groups of patients, it is not possible to show definitively whether the two hospitals make a cost-effective contribution to health services for the local populations served. Neither a review of the literature nor the results of this study provide definitive evidence that general practitioner hospitals are preferable to district general hospitals, or indeed, domiciliary care, for the types of cases they currently treat. There is a clear need for empirical research on the costs and benefits of general practitioner hospitals versus alternative forms of care. Ideally, a prospective case-control design incorporating some form of cost-effectiveness analysis would be required, preferably undertaken on a population rather than hospital basis. It may be worth comparing the costs of care, service utilisation and outcomes in socio-demographically similar populations with and without access to a general practitioner hospital. It would also be worthwhile to ask what the patients generally value most in the care they receive, whether from the general practitioner hospital, district general hospital, nursing home or domiciliary support. Without such studies, the role of the general practitioner hospital both in Northern Ireland and elsewhere will remain uncertain.

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REFERENCES

1. Smith J Weston, O'Donovan J, Hoyle G, Clegg D F G, Khalid T. Comparative study of district and community hospitals. *Br. Med. J.* 1973; **2**: 471-4.
2. Tucker H J. The role and function of community hospitals. King's Fund Project Paper 70. 1987.
3. Grant J A. Community Hospitals – time to come off the fence. *J R Coll Gen Pract* 1989; **39**: 226-7.
4. Weeks M M, Whitehouse A, Pearce V R. Care of the elderly in GP hospitals. *Update* 1985; **30**: 643-8.
5. Royal College of General practitioners. General Practitioner Hospitals. Occasional Paper 23: 1983.
6. Rickard J H. Cost effectiveness analysis of the Oxford Community Hospital Programme. Oxford: Health Services Evaluation Unit, Department of the Regius Professor of Medicine, University of Oxford, 1976.
7. Trevelyan M H, Cook J. Use of acute medical and general practitioner beds by the practitioners working in one new town. *J R Coll Gen Pract* 1974; **24**: 477-87.
8. Kernick D P, Davies S E. The community hospital: a three year study. *Br. Med. J.* 1976; **2**: 1243 5.
9. North N T, Hall D J, Kearns W E. First year of an inner city general practitioner community hospital. *Br. Med. J.* 1984; **288**: 1209-11.
10. West Midlands Regional Health Authority. Community Hospital Planning. Birmingham: WMHRA, 1984.
11. Royal College of General practitioners. Community hospitals – preparing for the future. Occasional Paper 43: 1990.

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'Assessment and care management' - a hospital perspective

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SUMMARY

Patients placed from hospital to nursing or residential homes or to home under the intensive domiciliary care scheme were compared before and after the introduction of 'assessment and care management' on the 1st April 1993. In geriatric medical wards there was a 69% increase in the average length of stay for patients assessed and care managed and a 52% increase in the length of stay for self-funding patients compared with patients placed before the introduction of assessment and care management. Care managed patients discharged on the intensive domiciliary care scheme had a 66% increase in their length of hospital stay compared with care managed patients placed in private nursing homes. In contrast, the length of stay for care managed patients in other hospital wards was half that for geriatric medical wards.

INTRODUCTION

The increased number of beds in private and voluntary nursing and residential homes in the 1980's,[1] and the associated cost to the Exchequer,[2] were factors leading to the Community Care Act (1990)[3] and the introduction of 'assessment and care management' on 1st April 1993.[4] Community care is meant to be a better option, rather than a cheaper one. Its key aim is to maintain elderly people who would otherwise need institutional care at home and if the person does need institutional care to admit them appropriately. Its key principals are comprehensive multi-disciplinary assessment of clients with complex needs, and devolution of budgets to care managers who have the power to purchase whatever was assessed as being necessary from any source.[4] 'Assessment and care management' is the process of how the need for care is assessed and then delivered, initially for nursing home and residential care. Through this process the Eastern Health and Social Services Board aims to reduce the level of institutional care in the elderly from 18% to 12% by 1997.[5]

Since more than 50% of clients placed in private nursing homes are admitted directly from hospital, the process of 'assessment and care management' from hospital is of particular importance. There have been few studies of how this process is working in hospital. A recent evaluation of care management reported no complaints about delays in assessment, discharge, or evidence of

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'blocked beds'.[6] The present study looked into placement of patients from hospital to nursing and residential homes, or to their own home, under the intensive domiciliary care scheme for three months before and nine months after 'assessment and care management' was introduced.

METHODS

Patients admitted to the Department of Health Care for the Elderly in the Ulster Hospital, under our care who were discharged for the first time to nursing or residential homes, or to their own home, under the intensive domiciliary care scheme were studied. Patients admitted after 31/12/93 were not included. Those discharged between 1/1/93 and 31/3/93 formed group 1, and those discharged after 1/4/93 via 'assessment and care management' formed group 2. Patients discharged after 1/4/93 to nursing or residential homes but not via 'assessment and care management' (self-funding) formed group 3 and those discharged after 1/4/93 from all other wards in the Ulster Hospital (mainly medical) via 'assessment and care management' formed group 4.

To avoid bias in lengths of stay, patients receiving continuing care in hospital who were subsequently discharged were excluded. Patients who died in hospital, or who were discharged to respite or convalescent care, or to rehabilitation units, or to hospices (Beaconfield or the Northern Ireland Hospice) were also excluded. Seven patients were excluded from group 4 due to insufficient data.

Information was collected retrospectively from the full case notes, and social work records in groups 1 to 3, and entered onto a computer database. Age, sex, domicile, source of admission, type of admission, date of admission, date of commencement of comprehensive assessment ('assessment and care management') by the social worker, date of referral to the care manager, main diagnosis, discharge date and discharge placement were recorded. For patients who were self-funding and for those patients who were discharged before 1/4/93, the date of referral to relatives for placement following discussions between the social worker, patient and carer, was taken as the equivalent of the date of referral to the care manager. For group 4 patients information was drawn only from social work records. This included age, date of admission, date of commencement of comprehensive assessment ('assessment and care management') by the social worker, date of referral to the care manager, discharge date and discharge placement. All group 4 patients were care managed.

Main diagnoses were divided into systems. If no underlying cause was found the diagnoses of 'falls' and 'poor mobility' were recorded under the locomotor system. The abbreviated mental test score [7] (maximum = 10) was recorded for groups 1 to 3, a score of less than 7 out of 10 was classified as dementia. The Barthel score [8] (maximum = 20) was used to measure functional ability in groups 1 to 3. A score of 0 to 6 was classed as severe disability, 7 to 13 as moderate disability and > 13 as mild disability.

The data were analysed using FileMaker Pro for Windows (Database) and STATVIEW on an Apple Macintosh computer using Chi-squared, Fisher's exact test, Anova and Fisher's protected least significant difference for use with unequal sample size (for inter-group comparison).

Intensive domiciliary care scheme.

The intensive domiciliary care scheme is a scheme introduced in 1993 to provide home care to allow an elderly person to remain at home. It is not means tested at present.

Assessment and care management.

Assessment identifies needs and determines eligibility. Care management is a process of organizing the inter-related tasks of needs assessment and the design, management and monitoring of care, centred on individual requirements.[4]

TABLE 1

Admission details, mental and functional ability and completion times of different stages in 'assessment and care management' between groups. 95% confidence intervals are in italics.

Age (years)	*Number*	*Mental test score (out of 10)*	*Barthel score (out of 20)*	*Length of stay (days)*	*Time from admission to referral to care manager (days)*	*Time from referral to care manager to discharge (days)*
Group 1 – pre-April 1st 1993						
83.9	23	5.9	9.6	35.4[c]	18.7[g]	16.6
81.2-86.6		*4.6-7.2*	*7.9-11.3*	*27.1-43.7*	*10.8-26.7*	*12.5-20.8*
Group 2 – post-April 1st 1993, care managed						
82.5	76	5.6	8.7[a]	59.8[d]	37.2[h]	22.6[j]
80.8-84.2		*5.1-6.2*	*7.7-9.7*	*51.0-68.6*	*30.0-44.5*	*17.6-27.5*
Group 3 – post-April 1st 1993, not care managed						
84.7	18	6.2	12.0[b]	53.9[e]	28.4	25.4[k]
82.4-86.9		*4.9-7.5*	*10.0-14.0*	*32.5-75.2*	*13.9-42.9*	*10.9-39.9*
Group 4 – post-April 1st 1993, all other wards, care managed						
81.6	31	—	—	29.4[f]	17.6[i]	11.8[l]
79.1-84.2		—	—	*24.6-34.2*	*14.3-20.9*	*9.2-14.4*
ANOVA		NS	p=0.015	p<0.0001	p=0.0012	p=0.031

a b (p=0.0040), c d (p-0.0022), d f (p<0.0001), e f (p=0.0135)*

g h (p=0.0038), h i (p=0.0006)*

j l (p=0.0094), k l (p=0.018)*

* Fisher's protected least significant difference test for unequal sample size.

RESULTS

Sixteen out of 23 patients in group 1, 56 out of 76 in group 2 and 15 out of 18 in group 3 were female (NS). Fourteen group 1 patients, 69 group 2 patients and 20 group 3 patients were admitted from home (NS). Eleven group 1 patients, 50 group 2 patients and 13 group 3 patients had diagnoses relating to the locomotor or central nervous system (NS). Nine group 2 patients were admitted to hospital via a domiciliary visit compared to 1 patient in group 1 and none in group 3 (NS).

There was no significant difference in abbreviated mental test scores between groups 1 to 3. Five patients in group 1, 25 in group 2 and 1 in group 3 had severe functional disability (X^2 = 13.686, p = 0.0084). The mean Barthel scores in groups 2 and 3 were 8.7 and 12.0. There was a significant difference in lengths of stay between the different groups, with patients care managed in the other wards having the shortest length of stay. This was due to a significant decrease in the time from admission to referral to the care manager between groups and from referral to the care manager to discharge (Table 1).

In group 1, 22 patients were discharged to a private nursing home and one to home under the intensive domicilialy care scheme. In group 2, 49 patients were discharged to a private nursing home (3 of which were homes for the confused elderly), 20 were discharged home on the intensive domiciliary care scheme (9 of the 20 were male compared to 8 of the 46 discharged to private nursing home, X^2 = 4.305, p = 0.038) and 7 were discharged to residential accommodation. In group 3, 14 patients were discharged to a private nursing home and 4 to residential accommodation. In group 4, 21 patients were discharged to a private nursing home (4 of which were homes for the confused elderly), 4 were discharged to home on the intensive domiciliary care scheme and 6 were discharged to residential accommodation.

Group 2 was subdivided by placement. Group 2a consisted of the 49 patients placed in a private nursing home, group 2b consisted of the 20 patients discharged home on the intensive domiciliary care scheme and group 2c consisted of the 7 patients placed in residential accommodation. There were significant differences in the rate of dementia and degree of disability between the groups. 33 (67%) of group 2a patients had dementia compared with 13 (35%) of group 2b patients (X^2 = 6.918, p = 0.0085) and 14.3% of group 2c patients (p = 0.0375, Fisher's exact test). This was reflected in the abbreviated mental test and Barthel scores. Patients discharged to a nursing home had the lowest scores . There was a significant difference in the average length of stay between the groups. Patients discharged to the intensive domiciliary care scheme had the longest hospital stays. There were significant differences between the mean time between admission and referral to the care manager and between referral to the care manager and discharge between the groups. Patients discharged to the intensive domiciliary care scheme had the longest times. There was no significant difference in mean age between groups (Table 2).

For patients placed in nursing homes there was a mean length of time of 14 days (95% confidence interval 9 days to 19 days) between admission and onward referral to the social worker by the consultant geriatrician, and of 17.5 days (11.9 to 23.1) from referral to the social worker to the case being handed over

to the care manager. Equivalent figures for patients placed under the intensive domiciliary care scheme were 15.5 days (6 to 25) and 35.4 days (19.3 to 51.5).

TABLE 2

Admission details, mental and functional ability and completion times of different stages in 'assessment and care management' between subgroups of group 2 patients. 95% confidence intervals are in italics.

Age (years)	*Number*	*Mental test score (out of 10)*	*Barthel score out of 20)*	*Length of stay (days)*	*Time from admission to referral to care manager (days)*	*Time from referral to care manager to discharge (days)*
Group 2a – post-April 1st 1993, care managed, private nursing home						
83.5	49	4.9[a]	7.2[d]	50.5[g]	31.5	19.0[i]
81.6-85.4		*4.1-5.6*	*6.0-8.4*	*41.8=59.4*	*23.8-39.3*	*14.6-23.3*
Group 2b – post-April 1st 1993, care managed, intensive domiciliary care scheme						
80.9	20	6.9[b]	10.1[e]	83.7[h]	50.9	32.7[j]
76.4-85.4		*6.2-7.7*	*8.6-11.6*	*60.4-107*	*31.5-70.4*	*17.5-47.9*
Group 2c – post-April 1st 1993, care managed, residential accommodation						
80.0	7	7.3[c]	15.3[f]	56.5	37.8	18.7
72.9-87.1		*5.9-8.6*	*13.9-16.6*	*30.3-70.9*	*19.1-56.6*	*6.7-30.7*
ANOVA		p=0.0007	p<0.0001	p=0.004	NS	p=0.048

a b (p=0.0009), a c (p=0.0087)*
d e (p=0.004), d f (p<0.0001), e f (p=0.002)*
g h (p=0.0009), i j (p=0.0161)*

* Fisher's protected least significant difference test for unequal sample size.

DISCUSSION

The concept of community care for the elderly, the mentally ill and the handicapped is that individuals need to live in a community, not an institution.[9] For 'assessment and care management' to be implemented the person at risk must first be identified.[4] The time taken for the ward social worker to fully process a case for 'assessment and care management' is 4-5 hours, on average. This workload, which involves contact with the patient (19% of social work time) and carer (25% of social work time), liaison with the care manager (10% of social work time) and other professionals, and completion of an assessment schedule (20% of social work time) and assessment of means, has not been fully allowed for in the planning process.[10] Even those patients who will be self-funding must initially be processed along the same lines as for the full care management process.

The number of subjects in the present study is small and the trends described need to be confirmed by a larger prospective multi-centre study which can better take into account the complexities of the inter-related issues that contribute to the final outcomes measured by placement and length of stay in the present study.

We found no evidence of delay in the ward social worker recognizing clients who might need 'assessment and care management'. Delays occurred when there was difficulty in obtaining adequate details from carers about the patient's financial status and social circumstances, and when carers failed to realize that care management was necessary. Confused patients who had no carers were a particular problem. The mean length of time before the consultant geriatrician referred a case for nursing home care, and for the social worker to process the case before the care manager took over, was 14 and 17.5 days respectively. This suggests that the complexity and availability of social work input into care management is an important factor in hospital stay. While an appraisal of care management[3] reported no complaints about the process, the present study showed a prolonged wait in hospital for patients subsequently discharged home on the intensive domiciliary care scheme (32.7 days for intensive domiciliary care scheme compared to 19.0 days for private nursing home, Table 2). This was due in part to the slowness of some district social services in staffing this scheme and providing adequate packages of care for severely dependent patients, and also to the complexity of arranging care for such patients. The mean length of time before onward referral to the care manager for the scheme was 50.9 days, with a mean of 15.5 days before referral by the consultant to the social worker and 35.4 days before the case was handed over to the care manager by the social worker. There was no significant difference in lengths of stay between care managed patients from South and East Belfast (mean 56 days) and North Down and Ards (mean 68.8 days) community units of management.

The average length of stay in those discharged before the introduction of care management (group 1) was 35.4 days compared with 59.8 days in assessment and care management patients (group 2). Part of this difference may be explained by the favourable financial climate which existed for placing patients in private nursing homes prior to April 1993.[11] In self-funding patients discharged after April 1st (group 3) the mean length of stay, which should have approximated that in group 1, was 53.9 days. One effect of 'assessment and care management' has been to prolong the period between admission and the case being passed on to the care of the care manager for further assessment and placement (Table 2). This prolongation was contributed to by the reluctance of some carers to consider care management, and their reticence in attending appointments with the ward social worker. The intensive domiciliary care scheme, which is not means tested at present, requires more complex planning than discharge to nursing home care. Financial concerns, availability of relatives and disputes between them, worries about ability to cope and tardiness in looking for suitable nursing and residential homes also contributed to delays in discharge. There was an average excess waiting time of 6 to 9 days for placement of group 2 and group 3 patients compared with group 1 patients (Table 1).

These results compare with a report from Bath where the mean length of stay of patients discharged for the first time to nursing and residential home care was

55.3 days in 1992 and 59.8 days post-April 1993, slightly greater than the equivalent length of stay in the present study. The numbers in the Bath study were small, it lacked any patient details, and no patients were reported as having been discharged on the intensive domiciliary care scheme.[11] In the present study lengths of stay in hospital for elderly patients care managed in other wards (mainly medical, group 4) was *half* that for patients care managed in geriatric wards. This was associated with earlier referral to the care manager (similar to pre-April lst, group 2) and a *halving* of the waiting time for placement. Mental test and Barthel scores were not available for these patients but nearly all were discharged to nursing home care and were therefore significantly disabled. There are some differences in selection of patients admitted to general medical wards compared with geriatric medical wards[12] but it would seem that care managers, ward staff and carers place more emphasis on early discharge from general medical wards than geriatric medical wards. General medical wards have much lower numbers of patients requiring assessment and care management and this may have contributed to the shorter hospital stay in this group.

In geriatric medical wards, geriatricians need to identify patients for referral to the care manager promptly, and ward social workers need to gain access to carers more rapidly to provide information for the whole process to be initiated. In doing so it is still important to provide adequate rehabilitation and not to rush the patient out to high dependency care in order to avoid 'bed-blocking.' For those patients who underwent assessment and care management from geriatric medical wards the Barthel and abbreviated mental test scores provide evidence of correct placement in nursing and residential homes. Patients placed at home under the intensive domiciliary care scheme were much more dependent than patients placed in residential accommodation.

Very few patients were placed in a private nursing home for the elderly mentally infirm, the policy being to place confused patients in ordinary nursing homes unless they have behavioural problems. In this area there is an excessive waiting time for elderly mentally infirm private nursing home places, now more than two months for some patients.

This study suggests that the process of 'assessment and care management' has led to longer lengths of stay in geriatric medical wards. The cause for this is multi-factorial, including the availability of packages of care, co-operation of carers, delays in referral to the care manager, complexity of the procedure, funding difficulties, and delay in the care manager facilitating discharge. At the time of writing there are over 30 patients in the Department of Health Care for the Elderly at the Ulster Hospital awaiting placement. Rationalization and simplification of the present number of forms required for assessment and care management is needed, along with the provision of adequate social work time. 'Assessment and care management' should be considered from the first day of admission of an elderly patient.

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REFERENCES

1. Consultative paper on the registration and inspection of private and voluntary nursing homes. Department of Health and Social Services, November 1988.
2. The Audit Commission: Managing social services for the elderly more effectively. HMSO, London 1985
3. National Health Service and Community Care Act 1990. HMSO, London 1990.
4. Assessment and care management. Implementation working group. Eastern Health and Social Services Board, Social Services Directorate, January 1993.
5. Strategy for future institutional care for elderly people. Eastern Health and Social Services Board, Planning Department, 1993.
6. People First Monitoring Evaluation. Eastern Health and Social Services Board, 1994.
7. Qureshi K N, Hodkinson H M. Evaluation of a ten-question mental test in the institutionalized elderly. *Age Ageing* 1974; **3**: 152-7.
8. Mahoney F I, Barthel D W. Functional evaluation: the Barthel Index. *Maryland Med J* 1965; **14**: 61-5.
9. Boyle M, Walkley M, Whitehead T. One method of making community care work. *Care of the Elderly* 1992; **4**: 128-30.
10. Sands B. Managing changes within a multidisciplinary team with the advent of assessment and care management. Project for ISM diploma course, Eastern Health and Social Services Board, August 1993.
11. Lewis P A, Dunn R B, Vetter N J. NHS and Community Care Act 1990 and discharges from hospital to private residential and nursing homes. *Br Med J* 1994; **309**: 28-9.
12. Todd M, Crawford V, Stout R W. Differences between "geriatric" and "medical" patients aged 75 and over. *Ulster Med J* 1993; **62**: 4-10.

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Review:

The L-arginine/nitric oxide pathway – biological properties and therapeutic applications

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INTRODUCTION

Severe sepsis and trauma are associated with protein catabolism, negative nitrogen balance and immunosuppression and represent a major threat to survival. Nutritional support in these circumstances maintains nitrogen balance and reduces both mortality and morbidity.[1-4] However catabolism associated with sepsis is not reversed by standard hyperalimentation formulations.[5] There is evidence that individual amino acids, used in pharmacological doses, may modify metabolism and modulate immune function during sepsis. Examples of such amino acids include, leucine, isoleucine and valine (branched chain amino acids) which inhibit muscle protein catabolism,[6] glutamine which improves gut immune function[7] and arginine which enhances systemic immune function.[8] This review will concentrate on the metabolic effects of L-arginine, its significance as a substrate for nitric oxide synthesis and its potential as a pharmacological agent.

ARGININE IN HEALTH

Nutrition – Arginine is a dibasic nitrogen-rich molecule and although it can be synthesized endogenously in many mammals including humans,[9] via the Krebs cycle, most is obtained from the diet. It shares an active transport system (y+) with other basic amino acids (lysine, ornithine and cystine) for absorption in the small intestine and there is also an active uptake mechanism within the kidney preventing excretion. Liver and kidney are both capable of synthesizing arginine from citrulline and ornithine via the urea cycle and by the same mechanism supply of arginine can control the synthesis of other amino acids (Figure 1). The liver has a relatively high concentration of the enzyme arginase

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(which degrades arginine into urea and ornithine), whereas renal arginase activity is low. The kidney therefore is the primary organ for conserving arginine.[10]

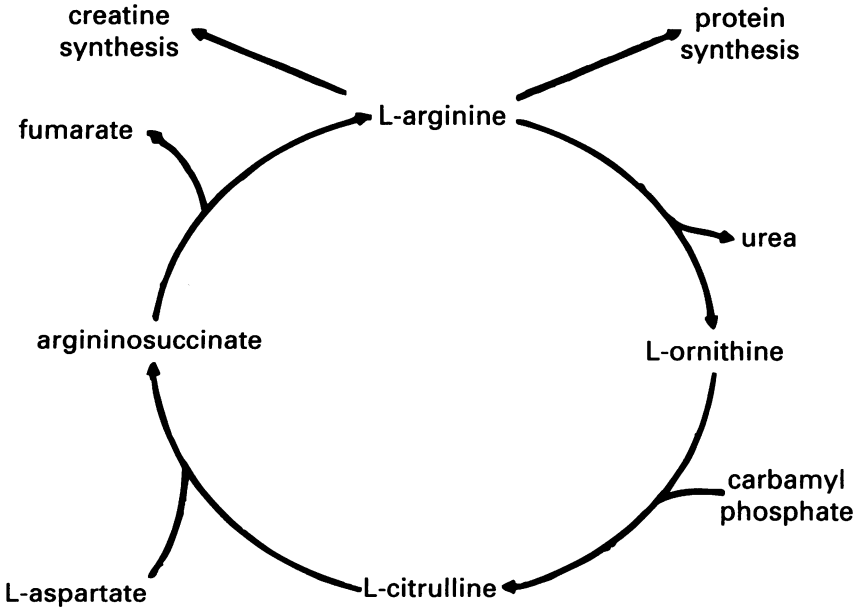

Figure 1 Krebs-Henseleit urea cycle

In health dietary arginine is not required for maintainance of nitrogen balance, but during growth or in illness and stress endogenous synthesis from citrulline is insufficient to meet body demands.[11,12] Thus arginine has been described as a semi-essential amino acid. In contrast, in vitro, arginine is an essential amino acid for cell culture systems[13] and is an essential precursor for polyamine, histone and nucleic acid synthesis which in turn are required for mitosis and manufacture of cellular proteins.

Secretagogue effects – Arginine is the most potent amino acid stimulator of insulin secretion when given either orally or intravenously.[14] There is however only a minor fall in plasma glucose concentrations due to simultaneous stimulation of growth hormone production.[15] Arginine also increases secretion of prolactin,[16] glucagon, pancreatic pancreozymin,[17] pancreatic polypeptide and adrenal catecholamine[18] but the importance of these secretory pathways is not fully understood.

Immunological effects – In vivo – Supplemental dietary arginine increases thymocyte production and thymic weight in healthy animals.[19] Thymocytes from these animals show increased proliferative responses to the mitogens phytohaemagluttinin and conconavalin A.[20] This effect is not noted in surgically or medically hypophysectomised animals suggesting that the hypothalamic/ pituitary axis may be responsible for some if not all of the *in vivo* immune effects

of arginine. In athymic nude mice arginine improves T cell function (as assessed by delayed type hypersensitivity responses) indicating that it can exert its effects on peripheral lymphocytes and not just those within the thymus.[21] Arginine by improving host immunity enhances rejection of skin allografts in mic.[22] *In vitro* – T lymphocytes taken from healthy animals and humans given pharmacological doses of arginine show increased blastogenic responses to mitogens[23-25] and also demonstrate an increase in the T helper to T suppressor cell ratio.[26] Arginine has also been shown to be essential for maximal generation and activation of cytotoxic T cells and natural killer (NK) cells.[27] Neutrophils and macrophages utilise arginine specifically for production of nitric oxide and stimulation of both cell types increases nitric oxide synthesis. Depletion of arginine from cell culture medium decreases the cytolytic activity of activated macrophages and similarly the microbiostatic and tumouristatic effects of macrophages are dependent on nitric oxide as the effector molecule.[28, 29]

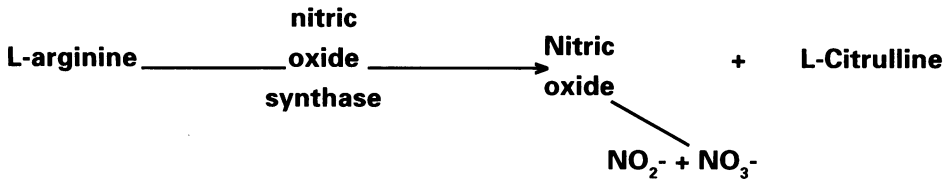

Figure 2 – Arginine /nitric oxide pathway – showing production of nitric oxide from L-arginine with L-citrulline as the major co-product and nitrite (NO_2-) and (NO_3-) as by-products.

Nitric oxide (NO) – This labile bioregulatory molecule is synthesized by many cell types from L-arginine with L-citrulline as the major co-product (Figure 2). It is highly lipophilic and therefore rapidly traverses cell membranes making it an effective intra- and inter-cellular messenger. It has a very short half-life (3-9 seconds) and must be produced in large quantities or over long periods to have prolonged biological effects.[30] When produced by cells such as macrophages it can rapidly enter microorganisms and tumour cells and exert cytostatic and cytotoxic effects by increasing cyclic-GMP synthesis and inhibiting host mitochondrial electron transport and DNA replication.[31, 32] Induction of nitric oxide synthesis has also been demonstrated in neutrophils[33] and cloned T lymphocytes[34] but its role in cellular immunological activity is uncertain.

ARGININE IN DISEASE

Following major surgery, trauma and sepsis normal intra- and extracellular amino acid homeostasis is deranged, with consequent impairment of metabolic and physiological responses. There is decreased intestinal absorption of arginine during sepsis[35] and following resuscitation from haemorrhagic shock.[36] Plasma concentrations of arginine are low in sepsis and show a strong negative correlation with survival.[37] In children with severe burns serum arginine concentrations are decreased and are associated with depressed immune function.[8]

Effects of arginine in experimental models– In various animal models intervention with arginine supplementation results in improved biological and immune parameters and increased survival.

Sepsis – parenteral administration of arginine both before and after caecal ligation and puncture results in increased survival compared with controls.[38] Oral supplementation in established acute peritonitis does not confer increased survival[39] which may be due to sepsis-induced gastrointestinal malabsorption.[5, 36]

Trauma and burns – a diet supplemented with 1% arginine improves nitrogen balance and enhances wound healing in rats assessed by increased wound breaking strength and collagen content.[19] There is an associated reduction in thymolysis which is dependent on an intact hypothalmic/pituitary axis. Arginine reduces nitrogen loss and abrogates thymolysis and T cell dysfunction following unilateral or bilateral femoral fractures.[20, 40, 41] Supplemental arginine increases delayed type hypersensitivity reactions and bacterial containment in guinea pigs with 30% full thickness burns.[42] In this burn model there is also increased resting metabolic expenditure on diets supplemented with 1% and 2% arginine.

Jaundice – obstructive jaundice results in impaired immune function with increased susceptibility to infection. Dietary supplementation with arginine following ligation of the common bile duct resulted in improved T cell function assessed by delayed type hypersensitivity reactions[43] and decreased mortality following subsequent caecal ligation and puncture.[44]

Malignancy – supplemental dietary arginine has inhibitory effects on many types of transplanted or chemically induced experimental tumours. *In vivo* arginine reduces the incidence, increases latency, decreases growth and metastatic spread, and improves survival in animals with tumours.[45-49] These effects of arginine are associated with increased macrophage phagocytosis[48] and some if not all of the effects may be dependent on the production of nitric oxide by immune cells.[50, 51] Tumours in rodents supplemented nutritionally with arginine are more susceptible to chemotherapy than those in protein depleted animals.[52, 53] These arginine effects are however dependent on the type of tumour present, as protein depleted animals with poorly immunogenic tumours do not respond as well as those with an obvious immune response.[54]

Inflammatory bowel disease – the study of malnutrition, and the immune system dysfunction in inflammatory bowel disease has stimulated interest in the therapeutic effects of dietary supplementation using various amino acids including arginine, glutamine and the branched-chain amino acids. Recent evidence suggests that there is increased mucosal nitric oxide synthase activity in inflammatory bowel disease[55] but the pathophysiological role of this remains uncertain. Using an animal model with many of the local and systemic features of Crohn's colitis,[56] and with increased nitric oxide synthase activity,[57] the involvement of nitric oxide in the disease process has been investigated. Supplemental 2% arginine in drinking water increases the severity of colitis with subsequent weight loss, thymolysis and splenolysis. This proinflammatory effect of arginine on the colonic mucosa is blocked by addition of the nitric oxide synthase inhibitor L-NAME (100mg/l) to the drinking water.[58] Similar research using a model of ileitis has demonstrated a reduction in inflammation and mucosal nitrite production following inhibition of nitric oxide synthesis.[59] This

would suggest that nitric oxide is a major mediator in the mucosal inflammatory process and that its proinflammatory effects are increased by additional arginine in the diet.

CLINICAL APPLICATIONS OF L-ARGININE

The anticatabolic and immunostimulatory effects of arginine in cell culture systems and animal models have suggested that arginine has potential as a therapeutic agent in various clinical situations. Disadvantages of oral arginine treatment include its distinctive bitter taste, and increased gastrointestinal water excretion with resultant diarrhoea.

Healthy Volunteers – in healthy humans, as in animal studies, oral arginine supplementation increases peripheral blood lymphocyte mitogenesis, decreases the number of T suppressor/cytotoxic cells and increases the T helper to T suppressor cell ratio.[24] The delayed type hypersensitivity response [24, 60] is increased as is the number of circulating NK and lymphokine-activated killer cells.[61]

Sepsis and Trauma – parenteral administration of arginine (500mg/kg/day) improves nitrogen balance and reduces protein catabolism secondary to surgery and sepsis.[62] In these situations arginine also helps maintain the urea cycle with a resultant reduction in ammonia toxicity.[63] In patients with the acquired immune deficiency syndrome arginine improves T cell mitogenesis and the T helper/suppressor ratio, but so far this has not been shown to confer increased protection against the effects of the viral infection.[8] In addition to reducing catabolism, supplemental arginine prevents reduction in lymphocytic immune response and decrease in T helper cell occurring in patients undergoing major abdominal surgery.[8, 60] Arginine therapy also improves wound healing[25] and reduces the length of stay in hospital following major cancer surgery.[60]

Malignancy – in women with breast cancer supplemental arginine increases the quantity and cytotoxic capability of circulating NK and lymphokine activated cells.[64] However in apparent contrast to animal and *in vitro* studies arginine appears to stimulate cell growth by stimulating protein synthesis within the tumour substance.[65] This paradox may be therapeutically beneficial and may be due to an increased lymphocytic infiltrate in such tumours. Furthermore this enhanced tumour growth might suggest increased cell sensitivity to chemo or radio-therapy.

Combined therapies – Recently clinical studies have included arginine as one component of enteral dietary supplementation.[66, 67] The combination of arginine, ribonucleic acid and short chain fatty acids improves blastogenic responses in peripheral blood lymphocytes from intensive care patients and those with gastrointestinal malignancies. In these studies postoperative infection was reduced, wound healing was improved and there was a significant decrease in the duration of stay in hospital.

CONCLUSIONS

Arginine is a semi-essential amino acid in health but becomes essential during periods of growth, illness or stress. In addition arginine given in pharmacological doses enhances hormone secretion and is a potent modulator of immune system function. By enhancing T lymphocyte function it improves wound

healing and the immune response to sepsis in experimental animals and humans. In animal models and in cell culture systems arginine increases macrophage activity against microorganisms and tumour cells. Therefore supplemental arginine may be of value in patients undergoing major surgery or following trauma and sepsis. Although more information is required arginine treatment may enhance adjuvant chemo therapy and radio therapy in malignant disease.

An arginine/nitric oxide pathway has been demonstrated in most cells of the immune system, and nitric oxide may be the bioactive molecule by which arginine has its immune effects. Nitric oxide has important homeostatic roles, but increased production by immune cells may result in pathophysiological changes in conditions such as inflammatory bowel disease. In these conditions where increased production of nitric oxide is detrimental and stimulates the inflammatory process, systemic and topical administration of nitric oxide inhibitors may have therapeutic potential.

REFERENCES

1. Mullen J L, Buzby G P, Matthews D C, Smale B F, Rosato E R. Reduction of operative morbidity and mortality by combined preoperative and postoperative nutritional support. *Ann Surg* 1980; **192**: 604-13.
2. Buzby G. Perioperative nutrition support. *JPEN* 1990; **14**: Suppl 5 197S-9S.
3. Moore F A, Moore E E, Ammons L A, McCroskey B L. TEN vs TPN following major abdominal trauma: reduced septic morbidity. *J Trauma* 1989; **29**: 916-23.
4. Alexander J W, Macmillan B G, Stinnett J D, et al. Beneficial effects of aggressive protein feeding in severely burned children. *Ann Surg* 1980; **192**: 505-17.
5. Siegel J H, Cerra F B, Coleman B, et al. Physiological and metabolic correlations in human sepsis. *Surgery* 1979; **86**: 163-93.
6. Bower R H, Muggia-Sullam M, Vallgren S, et al. Branched chain amino acid-enriched solutions in the septic patient: a randomized, prospective trial. *Ann Surg* 1986; **203**: 13-20.
7. Burke D J, Alverdy J C, Aoys E, Moss G S. Glutamine-supplemented total parenteral nutrition improves gut immune function. *Arch Surg* 1989; **124**: 1396-9.
8. Barbul A. Arginine and immune function. *Nutrition* 1990; **6**: 53-8.
9. Ratner S. Enzymes of arginine and urea synthesis. *Adv Enzymol* 1973; **39**: 1-90.
10. Rogers Q R, Freedland R A, Symmons R A. In vivo synthesis and utilization of arginine in the rat. *Am J Physiol* 1972; **223**: 236-40.
11. Nakagawa I, Takahashi T, Suzuki T, Kobayashi K. Amino acid requirements of children. Minimal needs of tryptophan, arginine, histidine based on nitrogen balance methods. *J Nutr* 1963; **80**: 305-10.
12. Rose W C, Haines W J, Warner D. The amino acid requirements of man. V. Role of lysine, arginine and tryptophan. *J Biol Chem* 1954; **206**: 421-30.
13. Eagle H. Amino acid metabolism in mammalian cell cultllres. *Science* 1959; **130**: 432-7.
14. Floyd J C, Fajans S S, Conn J W, Knopf R F, Rull J. Stimulation of insulin secretion by amino acids. *J Clin Invest* 1966; **45**: 1487-502.
15. Merimee T J, Lillicrap D A, Rabinowitz D. Effect of arginine on serum-levels of human growth-hormone. *Lancet* 1965; **2**: 668-70.
16. Barbul A. Arginine: Biochemistry, physiology and therapeutic implications. *JPEN* 1986; **10**: 227-38.
17. Weir C G, Samols E, Loo S. Somatostatin and pancreatic polypeptide secretion. Effects of glucagon, insulin and arginine. *Diabetes* 1979; **28**: 35-40.
18. Imms F J, London D R, Neame R I B. The secretion of catecholamines from the adrenal gland following arginine infusion in the rat. *J Physiol* 1969; **200**: 55-6.
19. Barbul A, Wasserkrug H L, Levenson S M. Arginine: Thymotrophic and wound healing promoting agent. *Surg. Forum* 1977; **28**: 101-3.
20. Barbul A, Wasserkrug H L, Seifter E, Rettura G, Levenson S M, Efron G. Immunostimulatory effects of arginine in normal and injured rats. *J Surg Res* 1980; **29**: 228-35.

21. Barbul A, Efron J E, Shaw T, Wasserkrug H L. Arginine increases the number of T-lymphocytes in nude mice. *JPEN* 1989; **13**: 7S, Abstr. 14.

22. Rettura G, Levenson S M, Barbul A, Seifter E. Supplemental arginine and ornithine promote allograft rejection. In proceedings, *183rd Meeting of the American Chemical Society*, AGFD, All, 1982.

23. Reynolds J V, Daly J M, Zhang S, et al. Immunomodulatory mechanisms of arginine. *Surgery* 1988; **104**: 142-51.

24. Barbul A., Fishel R S, Shimazu S, et al. Intravenous hyperalimentation with high arginine levels improves wound healing and immune function. *J Surg Res* 1985; **38**: 328-34.

25. Barbul A., Lazarou S A., Efron D T, Wasserkrug H L, Efron G. Arginine enhances wound healing and lymphocyte immune responses in humans. *Surgery* 1990; **108**: 331-37.

26. Barbul A, Wasserkrug H L, Sisto D A, Efron G. Arginine stimulates lymphocyte immune response in healthy human beings. *Surgery* 1981; **90**: 244-51.

27. Moriguchi S, Mukai K, Hiraoka I, Kishino Y. Functional changes in human lymphocytes and monocytes after in vitro incubation with arginine. *Nutr Res* 1987; **7**: 719-29.

28. Hibbs J B, Taintor R R, Vavrin Z. Macrophage cytotoxicity: Role for l-arginine deiminase and imino nitrogen oxidation to nitrite. *Science* 1987; **235**: 473-6.

29. Adams L B, Franzblau S G, Vavrin Z, Hibbs J B, Krahenbuhl J L. l-arginine-dependent macrophage effector functions inhibit metabolic activity of Mycobacteria leprae. *J Immunol* 1991; **147**: 1642-6.

30. Moncada S, Palmer R M J, Higgs E A. Nitric oxide: Physiology, pathophysiology, and pharmacology. *Pharm Rev* 1991; **43**: 109-42.

31. Granger D L, Traintor R R, Cook J L, Hibbs J B. Injury of neoplastic cells by murine macrophages leads to inhibition of mitochondrial respiration. *J Clin Invest* 1980; **65**: 357-70.

32. Radomski M W, Palmer R M J, Moncada S. Comparative pharmacology of endothelium-derived relaxing factor, nitric oxide and prostacyclin in platelets. *Br J Pharmacol* 1987; **92**: 181-7.

33. Wright D C, Mulsch A, Busse R, Osswald H. Generation of nitric oxide by human neutrophils. *Biochem Biophys Res Commun* 1989; **160**: 813-6.

34. Kirk S J, Regan M C, Barbul A. Cloned murine T lymphocytes synthesise a molecule with the biological characteristics of nitric oxide. *Biochem Biophys Res Commun* 1990; **173**: 660-5.

35. Gardiner K R, Gardiner R E, Barbul A. Sepsis decreases intestinal absorption of arginine. *Br J Surg* 1993; **80**: 656-7.

36. Sodeyama M, Kirk S J, Regan M C, Barbul A. The effect of hemorrhagic shock on intestinal amino acid absorption in vivo. *Circ shock* 1992; **38**: 153-6.

37. Freund H R, Atamian S, Holroyde J, Fischer J E. Plasma amino acids as predictors of the severity and outcome of sepsis. *Ann Surg* 1979; **190**: 571-6.

38. Madden H P, Breslin R J, Wasserkrug H L, Efron G, Barbul A. Stimulation of T cell immunity by arginine enhances survival in peritonitis. *J Surg Res* 1988; **44**: 658-63 .

39. Gonce S J, Peck M D, Alexander J W, Miskell P W. Arginine supplementation and its effect on established peritonitis in guinea pigs. *JPEN* 1990; **14**: 237-44.

40. Sitren H S, Fisher H. Nitrogen retention in rats fed on diets enriched with arginine and glycine. *Br J Nutr* 1977; **37**: 195-207.

41. Barbul A, Wasserkrug H L, Yoshimura N, Tao R, Efron G. High arginine levels in intravenous hyperalimentation abrogate post-traumatic immune suppression. *J Surg Res* 1984; **36**: 620-4.

42. Saito H, Trocki O, Wang S-I, Gonce S J, Joffe S N, Alexander W. Metabolic and immune effects of dietary arginine supplementation after burn. *Arch Surg* 1987; **122**: 784-9.

43. Kennedy J A, McCrory D C, Kirk S J, Rowlands B J. Effect of L-arginine on immune suppression and catabolism of obstructive jaundice. *Br J Surg* 1992; **79**: 1226.

44. Li H, Xiong S-T, Zhang S-X, Luo Y, Feng Z-H. Effect of arginine on immune function in rats with obstructive jaundice. *J Tongji Medical University* 1991; **11**: 150-4.

45. Weisburger J H, Yamamoto R S, Glass R M, Frankel H H. Prevention by arginine glutamate of the carcinogenicity of acetamide in rats. *Toxicol Appl Pharmacol* 1969; **14**: 163-75.

46. Takeda Y, Tominaga T, Tei N, et al. Inhibitory effect of l-arginine on growth of rat mammary tumors induced by 7, 12-dimethylbenz(a)anthracene. *Cancer Res* 1975; **35**: 2390-3.

47. Cho-Chung Y S, Clair T, Bodwin J S, Hill D M. Arrest of mammary growth in vivo by L-arginine: Stimulation of NAD-dependent activation of adenylate cyclase. *Biochem Biophys Res Comm* 1980; **95**: 1306-13.

48. Tachibana K, Mukai K, Hiraoka I, Moriguchi S, Takama S, Kishino Y. Evaluation of the effect of arginine-enriched amino acid solution on tumor growth. *JPEN* 1985; **9**: 428-34.

49. Moriguchi S, Mukai K, Hiraoka I, Kishino Y. Functional changes in human lymphocytes and monocytes after in vitro incubation with arginine. *Nutrition Res* 1987; **7**: 719-29.

50. Keller R, Keist R, Klauser S, Schweiger A. The macrophage response to bacteria: Flow of L-arginine through the nitric oxide and urea pathways and induction of tumoricidal activity. *Biochem Biophys Res Comm* 1991; **177**: 821-7.

51. Albina J E, Caldwell M D, Henry W L, Mills C D. Regulation of macrophage functions by l-arginine. *J Exp Med* 1989; **169**: 1021-9.

52. Reynolds H M, Daly J M, Rowlands B J, Dudrick S J, Copeland E M. Effects of nutritional repletion on host and tumor response to chemotherapy. *Cancer* 1980; **45**: 3069-74.

53. Daly J M, Reynolds H M, Copeland E M, Dudrick S J. Effects of enteral and parenteral nutrition on tumor response to chemotherapy in experimental animals. *J Surg Oncol* 1981; **16**: 79-86.

54. Reynolds J V, Thom A K, Zhang S M, Zeigler M M, Naji A, Daly J M. Arginine, protein calorie malnutrition and cancer. *J Surg Res* 1987; **45**: 513-22.

55. Boughton-Smith N K, Evans S M, Hawkey C J, et al. Nitric oxide synthase activity in ulcerative colitis and Crohn's disease. *Lancet* 1993; **342**: 338-40.

56. Morris G P, Beck P L, Herridge M S, Depew W T, Szewczuk M R, Wallace J L. Hapten-induced model of chronic inflammation and ulceration in the rat colon. *Gastroenterology* 1989; **96**: 795-803.

57. Boughton-Smith N K, Evans S M, Whittle B J R. Elevated nitric oxide synthase activity in inflamed colon from a rat model of colitis. *Gut* 1992; **Suppl**: W45, S12.

58. Neilly P J D, Anderson N H, Kirk S J, Gardiner K R, Halliday M I, Rowlands B J. L-arginine exacerbates the inflammatory response in experimental inflammatory bowel disease. *Gut* 1993; **34**: Suppl: F237, S60.

59. Miller M J S, Sadowska-Krowicka H, Chotinaruemol S, Kakkis J L, Clark D A. Amelioration of chronic ileitis by nitric oxide synthase inhibition. *J Pharm Exp Ther* 1992; **264**: 11-16.

60. Daly J M, Reynolds J, Thom A, et al. Immune and metabolic effects of arginine in the surgical patient. *Ann Surg* 1988; **208**: 512-23.

61. Park K G M, Hayes P D, Garlick P J, Sewell H, Eremin O. Stimulation of lymphocyte natural cytotoxicity by L-arginine. *Lancet* 1991; **337**: 645-6.

62. Elsair J, Poey J, Isand H. Arginine chlorhydrate on nitrogen balance during the three days following routine surgery in man. *Biomed Press* 1978; **29**: 312-17.

63. Fahey J L. Toxicity and blood ammonia rise resulting from intravenous amino acid administration in man. The protective effect of l-arginine. *J Clin Invest* 1957; **36**: 1647-55.

64. Brittenden J, Park K G M, Hayes P D, Ashby J, Heys S D, Eremin O. Effect of arginine on natural cytotoxicity in cancer patients and healthy volunteers. *Br J Surg* 1992; **79**: A442.

65. Park K G M, Heys S D, Blessing K, Kelly P, McNurlen M A, Eremin O, Garlick P J. Stimulation of human breast cancers, in vivo, by dietary, l-arginine. *Clin Sci* 1992; **82**: 413-7.

66. Cerra F B, Lehmann S, Konstantinides N, et al. Improvement in immune function in ICU patients by enteral nutrition supplemented with arginine, RNA, and Menhaden oil is independent of nitrogen balance. *Nutrition* 1991; **7**: 193-9.

67. Daly J M, Lieberman M D, Goldfine J, et al. Enteral nutrition with supplemental arginine, RNA, and omega-3 fatty acids in patients after operation: Immunologic, metabolic, and clinical outcome. *Surgery* 1991; **112**: 56-67.

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Historical Note:

Random recollections of World War II

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I was a boy at the Royal Belfast Academical Institution during World War I. I suppose one of my most vivid recollections of that period was reading the morning paper on July 2 1916 and seeing the entire front page filled with the names of those who had been killed the day before in the Battle of the Somme. I was a member of the Inst Officers Training Corps, and we took our training very seriously, because many of us thought that we would very likely be called up – in fact, I was 17 years of age when the war ended in 1918.

At the back of Inst there were trenches similar to those at the Somme. They were separated by not much more than the breadth of the Malone Road, which showed how close we were to the enemy. One of our exercises was to throw hand grenades from our trench into the enemy trench. The pin was pulled out and you counted "101, 102, 103, 104" and then threw the grenade. It exploded at 105. As the distance was so short, if we threw it too soon, the enemy might throw it back at us. Incidentally, hand grenades were very easily available because the local engineering firm of Mackies was producing them by the thousand, and it was very easy for us therefore to have any that were defective. A hand grenade was only slightly larger than a large orange, but it took a very good throw to reach the enemy trench. One of the first VC's in World War I was won by an Ulsterman who, when the hand grenade slipped from the soldier's hand, and he knowing that it would kill all those in the trench, threw himself upon it and was blown to bits.

At home, my father, a general practitioner in the Knock area of Belfast, was visiting the Ulster Volunteer Force Hospital once or twice per week to give anaesthetics. The UVF Hospital at that time was a low, single storey building in the grounds of Queen's University near to the present Whitla Hall and against a wall that separated the University from the Botanic Gardens. My mother was coming to town two or three days per week to the Old Town Hall to make bandages for the troops. She was also making pads of sphagnum moss. This was a moss found in the bog at Fair Head and when dried was used instead of cotton wool. It was gentle and absorbent. I remember well Armistice night when my mother had candles in every window in our house, red, white and blue candles in all the front windows. I mention this to show how deep was the sentiment in those days and with what great relief we welcomed the end of the war.

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This is an abbreviated version of Sir Ian Fraser's recollections, the full text being filed in the archives of the Royal Victoria Hospital.

QUEEN'S UNIVERSITY

On going up to Queen's I at once joined the OTC. It was indeed a very different OTC to the one at school. There was no longer the threat of war hanging over us; it was a very much more relaxed atmosphere. We had an excellent band, and out of this there developed a very good pierrot company called the Queen's Jesters, a very talented concert party which toured most of the major towns in Ulster. The most active part of the OTC was the medical section; we had no Territorial Army then. Today we have a flourishing Territorial Army in Ulster but no medical section in the Queen's OTC. Many members of the Jesters later reached high position in the medical world; one became a leading neurologist in Belfast, another a leading psychiatrist in New York, one became a member of Parliament in Africa, and the leading 'lady' who, with pleasure, used to show off her/his legs in elegant white stockings, became an Air Vice Marshal in the RAF medical branch. A very valuable member of the Jesters was Dicky Hunter. He at that time was No 2 in the anatomy department. He had studied art in Paris and when there had taken a great interest in the theatre. He was a regular member of the 'Claque', which gave him free tickets to any show, so his knowledge of stage management was of great value to the Jesters. Later in his career he left the anatomy department to become secretary to the university. He still kept his stage interest by becoming the ringmaster at the annual Christmas circus. Not all the university top brass approved of its chief officer appearing on stage with tall hat, whip and pink coat. Dicky was a man of many parts – a talented artist, a knowledgeable gourmet, a stretcher bearer in World War I and an interesting eccentric. Later, when I was No 3 in the department I got to know him very well and we became very close friends. The annual camp occasionally got 'out of hand'. They had a notorious visit to the Isle of Man and were asked never to return there. They were also banned from Warrenpoint. One lecture I remember particularly well was given by General Sir Henry Wilson. It is very memorable to me because he was shot dead a few weeks later by a member of the IRA on his doorstep in London.

CALL UP

When the second World War started I naturally threw my hat into the ring and expected to be called up at once, but it was only regulars and Territorials who were called up at the beginning of the war and it was not until after Dunkirk that the serious 'build up' took place. After Dunkirk, Ulster was flooded with troops. We got far more than our fair share because it meant that English troops could be stationed away from home and also in an area that was probably safer from a bombing point of view. There were so many ground troops that we had not sufficient hospital accommodation, and so the Stranmillis Teacher Training College was urgently commandeered.

Lt Col Dan McVicker, a retired regular, became Commanding Officer and I was put in charge of the surgical section. My interview for the Army was interesting. I was interviewed by the ADMS, a retired RAMC Colonel. He claimed that he had been the youngest ADMS in World War I and now was the oldest in World War II. He intended to do a medical examination but unfortunately there was no stethoscope and no blood pressure apparatus. However, he was lucky to find some coloured wool in a drawer, and when I was able to distinguish red from green my medical examination ended and I was given my commission into the RAMC. At this time I am afraid medical examinations for all services were fairly

superficial. I remember as a joke in the RAF they said that no longer did they examine your eyes, they just counted them.

After Dunkirk, in addition to the troops coming over here, we also had evacuated to us four general hospitals, each of 600 beds. One of these, a London based hospital, took over Campbell College and the pupils from Campbell had to go up to Portrush. Another took over a school in Bangor and again the pupils were evacuated. A third took over the Musgrave Park Hospital; the fourth was never given a base but remained mobile. There was always a fear that Hitler might land in the Republic of Ireland and it would be a good idea to have a hospital on wheels ready to move south at a moment's notice. On both sides of Malone Park there were trucks hidden under the trees. The fourth hospital was based on very muddy land at Whiteabbey. I remember some of the officers coming to my home to see if we could give them any old carpet from our attic as the tents were pitched on very wet grass. These four units were all manned by highly qualified specialists, and they were all frustrated with having nothing to do; in fact, some consultants resigned from the fourth hospital to go back to their consultant appointments in Oxford. I, on the other hand, in Stranmillis was doing a full surgical list almost every day in the week, so I allowed one of these hospitals to take over one full day each week, merely to let the surgeons feel that they had something to do. It was during this time when they were in Campbell that the hospital was bombed, I hope accidentally. The bomb landed on the Officers' Mess across Massey Avenue, killing a Major Ward whose son John later became a very well respected surgical specialist in Dungannon, but sadly died when still quite a young man.

WEST AFRICA

At this stage it was quite clear that the idea of a second front must be considered if we were to defeat Germany as Germany, having conquered all the countries around it, could not at that time be beaten from an attack across the channel. Italy, Germany's weakest link and so-called ally, was obviously the best approach and very correctly described by Winston Churchill as "the soft underbelly of the Axis". To conquer Italy we must first have full control of the Mediterranean. To have the Mediterranean in our hands we must control North Africa, and to control North Africa the easiest way seemed to be to start from the eastern end from where we could build up a strong force with the help and friendship of Egypt. This of course meant an enormously long supply route round Africa instead of the short route across the Mediterranean. The worst sinkings of the war took place along the West African coast. Although we had four British colonies, The Gambia, Sierra Leone, Gold Coast and Nigeria, between these was French West Africa which sadly was now Vichy France and therefore could have allowed German submarines free access and the ability to re-fuel, so it was absolutely essential that we should hold and control West Africa. I spent about 2 years there, but that is another story.

It was when I was in West Africa that Singapore fell. The immediate results of this on us all were twofold: we lost overnight our supply of quinine and also rubber. We had been taking daily doses of quinine to prevent malaria. We usually took this in the morning, but if asked out for the evening our host would always have quinine on a tray for fear we had forgotten. Now with no protection we had an outbreak of malaria, most of us going down with it, including myself.

As yet the new drug mepacrine had not appeared. When it did appear it was very unpopular; it made the skin yellow and many of the troops through it made them less amorous! As far as the loss of rubber was concerned the War Office carried out a rather interesting experiment. They asked a few fairly senior surgeons to operate for one week doing our full list with bare hands and without gloves. I found this very distressing, but I carried out my full quota of abdominal and other operations, although I did cheat in one case as there was a knee joint to be opened, which I postponed for one week because in no way could I carry out this operation without gloves. I was always very worried about my own hands, but I also was perhaps more worried about the care taken by my assistant and the Sister. Fortunately nothing went wrong.

I was rather suddenly recalled to London to be told that a new drug, which had been experimentally used for some time in Oxford, had been given to the Army and this was to be tried out in the forward areas. It was a drug called penicillin, which I had never heard of before; in fact, I did not know how to spell it, but overnight I was told that I had now become the expert in this drug. However, it was decided that I could go to Oxford for two weeks to be fully trained under Florey and get my instructions. I was to be in charge of a small unit with just a pathologist and myself. This was a combined unit sponsored by the RAMC and the Medical Research Council, and Florey wished now to give it all the available penicillin in England. He had carried out his researches in a small way in Oxford, but now wished a major experiment/trial to be carried out. Florey had carried out a lot of research into osteomyelitis, bacterial endocarditis and venereal diseases, and suggested that they should be excluded from our research programme. I was willing that venereal diseases and cardiac problems should be excluded as I felt that in the forward area they were not likely to be met. Florey also suggested that I should not give penicillin to German wounded. This I absolutely refused to agree to, as I was there to do a research job and politics did not enter into the problem.

Florey at that time was experimenting with urine recovered from patients on large doses of penicillin. The drug only remained in the patient for less than three hours. One-third was used up in the body but two-thirds was excreted in the urine. With suitable treatment half of this could be recovered – today we would call this recycling. When it was recovered it was much purer than the original drug as the impurities had been taken up by the patient's kidneys. A policeman on long and large quantities of the drug produced much of the material. It was painless when injected as compared with the impure stuff, so the 'policeman's penicillin' was much sought after. Sadly this recycled penicillin was not available for us. We spent the two weeks in Oxford partly in the laboratory and partly in St Hugh's College. I should say that St Hugh's College, a ladies' college in Oxford, had been commandeered by the Army to become the major neurosurgical unit in Britain. Brigadier Hugh Cairns was in charge but he was away most of the time and Cecil Calvert, my colleague from Belfast, was left in charge. Cairns had an idea that no form of major brain surgery should be done in the forward areas, merely the application of first aid dressing, after which the patient could be brought to a specialist hospital where he could have special neurosurgical care.

On leaving Oxford my colleague and I went aboard the hospital ship "Newfoundland" to embark for Algiers. By coincidence the "Newfoundland" on

that same day had brought back to London an old friend of mine, Ted Lewis, who had just been released from an Italian POW camp. He looked very well except that he wore a pair of awful bright yellow boots which would be totally unacceptable, and I was very amused to see that the crown and pip on his shoulder were beautifully embroidered. I found that this had been done by nuns while he was a POW.

I should perhaps at this stage say that the "Newfoundland" was later sunk by enemy action. It was a very large, elegant, white ship with the Red Cross visible from every angle, but on the occasion when it was sunk it was carrying American nursing personnel. The Sisters were wearing battle dress with tin helmets, and perhaps the ship may have been thought to be carrying troops. I hope that this was the reason. I later saw many hospital ships sunk. The Italians very foolishly allowed the hospital ships to come in close with their fighting ships and so were accidentally hit.

On arrival in Algiers we were posted to a very large base hospital of nearly 2,500 beds and allocated a ward for our research. This was most unsatisfactory and very disappointing as the cases were all longstanding ones of deep infection, having been wounded very many weeks before. There were long septic tracks and sinuses which were unapproachable by penicillin. We stayed there for a short time and then moved forward towards the fighting area where the results were very much better, so much so that I insisted that our unit should be allowed to go in each day with the invading forces so that the penicillin could be administered almost within minutes or hours of wounding. We were allowed to do this until the complete conquest of North Africa was achieved.

The next campaign in the Mediterranean was the conquest of Sicily and then Italy. This became our next objective. All of these D Day landings were indeed very different. We landed in Sicily mid July 1943 having sailed from Malta at 3.00 am. We had an unopposed landing on the beaches. The only anxiety when walking along the beaches in the dark was when we got caught up with barbed wire. We were never sure whether there might be a mine attached to it or not. Our next landing in Catania was very different. We did find a disused schoolhouse which gave us some overhead cover when operating. We slept in the open on the lava slopes looking up at Mount Etna, 10,000 feet high, from which there was always a small wisp of smoke arising with an occasional spurt of hot lava. We always hoped it would never really erupt. On arrival it was interesting to see the remains of the airborne assault. There were masses of gliders, some intact and some badly damaged. There were still a few parachutes visible but not many. Those not reclaimed were quickly used by the local inhabitants, and within a few days I saw small children with elegant shirts and skirts made from our valuable parachute silk. I have also heard it said that even a few of our QA nurses who were handy with a needle had made elegant nightdresses for themselves. It was a night drop, the parachutes were dark in colour and very elegant. The 'drop' had not been a very successful one, due either to premature release from the aeroplanes or due to unexpected bad weather and strong winds. The official record shows that out of 134 gliders as many as 47 had dropped in the sea. Sadly, one man to be dropped in the sea and never to make land was a very nice young doctor who had been my house surgeon in the Royal Victoria Hospital only two or three years before.

During our time in Sicily we took quite a few Italian prisoners. Many Sicilians were very delighted that the war was over as far as they were concerned. We used some as orderlies and I was quite amused that on some occasions they would very willingly take a drink of water to a patient but they always refused to take it to a German. Having conquered Sicily and after a short delay we then landed at the tip of Italy. This was a totally unopposed landing as the entire town had been evacuated. One could enter any house as the doors were widely open, and I am sure many of our troops did. The only memento that I picked up there was a piece of a broken statue of Mussolini which had been thrown out of an upstairs window. I found it on the roadside. I later got the head mounted and it now sits on my desk as a paperweight. Mussolini was becoming more and more disliked.

The next attack was at Salerno where again we landed on D Day. This was a very different occasion. The enemy knew of our arrival and Salerno had to be held at all costs because it opened up the way to Rome. Although we made a very successful beach landing, the enemy were still in control, hiding in caves up the mountain overlooking the beach, and although the Navy kept pounding the mouth of the caves from the sea, yet the enemy with their guns on wheels were able to retract deeply into the caves to reappear suddenly at any moment. In fact, it was only when we sent the Ghurkas up with their kukris that the enemy was finally cleared out. When we landed I was very impressed to find six young men all in uniform with no badges. I found they were Quakers; they were quite willing to go ashore with us and even to be killed while clearing the minefields, but their religion would in no way allow them to kill. One casualty a second lieutenant in the Scots Guards, I still remember very well. When admitted he had a label round his ankle which said "2nd Lt Ian Fraser, Scots Guards". He always said it was because of his name that I took him first. I am sure that that was not so. He had two bullets in his chest, one of which I removed and I told him then that the other should not be disturbed. I had a letter from him 45 years later to say that the bullet was still there, it was now next to his pacemaker.

The penicillin that we carried was of two types. One was a fine powder which we blew with an insufflator on to the open wound. The penicillin powder was well diluted with sulphonamide powder to give it bulk. The penicillin for intramuscular injection, on the other hand, was a fine brown powder which, when diluted, produced a deep yellow coloured fluid almost like mustard. This when injected into the patient caused tremendous pain because nine-tenths of the powder consisted of impurities derived from the fluid in which the mould had grown. The intramuscular injection had to be given every three hours because it passed out of the body in the urine very quickly. We unfortunately had none of Florey's 'policeman's penicillin' available. I remember very well when in Oxford seeing the then Mrs Florey going round each morning on her bicycle collecting the overnight bottles of urine and bringing them back to be processed in the laboratory. This was always known as the 'milk round'.

Our team varied a great deal depending on where we could work – sometimes on the beaches, other times in tents, occasionally we could find a derelict building, perhaps an old schoolhouse, but if the beach was not safe then we had to evacuate to a ship. I did a lot of work on two ships. One was the "Ulster Monarch", in which in peace time I had travelled many times from Belfast to Liverpool. They had converted the bar into an operating theatre. It was really

very inconvenient – low ceiling, bad light, no ventilation, small windows and, worse still, a small flight of steps from the deck which made it very inconvenient for our stretcher bearers. Another ship was the MS "David". This was a small ship that used to ply from Wales to Ireland and had been converted into a small Red Cross carrier suitable for overnight evacuation. It also had rather unsuitable operating facilities in the lounge. The "David" was later sunk, as indeed was the "Newfoundland". Sadly, when it was sunk, the medical officer on board was an old Belfast graduate, who hailed from Ballynahinch. He had been a student of mine some years before; it is indeed a small world. It is interesting to note that in this period in 1943 penicillin was still in small supply and very impure; less than one year later, for the Normandy landing, penicillin was pure and flowing freely. Of course by that stage we were getting large quantities from America.

I was taken ill myself and found myself in hospital in Salerno and Catania and was then transferred to Cairo where I spent a few days at the 15th Scottish Hospital, a very famous wartime hospital. I visited the operating theatre frequently when there, mostly to watch an old friend, Cliff Naunton Morgan, close colostomies. A colostomy was a very frequently performed operation in the forward area in any case of damage to the bowel. It had often been done rapidly as a life-saving operation, so the closure to return the bowel back to normal was not an easy one. Cliff was an expert at this particular operation, doing six or seven each day. I also had another friend in Cairo, a very skilled vascular surgeon but rather an eccentric. One day he asked me to accompany him to buy a carpet. We went to the shop where all the carpets and rugs were laid out before us, and we were given Turkish coffee. He bought nothing and we went on to another shop where the same thing happened. When we went to the third shop I asked him did he really want to buy a carpet? He said "No, but I really like Turkish coffee". I did not go into the third shop. I had seen enough carpets and rugs to last me for the rest of my life, and I also had had enough Turkish coffee. After the war I saw him frequently when he became Professor of Surgery in Manchester, and I had the pleasure of being an extern examiner with him. Maurice Boyd was still an eccentric and now had a monkey living with him.

After a short leave I was posted to Cambridge which at that time was headquarters for Eastern Command and was responsible for all the medical affairs in the South East of England. I was housed in an elegant house on the Huntingdon Road, with a lady who could not do enough for the Army. She also allowed my wife to come, which was delightful as only for a short period in Oxford had we been able to be together.

The consultant surgeon to Eastern Command was Major-General Heneage Ogilvie who had kindly asked me to join his surgical travelling club just before the war. He was based in the same house with us and so we saw a lot of him. He was writing a book on war surgery at the time and I think he had asked the War Office for someone to relieve him. As I was unattached I was given the job of acting as his deputy. It meant going all over this large area each day visiting various medical establishments including convalescent hospitals. Boarding schools had been commandeered as the dormitories were easily converted to suit our patients. One elegant well known ladies' school, Roedean, was one of them. We were amused that on the wall over each girl's bed there was a bell push button with the words 'Ring if you want a mistress'. I am afraid the soldiers rang the bells often but with little hope of success. Beneden, another girls' school,

was suitably converted. On one occasion by order of the War Office I was told that I must visit all hospitals, civil and military, from the Wash down south as far as Beneden to ensure that on a certain day they would evacuate all available patients inland and that, in addition, they would ensure they had a stock of 200 pints of blood. They all naturally willingly agreed, but all asked the same question "Please tell us when you want us to do it". Naturally I did not know the date of D Day nor, indeed, did I know myself whether I would be in England or in Normandy. This evacuation was to ensure that in the event of massive casualties in Normandy we would have enough empty beds available on the East coast. In actual fact this was not necessary as we had 22,000 beds in Normandy and I think there were only something like 14,000 casualties. Montgomery was a man who always over-prepared – fortunately.

NORMANDY

While still in Cambridge I was recalled to the War Office one day and told that I must join at once a hospital which would be going ashore shortly after D Day. I went up to Yorkshire where this unit was forming, actually in the wing of the local asylum. I had again the problem of getting to know an entirely new group of people whom I had never met before. They were all strangers to me. The CO was a regular Colonel who had been a Brigadier, and rather resented his demotion. My No 2 was a very active Canadian whose help I greatly appreciated. I also got to know the Sister in charge of the theatre who impressed me greatly. She was a tower of strength later on, older than the others, somewhat formidable but a superb worker. She and I got on very well. Each day we discussed tactics, got to know each other, and we were shown pictures of the fields that were to house our new hospital. These were all aerial photographs; naturally we knew the layout of the fields but we had no idea in what part of France they were. From these pictures we made a tentative plan for our ward: tents for our 1,000 beds, a very large area for the ambulances, (and this was so important as the ambulances often arrived at night in the blackout and so required great space), a special tent for 'triage', another for resuscitation, another for the operating theatre, with many tents far down the line for the walking wounded and minor cases who could be evacuated to England so empty beds would be always available. Eventually the time came for us to get ready to cross. We travelled all night down to Southampton in a train with the windows blacked out and then we went into a small wood or forest. On the trees here and there were loud speakers and although we were free to walk about we had to listen intently for our number being called on the loud speaker, because when the landing craft came back from Normandy we had to be ready to get into it at once.

The landing on the Normandy beach was uneventful. We never touched the beach, we had to jump into fairly deep water and wade ashore. I spoke to a very tall man beside me and said to him how lucky he was to be wet only up to the waist. He turned round and said how lucky I was because there was so little of me above the surface. There was always a danger that the landing craft might get stuck on the beach because in the Atlantic the tide retreated so quickly with a fall of something up to 15 feet between high and low water, very different indeed to landing in the virtually tideless Mediterranean. We had been given a very generous packed lunch and a tin of soup. On the top of the tin was a small projecting cap. When this was loosened and touched with a match we had at

once a can of soup so hot that one could not in any way even hold by hand. I bought one or two of these later as I thought they would be useful for a picnic on a cold day. We had quite a long march from the beach to our new area. It was made quite difficult for us as we had so often to jump into the ditch to let tanks and armoured vehicles pass, which had a much higher priority as far as the war effort was concerned. I was very interested to see the famous 'PLUTO' along the road – 'pipe line under the ocean'. This brought valuable oil for our tanks from England to Normandy.

I need not go into the details of getting the hospital going. We had tentage for 1,500 beds. We had 1,000 beds ready in five days with the remaining tentage still not used and our first intake was 500 which meant continuous working for several days. Our intake consisted of German wounded, British wounded and French civilians. We had no actual French troops here. I became very friendly with one German, a Colonel Zülch. He had been very badly injured by a Canadian tank and required very many pints of blood. He was a very nice, grey-haired, cultured man, and he and I became very close friends. I remember leaving the ward one night. I turned round and gave the patients a farewell wave; I got a very friendly response from all, particularly the Germans. At 9 o'clock next morning when I did my round these same Germans, who had been so friendly the night before, all turned away and refused to look at me. I had no idea what had happened until the Sister told me that in the middle of the night a young German had been admitted, so I went down to see this man. He turned out to be a very young soldier, 18-20 years of age, with long blond hair but very ill indeed. He was so ill that before examining him I asked the sergeant to bring me some blood at once because he needed resuscitation urgently. When this was produced our German friend opened his eyes and asked "Is this British blood?". As soon as I said it was, he said "I will not take it, I will die for Hitler", which he did some few hours later. With his death my old German friends became as friendly as they had been the night before. I did not realise how powerful the SS was until then.

Our operating theatre is worth mention. It was 'H'-shaped, the crossbar of the 'H' contained along one wall about 16 fish kettles all boiling away with primus stoves, and there was one large table covered with white towels on which our sterilised instruments were laid. The two vertical parts of the 'H' contained three operating tables. Entry was at the top and exit at the bottom. This was very important because in the blackout collisions could happen with rather disastrous results if the stretcher bearers tripped over the guy ropes in the dark. The three-table system was a very good one. While I was operating on one patient, the last case was being bandaged up and the notes taken, while the next case was being put on the third table. While operating, one was being informed about the next case, thus there was no waste of time. Another thing I was very strict about was how long an operating session should be. I insisted on eight hours. This meant eight hours operating, eight hours sleep, and eight hours operating. My young colleagues thought that they should do 12 hours but, if the work was going on continuously for several weeks, it was better to start the eight-hour system right from the beginning. I should mention that several tents went on fire when a primus stove was put too close to the side wall; I am glad to say this did not happen to us.

One patient whom I lost and which greatly depressed me was a Belgian Major with an abdominal injury. When I opened him up I found the abdominal cavity full of blood coming from a damaged spleen. With plenty of blood available I carried out what I thought was a satisfactory job, but he died some days later. We did a postmortem and I found that the bullet had made a small hole at the back of his stomach, a part which I could not see, but I still feel I should have been able to save his life. This was very sad as he had fought the whole war and was within months of seeing success. I still get a Christmas card from another man who had a torn popliteal artery which was not easy to repair, and so I was glad to get each year his card saying that he was playing squash again. I still get a card but now at 75 he no longer plays squash.

One man I remember being brought in. He had a bullet deep below his knee and when I began to speak to him I found he was an Ulsterman, Jack Kingan, a Lieutenant in the Irish Guards. It turned out that I knew his mother in Bangor so after I had finished the operation I got the Sister to bring me a piece of paper and I dropped a very quick note to his mother explaining that his life was not in any danger nor was his limb. It was very interesting to find later that my letter arrived in Bangor before the official notice from the War Office.

On arrival on the beaches I met Brigadier Arthur Porritt who was the consultant surgeon for the Normandy landing. He said "I did not expect to see you here, I thought you were off to India, but indeed I am very glad to have your help". From what Arthur Porritt had said, I felt I would be with the unit for a very short time, so when a few days later a man on a bicycle visited the camp selling Camembert cheese and knowing, or thinking, I was going home soon, I bought three or four and put them on the ground beside my camp bed in my tent. Normandy was very misty and often I could not see my bed but got into in merely by feeling it, but I knew from the smell that my cheese was still there. After two months in Normandy at Bayeux, and after the troops had made the final 'break through', our hospital became rather uninteresting. No longer were we getting the fresh casualties which made it so exciting. It was at that time I was asked to return to London. I packed my kit, but sadly, when I went to collect my cheese I found in the damp worms had got in and all that was left were empty boxes.

I was very sorry to lose contact with the Sister who had been such a help. I still remember one incident. We had been working all night with steam kettles nearby. It was very hot and we were all sweating profusely. Perhaps I was luckier than the others because I was what is today called topless. At dawn, looking at Sister I found her face was completely black. The dye from her hair had run down over her face. I got her to wash quickly before the young girls could see her. Post-war she became Matron of an important London hospital; in fact, I was asked by her to go over and gave out the prizes on one occasion. I was able to tell some stories about their charming white-haired Matron.

There was a small farewell party in the mess. I was sorry to leave all the boys who had been my close friends. I did hear later that the hospital moved up to Brussells and took over a long-existing hospital there. It became an important base hospital but it had lost the excitement of forward surgery. There were many parties and social events so perhaps I was better to move to pastures new. Before leaving I went to say goodbye to my old friend Colonel Zülch. A great

friendship had been made. My wife and I later sent some of my daughter's secondhand clothes as he and his wife had a daughter of the same age. They were very glad to get them, but later he became quite wealthy and he, his wife and son came to stay with us in Belfast. Later when he applied to the German government for a pension for his back injury he asked me to send in a report. It is interesting to think that old enemies can be such friends.

I remember so well going down to the RAF headquarters to see if I could get a flight back. I found a lot of despondent and disgruntled men. They were all heartbroken because they were the group who had been trying to feed men and supplies into Arnhem, which sadly turned out to be a disaster. However, after some delay, I found one pilot who was going back next day and was very willing to give me a lift.

INDIA : 1945

After very few days in Belfast I was asked to report at once to Poole Harbour to go to India. I said goodbye to the family with a heavy heart. I felt that I had already had enough overseas service. I was particularly sorry for my father, now well into his eighties, as I felt I might never see him again. In actual fact I am very glad to say I did. In Poole, again I met a new collection of people. They were mostly fairly senior officers of all the three services, all of us being sent out in anticipation of the proposed campaign to reconquer Malaya and Borneo. Many of us would have enjoyed being at home to enjoy the excitement of VE Day, perhaps in Paris or in London. In fact, I think we heard the good news on our way out to the flying boat – 8 May 1945.

We spent the night in an hotel in Poole and were due to be at harbour at 6.00 am next morning. We were going out to India in a Sunderland flying boat and the journey would take five days. We had overnight stops at Gibraltar, Djerba (a small island in the Mediterranean off Tunisia), Cairo, Bahrain and Karachi. It was my first, and only, flight on a flying boat. I did not know that the Sunderland could not take off from smooth water. We had to make a few journeys backwards and forwards to produce a few waves, and it was on the crest of a wave that the Sunderland could become airborne. There was a large circular cabin with aluminium seats all the way round. In the centre in an untidy mess we had put our kit and a small packed lunch. This really was hardly needed as we only flew six hours each day and mostly landed at our destination about midday. It was very nice to have half-a-day in Gibraltar with a good evening meal and an opportunity to see the monkeys.

Our cabin had plenty of window space and we enjoyed the view scanning over the sea. We were 20-30 people in all. On one occasion on the third day, when over land, the pilot said he was going to bring us down to have a good close view of the Garden of Eden. We saw the junction of the Tigris and Euphrates, but the land all round was brown, parched and bone dry. Adam, I felt, was very lucky to find a fig leaf; it must have been a different time of the year.

Having reached Karachi it was a simple train journey to Delhi which was GHQ for India. I was appointed as consultant surgeon to Central Command with Agra as the GHQ. This was a new appointment. They had already had for more than one year a consultant physician, my opposite number. This was reasonable as in the hospitals about 95% of the patients were medical problems - malaria,

dysentery, tropical diseases, etc, but now with the preparation for a Malayan campaign, with possibly many casualties, it was thought that a consultant surgeon was necessary. Living alone in a hotel I found a lonely life and often on my bicycle after dinner I would cycle out to see the Taj Mahal which was only 15-20 minutes away. With the moonshine and the croaking of the frogs it was a wonderful sight. The river Jumna which ran through Agra later to join the Ganges was also a sacred river. I had to visit Benares frequently as it was in my area and I was always astonished and appalled to see the dead bodies being brought along to be put into the Ganges or burnt on piles of wood on the river bank before ending in the sacred river from the burning ghats.

On the western side of my area was the state of Bikaner. The present Maharaja had presented to the British government at his own expense two hospitals for convalescent troops, and two small hospitals for Japanese prisoners. When we notified the Japs that we had some prisoners they said that this was impossible as Japanese were expected to commit suicide rather than be taken prisoner. However, after a visit from the International Red Cross they admitted that this was possible. On one occasion my visit coincided with a visit from Lord and Lady Mountbatten. We all had a very elegant dinner date in the open air with much elegant food covered with aspic. At six o'clock the next morning Lady Louis was due to inspect the four units with myself in attendance. At 6.00 am next day only Lady Louis and I were able to be present as all her entourage were down with diarrhoea and vomiting. Aspic in hot climates is something to avoid. It is the perfect medium for growing organisms; in fact, it is now the basic material that is used in laboratories for this purpose.

I was fortunate in India in running into so many old friends, so different to my previous trips where indeed I seemed to know so few. My job entailed visiting so many units that I was bound to meet some old Queen's University friends. Within a day or two of my arrival near Nagpur I ran into a Lt Col RAMC standing beside his broken down staff car. It was quite a surprise to me when he turned round and I found that it was John S Logan, now retired senior physician at the Royal Victoria Hospital, who has given such helpful service to his friends as archivist to that hospital. John on that occasion did not refuse a lift back to the Fourteenth Indian division at Chindwara.

Towards the end of my tour in India a sudden change took place as the idea of independence became a reality. We saw on the walls slogans such as 'British get out', 'English go home'. This did not involve my personal relationships with the Indian people except on one occasion. When on tour and coming back to my headquarters in Agra I would get the midnight sleeper train from Delhi. Usually my bearer would go along the platform and find a sleeper. If this was already occupied we always found a young officer who would jump up at once, delighted to offer me his berth while he would perhaps sleep on the floor. But this abruptly changed. My bearer would go to the same carriage, with possibly four junior officers apparently sound asleep or, if not, at least unwilling to be wakened. Now no one got out of bed, and so the brigadier slept on the floor. Independence was very rapid and its onset very rigid.

With the surrender of the Japanese on 15 August 1945 my period in India came to an end. I was told on one day that I could go home next day. I sold my bicycle to my bearer for £2. The journey was uneventful; a direct flight with one stop

to refuel. Once home I visited the Victoria Barracks to get my demob suit, shirt, socks, shoes, tie, etc. Although not of Savile Row calibre, I wore it until my friends said it was too shabby. I now had the great delight of getting a welcome from my family. My wife had done a magnificent job, and so my problem was to get to know my children, or should I say for them to get to know me.

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Historical note:

The History of the Ulster Obstetrical and Gynaecological Society

J F O' Sullivan

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"To all who have known doubt, perplexity and fear as I have known them,
To all who have made mistakes as I have,
To all whose humility increases with their knowledge of this fascinating subject.
This lecture is dedicated".

Ian Donald 1955

The need for this society, and its development over the past 40 years, must be considered in the context of the expansion of Health Service facilities, the discovery of new medical techniques and the changes in the community in general. There had been little change in the provision of services for midwifery patients after 1920 until the building of the Royal Maternity Hospital (1934) and Jubilee Maternity Hospital (1935), which replaced the Belfast Maternity Hospital in Townsend Street and the maternity unit (Ivy Cottage) at the then Union Infirmary on the Lisburn Road.

In 1936, Sir Dawson Bates, Northern Ireland Minister of Home Affairs ordered an Inquiry into Maternal Deaths in Northern Ireland. The Committee sat from 1936 until 1942 and published its report in 1943. This should be compulsory reading for all concerned with the provision of health care to pregnant women. During 1943-1944, the Nuffield Provincial Hospital Trust sent three surveyors to Northern Ireland – Dr Stanley Burns, Sir William P MacArthur and Dr Duncan Leys – who inspected all hospital services and made recommendations on the development of health services in the province. In reply to these proposals, the Northern Ireland Regional Hospitals Council published its recommendations in the famous 'red book' in 1946. The Council suggested that 70% of confinements should take place in hospital and that all patients should remain there for ten days after delivery – this would require the provision of 1,500 maternity beds. When the Northern Ireland Hospitals Authority was established on 5 July 1948, there were only 658 beds available for the care of maternity patients. Of these, 200 were in the two Belfast specialist units and the remainder were under the care of family doctors. On the appointed day there were only 11 specialists in

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Based on a lecture delivered on 5 May 1992 to celebrate the 40th anniversary of the foundation of the Ulster Obstetrical and Gynaecological Society.

obstetrics and gynaecology in the province. Six were attached to Royal Maternity Hospital, two to the Mater Infirmorum, two to the Samaritan and Jubilee Hospitals, and one was in Londonderry.

The majority of women were delivered at home and were looked after by district midwives. Many midwives were employed by the Boards of Guardians to look after the poor in their own homes. Others were in private practice and were paid by their patients. A third group of midwives were employed to look after women who did not fit into the other categories, and they were paid by voluntary charities – each large town having a Nursing Association to raise funds to pay the salaries of the nurse/ midwives. Dispensary doctors were available if called upon by the district midwife. Family doctors were booked by patients to attend them at home with a midwife. Many of these doctors or midwives opened small private nursing homes for their patients.

In 1944, many houses in Northern Ireland were in poor condition. A contemporary report showed that 15% were completely unfit for human habitation, 30% unfit and could only be repaired at vast expense, and 15% were unfit but could be repaired at moderate expense. Women were being delivered in many of these houses. Hospital confinements varied throughout the province. In 1948, in County Tyrone only 13% were delivered in hospital, women often having to travel outside the county for this; the highest number delivered in hospital was 46% in the County Borough of Londonderry. Patients delivered at home in the County Borough of Belfast had a 'back-up' facility available in the form of the consultant-led obstetric 'flying squad' based in the Royal Maternity and Jubilee Maternity Hospitals. Fees for this service, when used, were paid to the hospitals by the Belfast Corporation.

After the cessation of World War II many young doctors returned home to train as specialists in anticipation of the beginning of the National Health Service. In 1947, both the Royal Maternity and the Jubilee Maternity Hospitals were recognised by the Royal College of Obstetricians and Gynaecologists for training in the specialty. The first new specialist obstetricians were appointed in Newtownards and Omagh in 1949, and during 1951 another seven in Newry, Ballymena, Ballymoney, Magherafelt, Lurgan, Lisburn and Londonderry (replacement). These young men, after war service and a short period of training, suddenly found themselves in charge of beds but with no facilities. In many hospitals there was only one house officer shared by a general surgeon, a physician and an obstetrician. Anaesthetists, often graded as senior hospital medical officers, were not appointed until some years later, and all anaesthetics were administered by a house officer or nurse. There were no laboratory facilities. Off duty was not officially recognised.

These men needed support, guidance and companionship – even to discuss simple problems. Several informal meetings were held and it was decided to establish a society for obstetricians and gynaecologists. The idea of such a society was the brainchild of Mr Bill Laird of the Waveney Hospital, Ballymena, and he and his colleagues were guided in their deliberations by Mr H I McClure who was attached to both the Royal Maternity and Jubilee Maternity Hospitals. He advised that the society remain a learned one, have a social content and try to be linked with the Ulster Medical Society, which was an important source of postgraduate education in Northern Ireland at that time.

The inaugural meeting was held in the premises of the Ulster Medical Society, College Square East, on 5 May 1952. There were 24 founder members: G Boyd, W S Campbell, A J Dempsey, H L Hardy Greer, H C Lowry, R A E Magee, H I McClure, Professor C H G Macafee, J A Price and W R Sloan (all in Belfast); Emeritus Professor J English (Newtownards), C G Irwin (Ballymoney), W H Laird (Ballymena), S W Liggett (Londonderry), M R Neely (Newry), J H Patterson (Omagh) and J Watson (Magherafelt). The principal and senior registrars were G B Gibson, F Grant, H Kirk, A J Majury, C J H M Pinkerton, J C Purdon and M Roulston (all based in Belfast). Membership of the Society was open to all consultants and registrars and to others who held the Membership of the Royal College of Obstetricians and Gynaecologists but who had not yet become registrars.

A constitution was adopted at that first meeting. It was proposed to have four meetings per year, two in Belfast, one in a country hospital and one outside the province, and a distinguished guest speaker should be invited to one of the meetings. This format is still adhered to 40 years later. It was also agreed by the senior obstetricians in Belfast that they would not seek office as it was to be a young man's society. However, in view of his worldwide reputation, the members unanimously invited Professor Macafee to be their first president. Following this the presidency rotated between Belfast and country consultants until eventually there were more country than city members. The presidency is now offered on a seniority basis.

The first clinical meeting was held on 11 October 1952, in the Royal Maternity Hospital. The presidential address was entitled "The Doctors' Child" and Professor Macafee reviewed his management of the pregnancies of doctors' wives and female doctors. This was followed by a series of case reports and a clinico-pathological meeting with Professor J H Biggart and his staff in the Institute of Pathology.

Fig 1. Dr J M G Harley and Mr J F O'Sullivan at the first meeting of the Ulster Obstetrical and Gynaecological Society, Daisy Hill Hospital, Newry, 1957.

Soon the meetings took on a set format, always starting with an operating session. This was often the highlight of the meeting as country consultants had no means of seeing colleagues operate. The obstetrical statistics of the particular hospital where the meeting was held were presented. There were case reports and short talks, which were often the first occasion the registrars presented a paper and they were encouraged to play a full part in the society. Most meetings were held on Saturdays to allow as many as possible to attend, but the winter meeting was held on a Thursday to allow a joint meeting the same evening with the Ulster Medical Society at which the guest speaker was invited to give his lecture. This guaranteed a large audience! Eventually, all meetings of the society were held on a Thursday. The 'away' meeting was held in the hospital in which the guest speaker worked. The

first was held in Liverpool where the members were the guests of Professor T N A Jeffcoate and his colleagues. The last operating session to be held for members of the society was in the Ulster Hospital in 1971 when M R Neely demonstrated the use of a new instrument – the laparoscope. This had only recently been introduced into clinical practice in England by the late P C Steptoe who became better known for his subsequent work on *in vitro* fertilization. When the society was formed these operating sessions were invaluable, but with increasing membership only a few could watch in the operating theatres, and their benefit was lost.

In 1955, Professor Macafee presented a presidential medallion depicting the Madonna of the Street and her child. The inauguration of each president was followed by an address to the society. These have covered a wide range of topics and I divide them into several broad categories. Clinical subjects were: 'The Doctor's Child' by Professor Macafee, W S Liggett described his experience of abdominal hysterectomy in Londonderry, and W H Laird spoke on 'Vaginal repair in the management of prolapse'. Historical subjects were popular: Gavin Boyd spoke on 'William Smellie, Father of British Obstetrics' and many old books written by Smellie were on display. George Gibson chose 'Caesarean birth', M R Neely spoke on 'Hospital planning through the centuries', and Professor G Harley on 'The history of obstetrics in Belfast'. W S Campbell entitled his address 'The Samaritan Hospital Part I', and years later, M J Armstrong completed this story in 'The Samaritan Hospital Part II'. J K Houston spoke on 'The history of midwifery in the Belfast City Hospital'.

Some presidents chose educational topics: John Watson spoke on 'Medico-legal problems', R S Casement chose a difficult topic, 'Contraception and the Catholic patient' – just two years before the Papal encyclical 'Humanae Vitae' – and Professor J H M Pinkerton spoke on 'The evolution of the human pelvis'. Others preferred a recreational lecture: J H Ferris discussed his hobby of sailing and described a yachting holiday to Spain. W S Adams gave a talk on visiting war cemeteries, and described his visit to war graves of the Ulstermen killed in France during the First World War. W S Sproule chose 'A Greek tragedy' which was concerned with the origins of marathon running and concluded with his own experiences in the Belfast marathon.

Throughout the early years of the Society there were numerous complaints of inability to obtain locums to allow members to attend meetings – especially for the 'away' visits. Each member had to pay his own expenses and share in any further expense incurred in entertaining our hosts at the 'away' meeting. A formula was used for the latter where consultants paid two shares and registrars one share. Eventually, it was agreed that the funds of the Society should be used to host the guests. Later, the Hospitals Authority agreed to pay expenses and granted study leave to members to attend a maximum of three visits to any centre in the United Kingdom per annum. In the earlier years of the society, since many of the members had played representative rugby, efforts were made to make the 'away' meeting coincide with an Irish rugby match. This ceased in 1968 when members were unable to obtain tickets for a match at Twickenham because of an outbreak of foot and mouth disease in England, and in addition many of the new members were not rugby fans.

In 1977, the four Area Health Boards which had replaced the Northern Ireland Hospitals Authority decided to pay expenses for one visit outside the United

Kingdom each year. The first official visit outside Great Britain was to Malta during the presidency of the late Joe Verzin who was a native of that island. Since that visit there have been seven other visits to centres outside the British Isles, including Canada, Amsterdam, Leuven, Budapest and Paris.

Consultants working in isolation in the country appreciated the efforts of the society in helping them to improve their knowledge and skills. The 'away' meetings were invaluable. In Liverpool, in 1953, they attended five operating sessions. One session was devoted to the performance of a Manchester repair operation on a patient who had already had a subtotal hysterectomy for menorrhagia, by the then President of the Royal College of Obstetricians and Gynaecologists. Subtotal hysterectomy is rarely performed nowadays but 40 years ago it was the standard procedure, and considered best if a repair became necessary in later life because of the vaginal prolapse associated with the high birth rate at that time. The following year, during a visit to Aberdeen, the members saw a young registrar, A C Turnbull, later Nuffield Professor in Oxford, insert radium into a patient's uterus to suppress menses. In that hospital, hysterectomy, total or subtotal, was performed only in the presence of cancer or fibroids. Later Dr Turnbull read a paper on the use of radium in dysfunctional uterine bleeding in a series of 2,500 patients. At that time, vaginal hysterectomy was never performed in that hospital. In 1956, in Birmingham, Dr Burnett demonstrated his technique of burying both ovaries behind the peritoneum as a form of sterilisation acceptable to Catholic patients. This type of operation was performed at that time in many centres as it was suggested that it was temporary and could readily be reversed. In Manchester, in 1960, members saw Mr K V Bailey perform his operation of bisection of the ovaries and everting the raw surfaces in the management of infertile patients with what is now known as the polycystic ovarian syndrome.

Obstetrics was not neglected but for obvious reasons took the form of papers and case presentations. In Manchester, in 1959, the technique of x-ray pelvimetry was demonstrated: for many years, every primigravid patient booked in the Royal Maternity Hospital had x-ray pelvimetry performed to exclude cephalopelvic disproportion, but time has shown that this procedure, once hailed as a great advance, was of little value and it was abandoned in the early 1960's. Also during this visit to Manchester, Dr H C Walker demonstrated the procedure of amniocentesis and discussed his original liquor amnii studies in the management of Rhesus disease. Four years later at Liverpool in 1964, Professor C A Clarke described his original work on the discovery and use of anti D prophylaxis in the prevention of Rhesus disease. This work had developed from his hobby of breeding butterflies. Members were also told about the first clinical trials of the use of Syntometrine in the management of the third stage of labour. In Birmingham in 1973 we saw the first apparatus used in fetal monitoring with the patient mobile. In Glasgow in 1966 we had been honoured to see Ian Donald's prototype 'scanner' and to see him use it. It must never be forgotten that this man introduced ultrasound into medicine. On his retirement he was succeeded by a member of our society, Professor C R Whitfield who built on and expanded the work of his illustrious predecessor. Not all 'away' meetings were so interesting. In 1977 the Royal College of Obstetricians and Gynaecologists decided to hold 'study days' outside London – akin to the society's 'away' meetings. It was agreed that our society should join in such

meetings and the first was held that year in Newcastle upon Tyne. It was most uninteresting, being a series of lectures mainly on research subjects presented by young research fellows. Members were disappointed. It had not matched up to a visit to that town in 1955 when the society was the guest of the university department. Other 'away' meetings were held in conjunction with obstetrical and gynaecological societies in England, Wales and Scotland, and we joined obstetricians in Wales for the inaugural meeting of the Welsh Society.

Fig 2. The members of the Society meeting in Glasgow 1978 (Professor C R Whitfield was President).

Back row: I Hunter (registrar), A I Traub (registrar), G Murnaghan (QUB), D Smith (Ayr), H Ferris (Ards), A Ritchie (QUB).

Middle row: G Bancroft-Livingston (Luton), H Lamki (QUB), W Thompson (QUB), D Martin (Londonderry), K Houston (Belfast), P Pedlow (Stevenage).

Front row: A Gordon (Hull), W Laird (Ballymena), C Whitfield (Glasgow), G Harley (Belfast), T Myles (Craigavon), J Verzin (Belfast).

[E Holland (Newry), T Mulholland (registrar) and K Greig (Glasgow) not in picture].

Irish Universities award the primary degree of Bachelor in the Art of Obstetrics – a degree which the General Medical Council does not register. For generations of doctors, obstetrics was surely an art tempered by experience. During the forty years of this society, science has made marked progress in the specialty. In 1958, when the members visited various hospitals in London, they saw the Hogben test for pregnancy diagnosis being performed repeatedly in the male toad. This was a major advance as the mice used in the previous Aschheim-Zondek test had to be killed to get a result. In the middle 1960's, an

immunological test was introduced. Until that time, in everyday work, members had to send specimens of urine (with a fee of 10 shillings) to Edinburgh for a pregnancy diagnosis test. Now, the test is so simple and accurate that there is a 'do-it-yourself' kit for the patient which can be purchased from a pharmacy.

The Northern Ireland Blood Transfusion Service was established in 1946. Routine screening for blood grouping and the rhesus factor was rapidly introduced in the hospital service. When the society was founded there were laboratories only in the Royal Victoria, Belfast City and Mater Infirmorum Hospitals in Belfast and in the City and County Hospital in Londonderry. There were also several private laboratories in Belfast and one in Londonderry. Members of the society had in large part to depend on tests which they could perform in the clinical room, and at the majority of meetings there was an interesting clinico-pathological session. As hospital laboratories developed there were great expectations that biochemical tests would solve many problems, and numerous lectures were given on these tests, such as 24-hour urinary oestriol estimation, the oestriol/creatinine ratio, human placental lactogen and heat stable alkaline phosphatase. One member of the society was awarded the Blair Bell Gold Medal by the Royal College of Obstetricians and Gynaecologists for his work on the latter substance. In 1964, Erica Watchel spoke to the society on the new method of exfoliative cytology for the diagnosis of pre-malignant and malignant cells in the cervix. Members of the society were asked by the Department of Health to advise on the number of such tests that would be required per annum. After much deliberation, a figure of not more than one thousand per year was suggested. How wrong they were !

In 1966, several lectures were given on the place of electronic fetal monitoring during labour. This service eventually started in the Royal Maternity Hospital in 1969 and in the Jubilee Maternity Hospital in 1971. Although members had seen ultrasound scanning in Glasgow in 1966, it was not until 1971 that a commercial machine became available to the staff of the Royal Maternity Hospital. As the Health Service developed, more consultant-staffed units were opened, the last in 1962. Further expansion resulted in many of these becoming 'two-man' units over the next 10 years. In 1973 the Mater Infirmorum Hospital joined the National Health Service, although their obstetrical staff had always been members of the society. During the 1990's it is anticipated that the number of new consultants will increase with the reduction of junior doctors' hours, but this will only mean a slight increase in membership of the society as some junior posts will inevitably disappear.

Pain relief in labour has always exercised the minds of the obstetricians. Drugs used have varied from chloral hydrate through scopolamine to pethidine. Dr Muir of Glasgow spoke to the society in 1968 on the benefits of epidural analgesia in labour. This service is now widely used by members living in Belfast and Ballymena, but still depends on the availability of our anaesthetic colleagues. Care of the newborn was vastly improved by the development of the neonatal service in which paediatricians devote themselves entirely to the care of the newborn. As a mark of respect to our colleagues in the other specialties mentioned, the society has invited several pathologists, anaesthetists and neonatologists to become Honorary Members of the society. Other Honorary Members are the Professor of Endocrinology and the Professor of Genetics.

During the past 40 years, the standard of training for membership of the Royal College of Obstetricians and Gynaecologists has improved, the content of the examination has altered and the format of the examination has changed. The changes in the examination followed recommendations by one of our members, the late Professor C H G Macafee. Some of the improvement in midwifery has been related to the introduction of family planning. The first such clinic in Belfast was at The Mount but it closed after the last war. There was then a very limited service in the Royal Maternity Hospital. The Family Planning Association, working closely with our members, gradually introduced a service, and Dr Joyce Neill and Professor Pinkerton lectured to members of the society. The introduction of the oral contraceptive pill, and making family planning a fee-for-service part of the Health Service, changed the whole concept and members of the society played a prominent part in this development.

From the beginning a guest speaker had visited the province every year, with expenses at first paid by the Ulster Medical Society. Through the efforts of Professor Pinkerton and the generosity of the Ortho-Cilag Company, an annual lectureship was established, which rotated between a clinical and a scientific subject. The first lecture under these auspices was given by Professor N Morris in 1969. With sponsorship from the Syntex Pharmaceutical Company, I was able to establish an annual Registrar Prize to be awarded to a registrar in obstetrics, neonatology or anaesthesia for original work, which was first awarded in 1975. Since that date there was only one year in which the work submitted was considered not to be of a high enough standard for the prize. These grants from the pharmaceutical industry have been of great benefit to the society.

In reading the minutes of the society one is often struck by a sentence written by the recorder of a meeting. For example in 1955 it was recorded that Gavin Boyd used glass slides in a magic lantern to illustrate his talk – nowadays, no lecture is given without the aid of slides and maybe even a video record. At a meeting in 1959, John Watson described the use of symphysiotomy an operation no longer practised. In 1963, M R Neely spoke on induction of labour using a stomach tube in patients with an unfavourable cervix. In 1968, C R Whitfield described the use of the first 100 vials of anti D serum in the prevention of Rhesus disease. Our worst attended meeting was with the Ulster Medical Society and other societies who held a residential study group in the Slieve Donard Hotel, Newcastle, in 1981. Only 11 members of our society attended our own part of the meeting – it was held on a Friday !

What of the future? Obstetricians have always investigated and discussed maternal mortality, perinatal mortality: this form of medical audit has now become compulsory. The meetings were a place where one learned from ones' colleagues. There was no other source of learning about the specialty apart from the Royal Society of Medicine. The Royal College of Obstetricians and Gynaecologists remained chiefly an examining body for many years. It was only in 1961 that they introduced two study days per year and then in 1970 they introduced the consultant refresher courses which were held on alternate years. Soon, continuing medical education will be compulsory if a member wishes to be retained on the specialist register.

The society has fulfilled the aims of its founders. It has remained a learned society, raised the standards of obstetrics and gynaecology in the province and has encouraged friendship among all its members. It has grown from the 24 foundation members to the present 110 members, active, retired and honorary. We have seen many changes. In 1952 when the society was formed there were 28,760 births, 31 maternal deaths and 11 deaths associated with pregnancy in Northern Ireland. Just over 25% of births took place in the care of members of the society. In 1992 there were 25,572 births, no maternal deaths and no associated maternal deaths. Ninety-five per cent of all births were now looked after by members of the Society. Stillbirths were not registered until 1961, so no figures are available for 1952 but in 1962 the stillbirth rate was 22.0 per 1,000 and the perinatal mortality rate 38.0 per 1,000. In 1992 the stillbirth rate had fallen to 4.7 per 1,000 and the perinatal mortality rate to 8.0 per 1,000. Members and honorary members are proud of this achievement.

Many of the changes presently taking place in the National Health Service are really based on a shortage of money. With the severe cuts in the amount of money allocated to travel by Trust Hospitals, the society study days will again be most important in postgraduate education and will contribute to the further improvement of the health of our patients. The President of our College always ends Council meetings by saying "May the College flourish". May I end this talk by congratulating members for their work during the past 40 years and also say "May the Ulster Obstetrical and Gynaecological Society continue to flourish".

I wish to thank Mr H Kirk, Mr W H Laird, Mr M R Neely and Mr J Watson, founder members of the society, for their help in preparing this talk. Mr W Sproule, President, kindly granted me access to the records of all the clinical and committee meetings of the Society. The photographs were kindly supplied by Mr P Pedlow. My thanks are also due to Miss May Weller who typed the script.

FURTHER READING

Donald I. Practical Obstetric Problems, 4th edition. London, *Lloyd-Luke* 1955.

Registrar General's Reports for Northern Ireland, 1952, 1962 and 1992. Belfast, *HMSO.*

A Survey of the Hospital Service of Northern Ireland. The Nuffield Provincial Hospitals Trust, Belfast, 1944.

A Plan for the Hospital Service of Northern Ireland. N.I. Regional Hospital Council, Belfast, 1946.

Report of Maternity Services Committee on Maternal Mortality and Morbidity in Northern Ireland. Belfast, *HMSO*, 1943.

Annual Reports of the Northern Ireland Hospitals Authority 1948-1972.

The Ulster Medical Journal, Volume 63, No. 2, pp. 223 – 234 October 1994.

Historical note:

Thomas Houston and the founding of clinical pathology at the Royal Victoria Hospital, Belfast.

M G Nelson

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At the turn of the century there was an exciting expansion of knowledge and understanding of disease. William Osler commented "diseases familiar to our fathers and grandfathers have disappeared – the public health measures have lessened the sorrows and brightened the lives of millions". It was the age of medical individualists, of men of outstanding ability, breadth of learning and great clinical skill. The doctor of this period was dependent upon his knowledge, his judgement, his five senses, few instruments and very few effective remedies. Help from a laboratory was only provided in teaching hospitals by University Departments of Pathology, which maintained autopsy and biopsy services and also performed some bacteriological examinations. As the clinical, as well as the academic commitments increased in these departments it became necessary to appoint assistants. In the year 1900, a Dr Thomas Houston was appointed Assistant Pathologist to Dr Lorraine Smith, then Lecturer in Pathology to the Queen's College Belfast, and Pathologist to the Royal Victoria Hospital. On that day clinical pathology at the Royal was born, for it was largely due to the efforts and influence of this man that the hospital laboratories, as we know them today, came into being.

Thomas Houston was born in 1868 at Ballyclabber in the Route and he retained his rugged North Antrim characteristics all his life. A son and grandson of the Manse, he came, via Coleraine Inst, to the Queen's College, Belfast, then a constituent college in the Royal University of Ireland, where he graduated with honours. After qualification, he was appointed to the Belfast Royal Hospital, first as house surgeon and house physician and then as assistant gynaecologist. He took his Doctorate of Medicine in 1899, which was the year that the hospital changed its name by charter to the Royal Victoria Hospital. The following year he embarked on his career as a laboratory worker.

The duties of the newly appointed assistant pathologist were clearly laid down to him as follows: That,

i) He shall attend at the hospital at 10.30 am at least four days a week and at other times when required.

M G Nelson, MD, FRCP, FRCPI, FRCPath, DTM and H.
Formerly Consultant Clinical Pathologist to the Royal Victoria Hospital, and Honorary Professor of Haematology, The Queen's University of Belfast.

ii) He shall assist the pathologist in the examination of cases in the hospital at the request of the physicians and surgeons in charge.

iii) He shall make arrangements by which he shall be in a position to furnish immediate reports on specimens submitted by the staff.

iv) He shall be responsible for the preparation of microscopic specimens and for the efficiency of the hospital museum.

v) He shall assist the pathologist in special investigations in the hospital which the pathologist may arrange to carry out.

vi) He shall, in the absence of the pathologist, take charge of the department.

It can be appreciated from these conditions of service that the discipline of clinical or hospital pathology was, at that time, not highly regarded. Indeed, it was more than a quarter of a century before clinical pathologists received their due recognition as fellow members of equal standing with physicians and surgeons on the medical staff of hospitals.

The old Belfast Royal Hospital in Frederick Street, to which Dr. Houston was appointed, was then regarded as grossly out of date and inadequate and an entirely new hospital was being planned. This new Royal Victoria Hospital, of some 300 beds, was built on the Grosvenor Road site and officially opened in 1903 by King Edward VII. A communication addressed to medical students at that time stated "This Institution presents excellent opportunities for the study of medicine and, being the largest hospital for the reception of injuries and surgical diseases occurring in the large manufacturing city and seaport of Belfast, it affords unusual facilities for acquiring a knowledge of practical surgery". However, in this new hospital, facilities for clinical pathology were both poor and primitive, consisting of a few dingy rooms on the hospital premises and the grace and favour use of university laboratories.

Towards the end of the 19th century, two techniques had revolutionised the study of the blood. One was a method of counting blood cells and the other was a method of staining them, using aniline dyes. "Haematology at once threw off its colourless shackles", said Dameshek, "and delighted its devotees with dazzling colours". Many were attracted to a study of diseases of the blood by the beauty of these stained preparations, and the science of haematology was initiated. Dr Houston was then appointed in 1905 as haematologist to the Royal Victoria Hospital and as a member of the auxiliary staff. This, as far as can be ascertained, was the first appointment of a haematologist to a hospital in Great Britain or Ireland. In the following year the new appointee reported 43 cases of anaemia, two of haemophilia, three of splenic anaemia, two of pernicious anaemia and one of splenomegalic leukaemia.

Early in his career Dr Houston had fallen heavily under the influence of Dr Almroth Wright, the dominant force in bacteriology of his day. Both were Irish and sons of clergymen, both had been educated in Ulster and had met during family holidays in Ballycastle. Almroth was descended from the Wrights of Donnybrook, Co Dublin, and his father, Charles Wright, was a peripatetic intellectual, scholar, teacher and divine. His mother, Ebba Almroth (after whom he was named) was, like her husband a zealous Protestant, and was descended from a distinguished Swedish scientific family. After chaplaincy in a number of

Fig. 1 Sir Almwroth Wright.

churches on the Continent, Charles Wright came, with his wife and family to Belfast and became Rector of St Mary's parish, where he remained for eleven years. His son, Almroth, 13 years old at the time, went to the Belfast Royal Academical Institution. He subsequently entered Trinity College Dublin in 1882, gaining his BA with first class honours (and Gold Medal), and qualified in Medicine the following year. After periods in Cambridge and Germany (where he worked under von Recklinghausen and others) he was appointed the Professor of Pathology at the Army Medical School in Netley. Here he began teaching the new science of bacteriology. For diagnosis and identification of bacteria, he used very simple apparatus in a system he described as "the technique of the teat and the capillary glass tube". From diagnosis, Wright rapidly progressed to the treatment and prevention of bacterial infections and is probably best known for his work in the prevention of typhoid fever. In 1902 he left Netley for St Mary's Hospital, Paddington, where he established a world renowned laboratory which not only carried out diagnostic tests, but produced a great variety of vaccines and ran a busy vaccine therapy clinic, as well as carrying out an extensive research programme. Eager young men came to his department to work under the master. One of these was to discover penicillin and gain worldwide renown, and the laboratory at St Mary's was eventually renamed the Wright/Fleming Institute. Almroth Wright was knighted in 1906 and lived to a ripe old age, forceful and controversial to the end.

Physician in charge of haematology and vaccine therapy

Despite the limited facilities available to him, Dr Houston carried out an investigation, with Dr John Rankin, into an outbreak of cerebrospinal fever which occurred in Belfast in 1907. Using the opsonic index and the "the rubber teat and capillary glass tube" methods of Wright, they studied the meningococci causing the outbreak and published their findings in the Lancet.[1] Two years later, in association with Dr S T (later Sir Samuel) Irwin, another paper appeared in the Lancet on the successful treatment of a typhoid carrier by inoculation with typhoid vaccine.[2]

The King Edward Memorial Building

King Edward VII, who came to the throne in 1901 and who presided at the opening ceremony of the Royal Victoria Hospital on the Grosvenor Road in 1903, died in 1910. Towards the end of that year, Sir John Byers, visiting gynaecologist to the hospital, informed the Medical Staff "that he had been

present at a meeting of some of the leading men in the city to consider the most appropriate form which the proposed memorial to the late King Edward VII should take". It has been decided by the meeting that the most suitable memorial would be an extension of the Royal Victoria Hospital. The Board of Management had accepted the suggested scheme and had requested the staff to formulate their demands so that some definite proposal could be laid before a meeting of the citizens, to be summoned in September 1910.

It was understood by the Board and by the City Hall committee that the proposed building should afford proper accommodation for the electrical department of the hospital and should also include a department for vaccine therapy and haematology. The space requirements for the electrical department were put forward by Dr Rankin and for haematology and vaccine therapy by Dr Houston; the equipment required cost about £500. The total space requested for haematology and vaccine therapy was a little under 1,500 sq ft, of which a mere 312 sq ft was allocated for laboratory purposes, the remainder being given over to vaccine therapy including 800 sq ft of waiting space for patients. At the meeting of staff when this was approved it was also recommended that "The Visiting Staff be increased by the addition of a Physician in Charge of the Department of Vaccine Therapy and Haematology, and a Physician in Charge of the Electrical Department". This promoted Drs Houston and Rankin to what would now be called consultant status.

Fig. 2 Dr T Houston at the laboratory bench.

Concern was soon expressed at a Medical Staff meeting in 1913 at the lack of facilities for clinical pathology in general within the hospital. Two proposals were made, one to recommend the establishment of facilities in the clinical classrooms of the medical wards, consisting of "a laboratory bench with equipment as specified – the cost not to exceed £105". The other suggestion was to create a post of a "full time officer to carry out the clinical pathological work under the direction of Professor Symmers (Professor of Pathology) and Dr Houston". The first of these proposals was acted upon, the second was frustrated by the outbreak of World War I in 1914.

The report of the activities of the haematology and vaccine therapy department for 1914 contained the following statement: "During the year, 290 new cases were received and 3,320 treatments given and considering the disadvantages under which the work has hitherto been done for want of room, these figures are most encouraging. The work requires an enormous amount of laboratory investigation but up to the present it has been seriously hampered for want of proper accommodation. This defect will, however, be remedied in the near future by the opening of the new King Edward VII Memorial Wing in which a suite of rooms will be devoted to the work of this department". The building was completed and officially opened in 1915. There was space for a new laboratory but this could not be used as the staff who were expected to work in the laboratory were serving in the war in France. As an interim measure Dr Rankin was appointed to be responsible for the laboratory and vaccine therapy clinic during the war years, and the planned clinical pathology services had to wait until the end of the war before they could really begin to operate.

Dr Houston at war

Thomas Houston was one of many members of the staff of the Royal Victoria Hospital who offered their services during World War I. He was posted with Dr McCloy, a fellow Queen's graduate, and Mr Willix, a laboratory technician, to the laboratory of the St John Ambulance Brigade Hospital in Étaples, France, with the rank of major. Here he developed an interest in the enterococcus, which remained an abiding passion all his life. He published a paper with Dr McCloy on the relation of the enterococcus to trench fever.[3] During his time at Étaples the laboratory was frequently visited by Sir Almroth Wright, from headquarters at Boulogne, which served to cement their lifelong friendship. In 1916 both hospital and laboratory were destroyed by aerial bombardment. After the raid the remaining laboratory equipment was salvaged and the whole operation moved to a safe area in Deauville on the Channel coast. During the war, Major Houston gained much practical experience in blood transfusions, which were to prove invaluable to him in peacetime and to the development of a transfusion service in Northern Ireland.

Post World War I

The year 1919 saw not only the end of the war but other important events in the Royal Victoria Hospital and its clinical pathology laboratory. Major Houston returned from France, having been twice mentioned in despatches, and awarded the OBE. It was not much of a laboratory to which the war hero returned, a suite of four empty rooms with one old autoclave. One of the very first things he did on his return was to persuade the medical staff to create a post

for Mr Willix, his faithful wartime technician, to join Mr McWatters, the only other technician in the laboratory. The next was to get some equipment. Fortunately the surviving laboratory equipment of the St John Hospital, Étaples, had been given to Major Houston by the St John Ambulance Brigade but he could not get it over to Belfast because sea transport was so busy. Through the usual grapevine, this difficulty reached the ears of one of the greatest friends and benefactors of the Royal – Lord Pirrie – who immediately despatched his private yacht to France and brought back most of the equipment. This was put into the King Edward Memorial Building and the laboratory duly opened. In its first year over 2,000 pathological examinations were carried out. The first resident clinical pathologist, Dr N C Graham, was appointed in the same year as assistant to Dr Houston, who retained the title of Physician in Charge of Haematology and Vaccine Therapy.

Dr Norman Clotworthy Graham

Dr Graham started his career as a bacteriologist in 1914, when he was appointed by the Medical Research Council to study the types of M. tuberculosis in Northern Ireland. Shortly afterwards, he enlisted in the Royal Army Medical Corps and during the war he was a Battalion Medical Officer in the famous Ulster Division in active service in the battlefields of Flanders. He was wounded in action, and was awarded the Military Cross. While convalescing at a base hospital he gravitated once again into laboratory work as a form of occupational therapy. The hospital Commanding Officer, a tempestuous Irishman from Cork, made repeated attempts to post Captain Graham into the Connaught Rangers

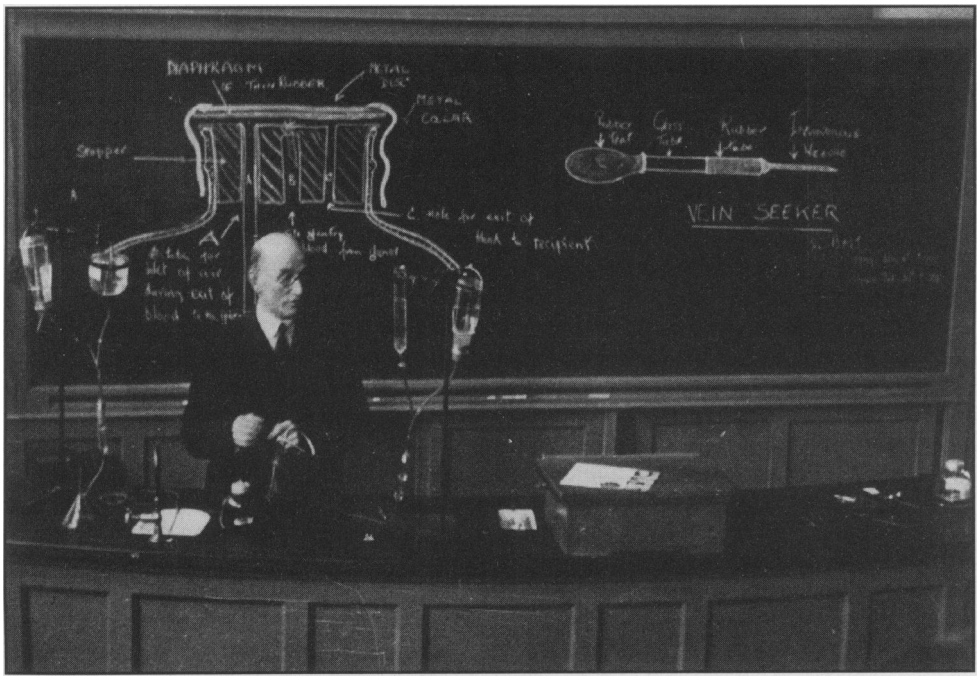

Fig. 3 Dr N C Graham demonstrating blood transfusion apparatus. (note the 'vein seeker' which is the main item illustrated).

but this was, for a time, resisted by his fellow medical workers. However, the higher command eventually plucked him out of the laboratory and dumped him into the Royal Flying Corps Medical Service, then very much in its infancy. Here he spent a considerable amount of his time visiting operational squadrons to deal with 'flying stress' among aircrews. It was during this period that he was involved in the postmortem examination on Baron von Richtofen, the great German flying ace, in order to decide whether he had been shot down from the air, as claimed by the Flying Corps, or by Army ground fire.

When Dr Graham returned to Northern Ireland after the war, he was appointed the first resident Clinical Pathologist at the Royal Victoria Hospital. However, he left this post after a short period to become Bacteriologist to the City of Belfast, and was subsequently appointed in 1926 as Lecturer in Bacteriology to The Queen's University of Belfast. He was affectionately known by generations of medical students as "Koch" or "the Hyphe". In the University department he acted as adviser to the hospital clinical pathological laboratories.

In 1919 there was a considerable extension of the serological division of the hospital laboratory. The government of the day was much concerned by the high venereal disease rate at the end of World War I and introduced a comprehensive venereal disease control programme. The Royal Victoria Hospital was considered a suitable centre and the laboratory at the Royal the place in which the investigations required for diagnosis could be carried out. During the first year over 3,000 Wassermann tests and over 300 other tests were carried out. These tests were paid for by local Government on a 'fee for service' basis and became an important and regular source of personal income for Dr Houston, whose post was that of an honorary, and therefore unpaid, member of the medical staff.

Clinical biochemistry

The fourth event in 1919 was the establishment, under Dr Houston, of a new section of the laboratory in an adjacent room in the King Edward Building to deal with biochemistry. This was one of the first hospital biochemical laboratories in the British Isles. Dr J A Smyth was appointed to take charge of this new development. Prior to the report by Folin and Wu in 1919 of a method to obtain a protein free filtrate of blood, biochemical tests had been limited. Following this discovery, clinical biochemistry blossomed, so that when Dr Smyth became Resident Biochemist to the Royal in 1922, and provided with new equipment and reagents, he was able to exploit a new, exciting and expanding area of medicine.

In 1923 the Medical and Surgical Staff reported "a biochemical laboratory has now been in existence for over a year. It is in full working order and during the past twelve months 1,400 examinations have been made. These have been mainly in the newer chemical methods of investigation, found so useful by physician and surgeon alike. A great factor in the volume of work coming to this laboratory has been the discovery of insulin by Banting and Best in the University of Toronto. This substance is the active principle of the Islets of Langerhans in the pancreas or sweetbread. It controls the utilisation of sugar in the system and is what is lacking in diabetic patients. The first use of insulin in human diabetes was in Toronto in 1922 but it was not until a year later that it

became available for use in the British Isles. It was possible to utilise it in this hospital at a very early date, owing to the ability of this laboratory to undertake blood sugar estimations and the other work necessary in connection with it. More than 60 patients have been treated with insulin and it has proved an invaluable addition to the treatment of diabetes. The whole outlook in this disease has been altered. From a steadily progressive and ultimately fatal disease, it may now be regarded as one which can be kept in check and, with proper care, need not shorten life. Without a biochemical department, the utilisation of insulin, this valuable remedy, would have been impossible. The Medical Staff take this opportunity of thanking the Board of Management for the generous and ready manner in which they voted the necessary funds for the purchase of insulin".

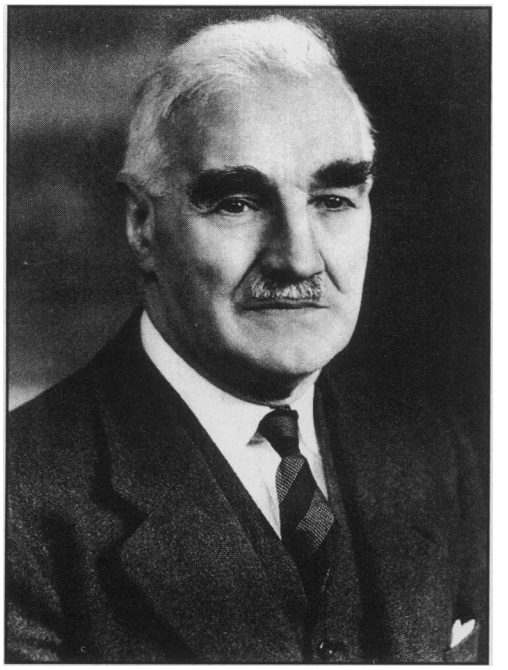

Fig. 4 Dr J A Smyth

The troubles

The early 1920's was the time of 'The Troubles' in Belfast and the Royal Victoria Hospital was in what was called in local parlance "a bad area". Frequent skirmishes between the security forces and the IRA took place in its environs. Dr Houston seemed quite oblivious of the dangers and would drive up the Grosvenor Road to his laboratory after dark with rifle fire criss-crossing the road. This was encapsulated in a couple of verses contained in the symposium in his honour on the conferring of his knighthood in 1927.

Nocturne 1921-22

The gunmen lurked round the Dunville Park
The lamps are out and the streets are dark
On the Grosvenor Road a corpse lay stark,
And the 'Specials' walked in dread.

But Tom, in the lab, ignored the thugs
And nightly tended his much loved bugs,
And coaxed the juice from the rabbits' lugs,
And calmly drove home to bed.

At the next staff meeting, Colonel Mitchell extended the good wishes of the staff to Sir Thomas Houston on the honour which had been bestowed on him. There is no record that Sir Thomas replied or thanked the staff, but he immediately referred to the milk supply of the hospital, as a sample of milk examined by the Public Health Authority had been found to contain "tubercle". A committee of

investigation was set up and a month later reported. It is interesting to note that the supplier was a Mr Morrison of Ballydrain, whose property was sold in 1958 to Malone Golf Club, so that the land on which the cattle grazed to supply the Royal Victoria Hospital with their milk has now become a golf course.

Despite his obsession with the enterococcus and with the therapeutic value of vaccine therapy, Sir Thomas maintained his interest in haematology. The report of the Pathological and Biochemical Laboratories and Vaccine Department for 1927 recorded "The Medical Research Council has entrusted to this laboratory, among others, the testing of the new liver extracts for the treatment of pernicious anaemia. These extracts are the final outcome of the work of Drs Minot and Murphy of Boston who, in 1926, reported the therapeutic value of liver in the diet of pernicious anaemia patients. After the clinical trials are completed the Medical Research Council will sanction the sale of these extracts. There is no doubt that this 'liver' diet has revolutionised the treatment of this disease. The extract will prove of great value to those patients who are unable to take whole liver". In the following year Sir Thomas was able to report among the 'blood affections', very satisfactory results in the treatment of pernicious anaemia by liver diet and liver extract.

Blood transfusion

During World War I, Dr Houston had gained considerable experience in blood transfusion technique. The service volunteer donors were rewarded with money, a bottle of port and a weekend pass. When he returned to Belfast after the war he found that recruitment of civilian donors was not so popular. In 1924 he reported "an increasing number of blood transfusions have been done by the laboratory staff during the year. It is thought advisable to have a permanent list of volunteer donors. An appeal was made to the public, through the press, and a good response was obtained. Laboratory staff have now a list of 74 tested donors who are willing to give their blood for transfusion in the case of an emergency". It is apparent that this waiting list was not adequate for the purpose, for in March 1927 the importance of blood transfusion, not only in medical cases but in surgical cases as well, was recorded and it was considered necessary to have a further list of persons willing to give their blood when required. "On several occasions the hospital had appealed to the healthy public of Belfast . . ." The response was said to be generous but not to be really sufficient to meet the requirements. "They had never any real difficulty in getting donors but they felt it was rather a hardship to call upon the same persons to give of their blood two or three times in the year. He wished to take the opportunity of thanking all those who had volunteered and especially members of the Royal Ulster Constabulary, who sometimes, in the middle of the night, had come to their assistance and given their blood". He thought it only required a little explanation to the healthy, male citizens of Belfast to secure a list of donors large enough to meet all requirements. It was the normal practice of this period for blood transfusions in the Royal Victoria Hospitals to be carried out by the staff of the clinical pathology laboratory. It was they, in times of emergency, who summoned the donors, either through Toc H or the Royal Ulster Constabulary, determined the blood groups, carried out the compatibility tests and transfused the blood into the recipients. It was not until World War II that resident medical officers and others were instructed in the technique of administration of blood.

The Institute of Pathology

In 1928, The Queen's University announced the intention of building and equipping Departments of Pathology and Bacteriology adjacent to the Royal Victoria Hospital. On receiving the news, the Medical Staff were quick to point out "the interdependence between the practical work of the hospitals and the scientific study of the phenomena and causes of ill-health". They "considered that such a step would be in the interests of the diagnosis and treatment of disease, the training of medical students and the advance of scientific knowledge". Consequently, the Medical Staff urged the Board of Management to facilitate the project by providing the university with the necessary site. The Institute was built in close proximity to the hospital, to which it was connected by an overhead bridge. It originally consisted of three floors of 3,600 sq ft each, the top floor of which housed the clinical pathology departments of bacteriology, biochemistry, haematology, and morbid anatomy or histopathology.

When the Institute of Pathology was formally opened in 1933, Sir Thomas Houston and Dr J A Smyth and their staffs vacated their cramped quarters in the King Edward Building and moved into what was to them, at that time, most generous accommodation. The year 1933 saw another significant event when Mr Albert Lamont joined the technical staff of the laboratory. He was plucked out of school by Sir Thomas, who informed the Medical Staff that he wished to have permission to appoint an additional laboratory apprentice as the new department would need extra assistance and that he had "got a suitable boy and wished to commence his training". This boy, therefore, commenced his training and became, eventually, the Chief Technician in Haematology, was awarded the MBE in 1983 and retired from 51 years of continuous service in one hospital in 1984.

In 1930 Dr Houston had been appointed Lecturer in Toxicology, a post which he retained for 26 years. Although not an inspiring lecturer, he retained the interest and respect of medical undergraduates. When Professor W W D Thomson was on sick leave, he was appointed by the Medical Faculty as temporary, acting, Professor of Medicine. A longstanding member of the Senate, he was in 1941 elected as a Pro Chancellor of the University, an appointment which he greatly cherished.

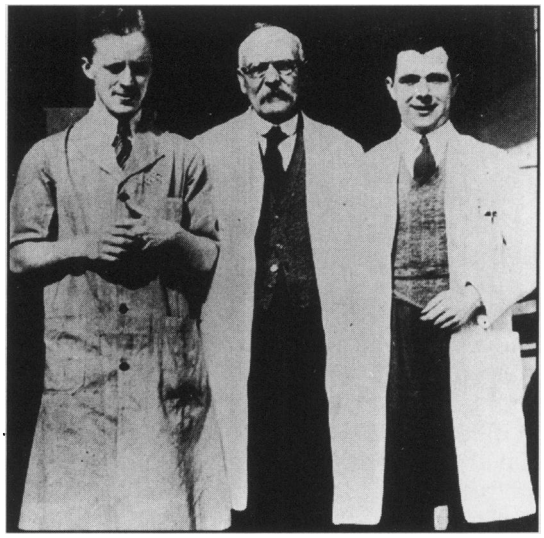

Fig. 5 The three original members of the technical staff. Mr A Lamont, Mr D Willix, Mr F Burns.

Sir Thomas reminded the Medical Staff in 1933 of his imminent retirement, which was due the following year. Various proposals were considered but not agreed by the time of the actual date. The temporary solution reached was to appoint Dr N C Graham "as Clinical Pathologist to the

Hospital on a salary of £350 per annum", while he remained the Lecturer in Bacteriology to the University. At the same time, Sir Thomas was granted permission to continue to use the laboratory and to be responsible for the vaccine therapy clinic.

Although a bachelor, a workaholic and somewhat of a recluse, he had a wide circle of friends to whom he was known as 'Tommy'. He was a busy man, spending much time at the hospital and in his outpatient vaccine therapy clinic, but more especially in the laboratory, providing a service in bacteriology and haematology, as well as an almost one-man blood transfusion service. Meanwhile he carried out research and published papers in the medical literature. His hospital appointment was still honorary, and he was engaged in private consultant practice. He seems to have had few hobbies, but could be seen on warm summer afternoons, bedecked in whites, playing tennis on the courts behind the Royal. He died in June 1960, 50 years after he was first appointed as house surgeon to the Royal. The clinical pathology laboratory and the services they provide today are his best and most permanent memorial .

After his death a junior colleague paid the following tribute: "Above all his achievements shone the simple, kindly, human qualities which won for him the unique position which he held in the School. It is for these qualities that 'Tommy' Houston, humblest and most unassuming of men, will be affectionately remembered by the many generations of students who knew him".

Sir Thomas Houston should be especially remembered as the father figure of the clinical pathological services currently provided at the Royal Group of Hospitals. The National Health Service eventually provided the essential financial resources to expand and allowed the creation of three separate divisions, bacteriology, biochemistry and haematology, each with its own consultant clinical pathologist in charge, whilst the histopathological service continued to be provided by The Queen's University Professor of Pathology.

The rapidly rising workload, combined with a widening range of laboratory techniques, necessitated the replacement of the old rubber teat and capillary glass tube methods and the introduction of computer-linked automated laboratory equipment. This required greatly increased laboratory space in order to house the equipment and the staff needed to operate it. At the same time there was a gradual and much greater clinical involvement by the consultant clinical pathologists, especially in haematology. In order to deal with patients suffering from haematological disorders, a day clinic was established to provide diagnostic and therapeutic facilities. Concomitantly, the Northern Ireland Haemophilia Centre was created and beds were available in both the Royal Victoria Hospital for the treatment of adults and in the Royal Belfast Hospital for Sick Children for the treatment of children suffering from leukaemia.

The point had now been reached when the four separate clinical pathology services each had its own laboratory, staff and head of department – a far cry from the small, cold room in the old Royal where a limited service was provided on a part-time basis by the head of the University Department of Pathology. The separation of the hospital laboratory disciplines had finally taken place on the foundations laid down by Sir Thomas Houston.

ACKNOWLEDGEMENTS

The idea of writing a history of the clinical pathology laboratory at the Royal Victoria Hospital was suggested to me by the Hospital Archivist, Dr J S Logan. This offered an opportunity of recording the contribution made by Sir Thomas Houston in developing clinical pathology and the service it provided to the Royal Victoria and associated hospitals.

This historical record would not have been possible were it not for the enormous help offered by Mrs Henry, secretary to the Northern Ireland Leukaemia Research Fund in the Department of Haematology, and from Mr Roy Creighton who was responsible for reproducing most of the illustrations.

REFERENCES

1. Houston T, Rankin J C. A note on the opsonic power of the serum with reference to the meningococcus of cerebrospinal fever occurring in the Belfast epidemic. *Lancet* 1907; i: 1213-5.
2. Irwin S T, Houston T. On a "typhoid carrier", treated successfully by the inoculation of typhoid vaccine. *Lancet* 1909; i: 311-3.
3. Houston T, McCloy J M. The relation of the enterococcus to "trench fever" and allied conditions. *Lancet* 1916; ii: 632-7.

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Case Report

Parosteal lipoma

R I Davis, H B Murtagh

Accepted 16 August 1994

Lipomas are very common benign tumours which are usually located in subcutaneous areas. Intra-osseous and parosteal lipomas are uncommon, the latter accounting for less than 0.3% of all lipomas[1]. This case of parosteal lipoma was originally diagnosed clinically and radiologically as an osteochondroma.

CASE REPORT

A 49 year-old female presented with a mass the size of a large grapefruit in the right gleno-humeral region. She described a constant ache from the lesion which had been present for about three months. There was some limitation of movement at the shoulder joint. There was no evidence of inflammation or lymphadenopathy. X-ray showed a large low density soft tissue mass in keeping with a lipomatous lesion. There was also a central focus of ossification with an unusual branching pattern resembling a candelabrum, which arose from the humeral cortex (Fig. 1). Spiral tomography confirmed the presence of a well defined fatty lesion containing an area of ossification and also showed that this mass abutted the lateral aspect of the uppermost part of the humeral diaphysis (Fig. 2). At excision biopsy it was thought the mass was an osteochondroma with an overlying lipoma.

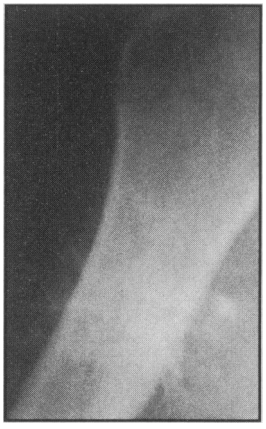

Fig 1. Parosteal lipoma arising from diametaphyseal area of humerus.

Fig 2. Tomograms showing fatty mass with ossification.

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The tumour was polypoid, weighed 205 grams and measured 10 X 8 X 6 cm. On sectioning it consisted of lobulated soft yellow fatty tissue. There were no mucoid areas and there was no haemorrhage or necrosis. Bone and calcified tissue were present at the base of the lesion. Histology showed mostly mature adipose tissue with a sparse fibrovascular stroma consistent with a benign lipoma. Lipoblasts were not identified. Within the fatty tissue there were thin trabeculae of cancellous bone (Fig. 3). At the base of the lesion there were several fragments of unremarkable cortical bone surrounded by fatty tissue. There was a partial covering of thickened periosteum and fibrocartilage on the surface of some of these fragments of cortical bone.

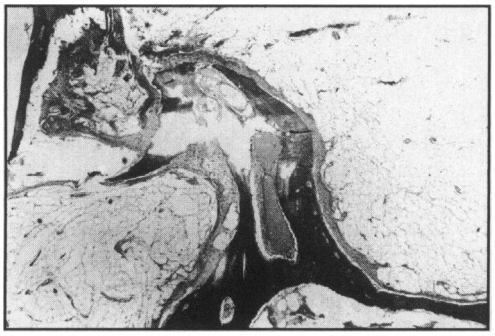

Fig 3. Parosteal lipoma consisting of fat with a central spicule of cancellous bone. H & E x 24.

DISCUSSION

Parosteal lipomas are rare. "Parosteal" refers to the outer layer of the periosteum and is thought to be more suitable than periosteal since it indicates contiguity with bone rather than implying a definite origin from the periosteum. Although over 100 cases have been reported in the literature many lack full documentation[2]. Patients are usually middle-aged and the lesions are most common in the peripheries, especially in the radius and femur[3]. The tumour characteristically consists of a lipomatous mass contiguous with the cortical bone, as in this case. There was also a radiating component of cancellous bone within the fat, intimately associated with the bony cortex at the site of attachment; an osseous component within the fat is not a required or consistent feature of parosteal lipomas[4].

The main radiological differential diagnosis is an osteochondroma but pathological examination clearly showed a lipomatous lesion with no evidence of a cartilage capped exostosis. The histological differential diagnosis includes a lipoma with ossification and a liposarcoma. Although metaplastic ossification is not uncommon in lipomata, the intimate localisation of the lesion to the cortical bone permits a diagnosis of parosteal lipoma. A liposarcoma is excluded by the absence of well defined lipoblasts.

Osteochondromas are the most common benign tumours of bone and radiologically can be confused with parosteal lipomas. This differential diagnosis may be helpful to consider in cases submitted clinically as "osteochondroma".

We thank Mr J R Nixon, FRCS, for permission to report this case.

REFERENCES

1. Fleming R J, Alpert M, Garcia A. Parosteal lipoma. *Amer J Roemtgenol* 1962; **87**; 1075-84.
2. Miller M D, Ragsdale B D, Sweet D E. Parosteal lipoma: a new perspective. *Pathology* 1992; **24**; 132-9.
3. Schweitzer G. Parosteal lipoma of the radius: a case report. *S Afr Med J* 1970; **44**; 648-9.
4. Moon N, Marmor L. Parosteal lipoma of the proximal part of the radius. *J Bone Jt Surg* 1964; **46A**; 608-14.

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Case Report

Ruptured silicone breast implant: a misleading chest X-ray

A C Dick, G T Deans, L Johnston, R A J Spence

Accepted 25 July 1994

The diagnosis of a ruptured breast implant can cause considerable confusion. We report a case of a ruptured breast prosthesis mimicking a primary lung tumour, to illustrate one of the diagnostic pitfalls that can occur in patients with silicone implants.

CASE REPORT

A 55 year old female was referred for investigation and possible surgery of a thyroid swelling. She had smoked 30 cigarettes per day for many years. Past medical history consisted of insertion of bilateral silicone breast implants 10 years previously. Clinical examination suggested a multinodular goitre and identified slight thickening superior to the left breast implant. Investigations revealed normal blood tests, and a multinodular goitre was confirmed on ultrasound scan. Routine chest X-ray (Fig. 1) identified an opacity in the left upper lobe showing features suggestive of a primary lung tumour. However, a lateral view failed to detect an abnormality in the thoracic cavity. CT scanning, performed with a view to percutaneous biopsy, revealed that the "lung tumour" was in fact related to the silicone implant (Fig. 2). Subsequent surgery confirmed rupture of the left breast prosthesis.

DISCUSSION

Routine chest radiographs may occasionally suggest the presence of a lung tumour which is found to be absent on subsequent investigations. Such pseudo-tumours can occur with skin lesions, subpleural silicotic nodules,[1] diaphragmatic hernia [2] or ECG leads [3]. Identification is important to prevent unnecessary intervention.

There has recently been debate in the medical [4] and lay press regarding potential complications of augmentation mammoplasty with silicone gel breast implants. Previous radiographic reports have been concerned with the detection

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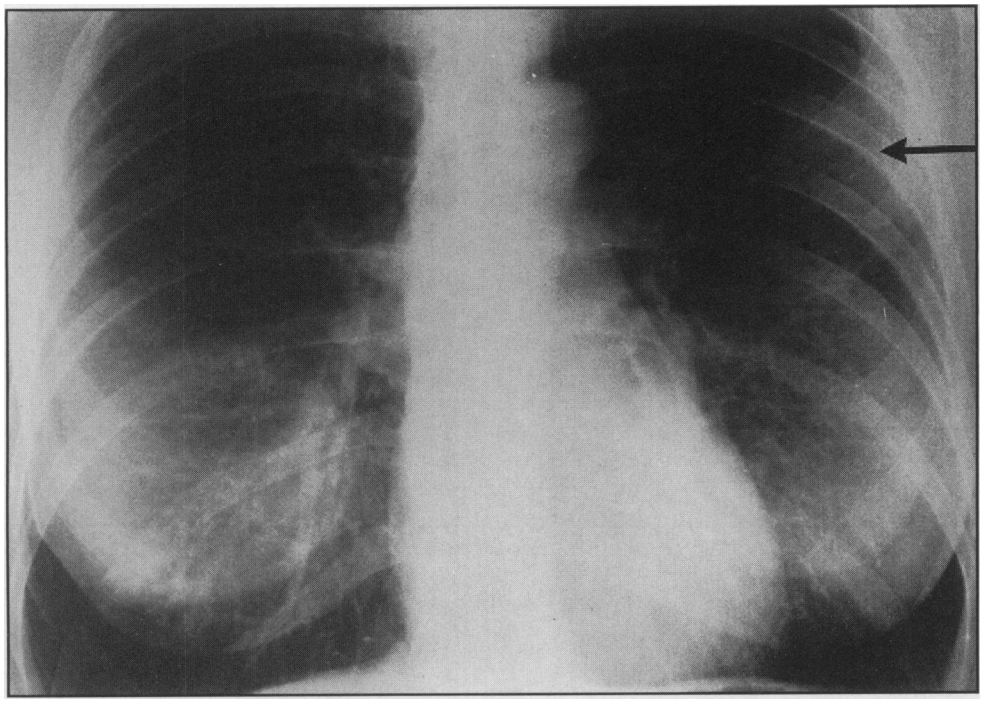

Fig. 1 Chest X-ray showing an opacity in the left upper lobe initially suggestive of a primary lung tumour.

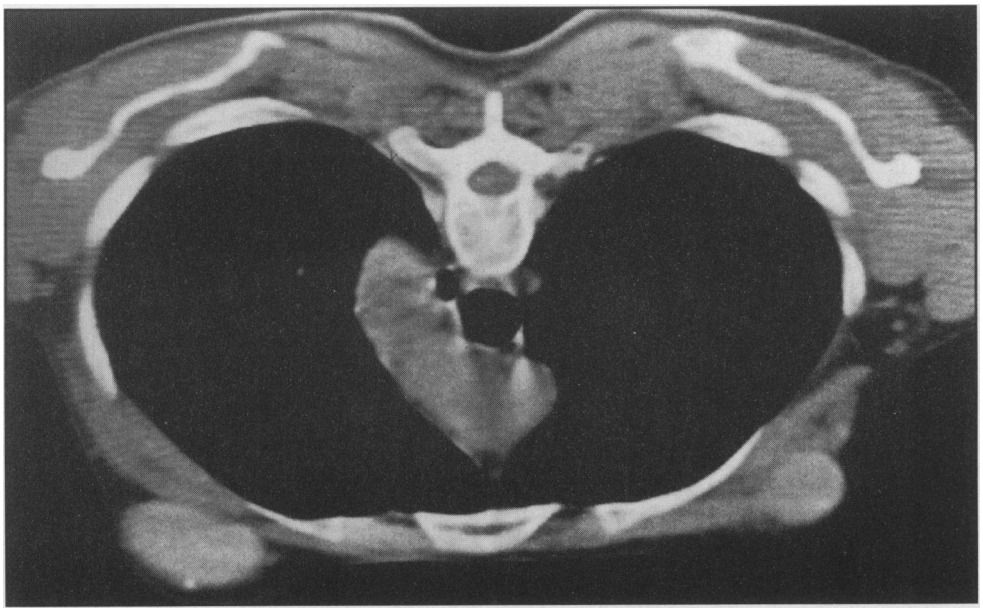

Fig. 2 CT scan of chest showing rupture of the left breast implant.

of malignancy [5] and rupture [6] following insertion of such implants. Silicone implant rupture is however notoriously difficult to diagnose. Clinical and mammographic examinations are reliable only if there has been gel migration away from the implant pocket, when nodules, asymmetry, decreased breast size, tenderness and a softer texture may be present[7]. In such circumstances, mammography is reportedly 90% accurate in diagnosing rupture[7]. The ability of ultrasound mammography to distinguish between silicone gel, muscle, haematoma and fluid collection makes it valuable in the diagnosis of some implant complications[8], and magnetic resonance imaging is attractive for evaluating rupture in younger patients in whom breast irradiation should be minimised [9].

In this case the chest radiographic appearances were characteristic of a primary lung tumour and thoracotomy was planned. It was only during work-up for this that the CT scan revealed the abnormality to be rupture of the silicone implant. The opposite situation, in which a primary lung tumour only became recognised on chest radiograph after removal of silicone breast implants has recently been reported[10]. This case highlights the diagnostic difficulties inherent in patients with silicone implants, and emphasises the extreme caution necessary in interpreting chest radiographs in such cases.

REFERENCES

1. Sugio K, Kaneko S, Yokoyama H, Ishida T, Sugimachi K. Subpleural silicotic nodule radiographically resembling a small bronchial carcinoma. *Respiration* 1991; **58**: 219-22.
2. van der Veen A H, van Geel A N, Slingerland R. An unusual coin lesion in the lung. *Neth J Surg* 1991; **43**: 261-62.
3. Kim T, Messersmith R N, MacMahon H. Pulmonary nodule mimicked by ECG lead artifact. *Chest* 1989; **95**: 237-8.
4. Berkel H, Birdsell D C, Jenkins H. Breast augmentation: a risk factor for breast cancer?. *N Eng J Med* 1992; **326**: 1649-53.
5. Leibmann A J, Kruse B. Breast cancer: mammographic and sonographic findings after augmentation mammoplasty. *Radiology* 1990; **174**: 195-8.
6. Levine R A, Collins T L. Definitive diagnosis of breast implant rupture by ultrasonography. *Plast Reconst Surg* 1991; **87**: 1126-8.
7. Andersen B, Hawtof D, Alani H, Kapetansky D. The diagnosis of ruptured breast implants. *Plast Reconst Surg* 1989; **84**: 903-7.
8. van Wingerden J J, van Staden M M. Ultrasound mammography in prosthesis related breast augmentation complications. *Ann Plast Surg* 1989; **22:** 32-5.
9. Brem R F, Tempany C M, Zerhouni E A. MR detection of breast implant rupture. *J Comput Assist Tomogr* 1992; **16**: 157-9.
10. Fitter A C, Cowie J C. Minerva. *Br Med J* 1992; **305**: 842.

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Case Report

Adenocarcinoma of the distal duodenum: two cases managed by pylorus preserving pancreatico-duodenectomy and adjuvant chemotherapy

L J Fon, G T Deans, D Gilroy, T Diamond

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Primary adenocarcinoma of the duodenum is a rare tumour which accounts for less than 0.5% of intestinal malignancies [1, 2], and the optimum treatment is unknown. Previous surgical treatment has consisted of Whipple type pancreatico-duodenectomy or segmental resection [3, 4, 5], while chemotherapy has been confined to purely palliative cases[3,6,7]. In pancreatic diseases, pylorus preserving pancreatico-duodenectomy appears to offer advantages over the classical Whipple procedure [8, 9]. However, there are few reported cases employing this operation in duodenal carcinoma[10], and there are no reports of the efficacy of chemotherapy to improve prognosis following potentially curative surgery.

Case 1. A 64 year old male presented with symptoms and signs in keeping with gastric outlet obstruction. Barium meal revealed a stricture, suggestive of a tumour, in the third part of the duodenum (Figure). CT scan confirmed this and revealed no evidence of metastasis (Figure). Laparotomy confirmed a stenosing tumour of the third part of the duodenum. Intra-operative ultrasonography revealed no evidence of hepatic metastases. A pylorus preserving pancreatico-duodenectomy was performed and the patient made an uneventful postoperative recovery. Histological examination identified a primary duodenal adenocarcinoma with involvement of two local lymph nodes. Four courses of adjuvant chemotherapy, consisting of 5-fluorouracil, cisplatin, epirubicin and folinic acid were given, without significant complication. There was no evidence of ascites or recurrence on ultrasound scan at one year and he remains well 24 months following surgery.

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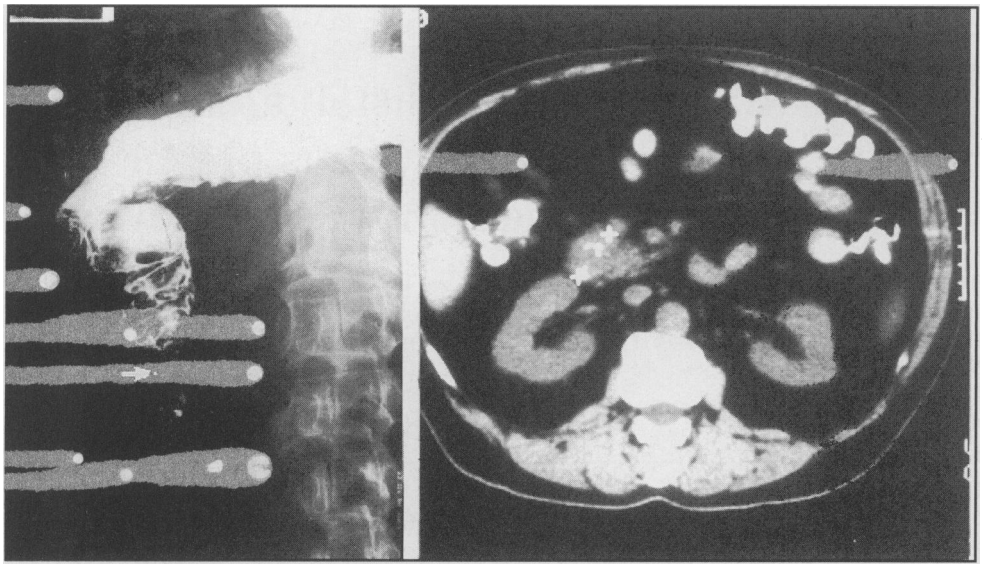

Fig. Barium meal and CT scan demonstrating a stricture in the third part of the duodenum (arrow) due to a 4 cm diameter solid lesion (seen between calipers).

Case 2. A 40 year old male presented with a two year history of epigastric pain, anorexia and weight loss, which had persisted despite long-term anti-ulcer medication. Endoscopy revealed an ulcerating lesion in the third part of the duodenum and biopsy identified this as an adenocarcinoma. CT scan confirmed the presence of a duodenal lesion but revealed no metastatic spread. Laparotomy confirmed a tumour of the third part of the duodenum with involvement of the transverse mesocolon. Intra-operative ultrasonography revealed no evidence of hepatic metastases. He underwent pylorus preserving pancreatico-duodenectomy and extended right hemicolectomy. Postoperatively he developed delayed gastric emptying which resolved spontaneously after one week. Histological examination revealed a primary duodenal adenocarcinoma with extension to the serosa but without lymph node involvement. He received four courses of chemotherapy (5-fluorouracil, cisplatin, epirubicin and folinic acid), without significant complication. He remains well 12 months following surgery.

DISCUSSION

Primary duodenal carcinoma is rare, accounting for 0.03 to 0.35% of all intestinal malignancies[1,2]. The proportion of lesions occurring in the 1st, 2nd, 3rd and 4th parts of the duodenum is 45%, 35%, 10% and 10%, respectively[11]. Tumours of the third part, are extremely uncommon[12]. Only 89 cases of infra-papillary duodenal carcinoma were cited in the literature from 1961-74[5,11], but duodenal carcinoma represents 25-48% of primary tumours of the small intestine[6,13]. The average age at presentation is sixty years, male to female ratio 3:1[1]. There is no racial predilection[14].

Symptoms associated with duodenal carcinoma are abdominal pain, weight loss (each present in 74% of cases), vomiting and anaemia (each present in 55% of patients)[15]. These symptoms are non-specific, the clinical picture often

mimicking peptic ulceration. However, as in our second case, there is failure to respond to ulcer healing medication. This emphasises the importance of considering rarer diagnoses, such as duodenal carcinoma, in cases where symptoms persist despite standard upper gastrointestinal investigation and anti-ulcer treatment. Late presentations include biliary obstruction with jaundice or an abdominal mass[6, 16]. The absence of specific symptoms and signs, coupled with the rarity of the condition, means that diagnosis is frequently delayed. The average time to correct diagnosis from initial consultation is between 7-10 months[6, 17] – in one of our cases, symptoms were present for two years before a definitive diagnosis was made. Consequently, a high index of suspicion is essential if the condition is to be recognised early.

Even with early referral, investigations may fail to make the diagnosis, thus making recognition difficult. Barium studies may not detect small tumours in the third part of the duodenum, since special posturing is necessary to visualise this region[11]. The accuracy of barium studies in this area is, therefore, 75% or less[4]. Barium studies are particularly valuable in lesions of the fourth part of the duodenum, which are often not reached by endoscopy. In general, however, duodenoscopy is preferable to barium studies, as it allows both direct visualisation and biopsy of the lesion. Failure to visualise the distal part of the duodenum limits the reported sensitivity to 89%[7], thus emphasising the importance of distal duodenoscopy during routine upper gastrointestinal endoscopy. We found computerised tomography and intraoperative ultrasonography useful in assessing local or distant spread, and therefore of benefit in determining the potential for curative resection of the tumour [18].

The preferred surgical procedure for treatment of distal duodenal carcinoma remains to be determined. Surgical options include palliative bypass, pancreatico-duodenectomy and local segmental resection. Although one series reported a significantly different survival at one year between pancreatico-duodenectomy and segmental resection (62% versus 45%, respectively), this was not maintained at five years (25% versus 20%)[5]. The low number of patients surviving five years probably explains this finding. Segmental resection is associated with a higher incidence of recurrence due to the limited resection of regional lymphatic and adjacent tissue [3]. The technique may still have a place in high risk patients or in patients with very small lesions, where satisfactory tumour clearance can be achieved with a tension free anastomosis. However, since regional spread is frequently encountered, radical surgery offers the best chance of cure. Pancreatico-duodenectomy is therefore recommended.[3, 4, 5] Admittedly there is patient selection, but the five year survival following pancreatico-duodenectomy is 40-45%, compared to the overall five year survival for duodenal carcinoma of 5-17% [3, 5, 6, 19, 20]. In pancreatic carcinoma, pylorus preserving pancreatico-duodenectomy is claimed to be easier, less time consuming and associated with less blood loss, a shorter hospital stay and better weight gain than the classical Whipple procedure[9]. In addition, the incidence of dumping, diarrhoea and enterogastric reflux are lower [20]. Claims that this operation is complicated by transient delayed gastric emptying and marginal ulceration more frequently than in the Whipple procedure have not been substantiated [21, 22]. Isotope studies have shown that delayed gastric emptying is common in both procedures, there being no significant difference in emptying of both liquids and solids between both techniques[22]. In one of our

cases, a delay in gastric emptying occurred but this was transient and resolved within one week. The advantages of pylorus preservation therefore, appear to outweigh the disadvantages and it seems logical to apply this technique to distal duodenal carcinomas.

Experience with adjuvant chemotherapy in duodenal carcinoma is also limited. Previous reports have restricted chemotherapy to purely palliative cases. Results in these poor prognosis patients have been promising, with a significant improvement in mean survival [3,4,6,7,11,3]. It is also suggested that postoperative palliative treatment with combined chemo-radiotherapy is associated with a better survival than that obtained by surgery alone[11,13]. Chemotherapy should, therefore, prove beneficial in better prognosis cases. In our limited experience, the combination of pylorus preserving pancreatico-duodenectomy and adjuvant chemotherapy is well tolerated. Combining adjuvant chemotherapy with pylorus preserving pancreatico-duodenectomy should, theoretically, provide the maximal chance of cure without significant increase in side effects.

Survival in duodenal carcinoma is related to the extent of tumour spread. The lack of specific symptoms results in two thirds of patients presenting with metastases, 32% having paraduodenal and 33-67% regional nodal involvement [5,17,23]. One of our cases had local nodal involvement. Median survival for cases without metastases and those with paraduodenal or regional nodal involvement is 42, 16 and 6 months, respectively [23]. The overall survival is generally poor, being 67% at one year and 5-17% at five years [5,6]. Even after potentially curative resection, five year survival rarely exceeds 15-25 %[6]. Infra-papillary tumours are thought to be associated with a better prognosis than supra-papillary lesions, the mean survival being 31-46 months compared to 27- 30 months, respectively [6,12].

We believe that combined modality treatment has a definitive role in the treatment of potentially curative cases of distal duodenal carcinoma. The rarity of the condition means that, even with longer follow-up, no single unit will see sufficient cases to draw conclusions or develop formalised guidelines on treatment. A prospective multicentre trial of this condition is required.

REFERENCES

1. Eger S A. Primary malignant disease of the duodenum. *Arch Surg* 1933; **27**: 1087-1108.
2. Iovine V M, TsangarisN. Primary carcinoma of the duodenum. *Am Surg* 1961, **27**: 744-50.
3. Cortese A F, Cornell G N. Carcinoma of the duodenum. *Cancer* 1972; **29**: 1010-15.
4. Moss W M, McCart P M, Juler G, Miller D R. Primary adenocarcinoma of the duodenum. *Arch Surg* 1974; **108**: 805-7.
5. Spira I A, Ghazi A, Wolff W I. Primary, adenocarcinoma of the duodenum. *Cancer 1977*; **39**: 1721-6.
6. Alwmark A, Andersson A, Lasson A. Primary carcinoma of the duodenum. *Ann Surg* 1980; **191**: 13-18.
7. Ouriel K, Adams J. Adenocarcinoma of the small intestine. *Am J Surg* 1984; **147**: 66-71.
8. Pitt H A, Grace P A. Pylorus-preserving resection of the pancreas. *Bailieres Clin Gastroenterol* 1990; **4**: 917-30.

9. Klinkenbijl J H, van der Schelling G P, Hop W C, van Pel R, Bruining H A, Jeekel J. The advantages of pylorus-preserving pancreatoduodenectomy in malignant disease of the pancreas and periampullary region. *Ann Surg* 1992; **216**: 142-5.

10. Van Ooijen B, Kalsbeek H L. Carcinoma of the duodenum. *Surg Gynecol Obstet* 1988; **166**: 343-7.

11. Sakker S, Ware C C. Carcinoma of the duodenum: comparison of surgery, radiotherapy and chemotherapy. *Br J Surg* 1973; **60**: 867-72.

12. Lowell J A, Rossi R L, Munson J L, Braasch J W. Primary adenocarcinoma of third and fourth portions of duodenum. *Arch Surg* 1992; **127**: 557-60.

13. Lillemoe K, Imbembo A L. Malignant neoplasms of the duodenum. **Surg Gynecol Obstet** 1980; **150**: 822-6.

14. Spinazzola A J, Gillesby W J. Primary malignant neoplasms of the duodenum: report of twelve cases . *Am Surg* 1963; **29**: 405-12.

15. Burgerman A, Baggenstoss A H, Cain J C. Primary malignant neoplasms of the duodenum excluding the papilla of Vater: a clinicopathological study of 31 cases. *Gastroenterology* 1956; **30**: 421-31.

16. Joesting D R, Beart Jr R W, van Heerden J A, Weiland L H. Improving survival in adenocarcinoma of the duodenum. *Am J Surg* 1981; **141**: 228-31.

17. Kenefick J S. Carcinoma of the duodenum. *Br J Surg* 1972; **59**: 50-5.

18. Kazerooni E A, Quint L E, Francis I R. Duodenal neoplasms: predictive value of CT for determining malignancy and tumour resectability. *Am J Roentgenol* 1992; **159**: 303-9.

19. Crane J M, Gobbel W G, Scott H W. Surgical experience with malignant tumours of the ampulla of Vater and duodenum. *Surg Gynecol Obstet* 1973; **137**: 937-40.

20. Rotman N, Pezet D, Fagniez P L, Cherqui D, Celicut B, Lointier P. Adenocarcinoma of the duodenum: factors influencing survival. *Br J Surg* 1994; **81**: 83-5.

21. Grace P A, Pitt H A, Longmire WP. Pylorus preserving pancreato-duodenectomy an overview. *Br J Surg* 1990; 77: 968-74.

22. Williamson R C, Bliouras N, Cooper M J, Davies E R. Gastric emptying and enterogastric reflux after conservative and conventional pancreatico-duodenectomy. *Surgery* 1993; **114**: 82-6.

23. Lai E C S, Doty J E, Irving C, Tompkins R K. Primary adenocarcinoma of the duodenum: analysis of survival. *Wld J Surg* 1988; **12**: 695-9.

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Case Report

Hypercalcemia associated with a parathyroid cyst

K J Cooper, A D K Hill, Y Sangwan, M Al-Adnani, N Menzies-Gow, A Darzi

Accepted 19 July 1994

Parathyroid cysts are rare. When they do occur they usually have no demonstrable endocrine function, but may cause respiratory distress by tracheal compression or may be mistakenly diagnosed as thyroid carcinomas or adenomas. We report a case of a parathyroid cyst which presented with hypercalcemia.

CASE REPORT:

A fifty eight year old man undergoing routine investigations was found to have hypercalcaemia. His calcium level was elevated to 3.63 mmol/l and the serum parathyroid hormone level was 18.4 pmol/l (normal range 1.0 - 5.3). Serum alkaline phosphatase was at the upper limit of normal at 113U/l. X-ray of the renal tract revealed no evidence of calcification. He was asymptomatic and had no palpable neck mass. Ultrasound examination revealed a 3 cm cyst-like structure inferior and posterior to the left lobe of the thyroid, with a well defined wall approximately 2 mm thick. Exploration of the neck revealed a large cyst (6.5 x 5 x 3 cm), which was removed and found to weigh 57 g. Three other parathyroid glands with a normal appearance were found. Macroscopic examination of the removed specimen showed a smooth lined cyst containing greasy, pearly material. Histology revealed a fibrous lined cyst, containing several islands of parathyroid tissue with clear cell cytology and moderate nuclear pleomorphism. Post-operatively serum calcium returned to normal. The patient was discharged on the fourth post-operative day.

DISCUSSION

Many theories have been advanced regarding the aetiology of parathyroid cysts. Goris[1] was the first to report a case. Nylander[2] has suggested that they arise from remnants of the third or fourth pharangeal pouch or are vestigial

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pharyngobronchial ducts, while Forsyth[3] proposed that they are formed by retained secretion of the parathyroid cells. It may be incorrect to label a structure which possibly arises from a pharangeal pouch remnant and secondarily incorporates parathyroid tissue within its wall as a parathyroid cyst. Welti[4] and Black[5] have reported the occurrence of cysts in the region of the parathyroid which resemble parathyroid cysts yet have no demonstrable parathyroid tissue.

Parathyroid cysts are most often misdiagnosed as nodular goitre, solitary adenoma of the thyroid, or as a thyroid carcinoma. Pre-operative diagnosis can be successful if the cyst is accessible to ultrasound guided fine needle aspiration. The fluid aspirated contains high levels of parathyroid hormone [6,7]. Parathyroid cysts are difficult to differentiate from thyroid cysts using ultrasound[8], but at the time of operation the distinction is easier. Mohr *et al* have suggested that pre-operative localisation of enlarged parathyroid glands facilitates the decision for surgical intervention and reduces the duration of the operation[9].

The gross characteristics are of a translucent, thin white walled cyst filled with clear waterlike fluid, in contrast to the brown liquid of thyroid cysts. They are lined by cuboidal or columnar epithelial cells and some parathyroid tissue is contained in the wall. Parathyroid cysts usually arise from the inferior glands, lateral to the thyroid and are easily dissected from it. Occasionally they have been found in the mediastinum[10].

Functional activity in these cysts is rare, but has been described[10]. It has been suggested that a functioning parathyroid cyst may actually be an adenoma with cystic degeneration[11]; the cellular pattern in the present case revealed no evidence of a parathyroid adenoma.

REFERENCES

1. Goris D . Exstirpation von drie cystischen Nebenschilddrusenlappen. Exstirpation de trois lobules parathyroidiens. *Abstract; Int Zentral Laryngol Rhinol Wissensch* 1906; **22**:190.
2. Nylander P E A. Über parathyreoideale Halszysten. *Acta chir scand* 1929; **64**: 539-47.
3. Forsyth D. The parathyroid glands. II . Their pathology in man. *Ouart J Med* 1908; **1**: 287-311.
4. Welti H. A propos des kystes parathyroidiens. *Mem Acad de chir* 1946; **72**: 33.
5. Black B M, Watts C F. Cysts of parathyroid origin: report of two cases and study of incidence and pathogenesis of cysts in parathyroid glands. *Surgery* 1949; **25**: 941-9.
6. Prinz R A, Peters J R, Kane J M, Wood J. Needle aspiration of nonfunctioning parathyroid cysts. *Am Surg* 1990; **56**: 420-22.
7. Petri N, Holten I. Parathyroid cyst: report of case in the mediastinum. *J Laryngol Otol* 1990; **104**: 56-7.
8. Graif M, Itzchak Y, Strauss S, Dolev E, Mohr R, Wolfstein I. Parathyroid sonography: diagnostic accuracy related to shape, location and texture of the gland. *Br J Radiol* 1987; **60**: 439-43.
9. Mohr R, Graif M, Itzchak Y, Drenzick Z, Dolev F, Charuzy I, Wolfstein I. Parathyroid localisation. Sonographic-surgical correlation. *Am Surg* 1985; **51** (5): 286-90.
10. Linos DA, Schoretsanitis G, Carvounis E. Parathyroid cysts of the neck and mediastinum. *Acta Chir Scand* 1989;**155**: 211-6.
11. Rogers L A, Fetter B F, Peete W P J. Parathyroid cyst and cystic degeneration of a parathyroid adenoma. *Arch Pathol* 1969; **88**: 476-9.

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Case Report:

Hereditary coproporphyria

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Hereditary coproporphyria, a form of acute hepatic porphyria, is a rare Mendelian autosomal dominant inherited condition with incomplete penetrance. The acute attack is usually followed by complete remission, but death may occur. Latent cases are recognised, and the characteristic neurovisceral symptoms and signs are shared by many other conditions. Acute porphyria should be considered in the differential diagnosis of any patient presenting with abdominal pain and neuropsychiatric upset.

CASE REPORT. A 21 year old female presented with a one week history of "'flu-like" illness, characterised by dry cough, generalized weakness and sore throat. Five days prior to her admission she had developed crampy central abdominal pain, which radiated to both loins. Trimethoprim was prescribed for a possible urinary tract infection. Her symptoms did not improve, and she was referred to her local hospital for investigation.

Examination revealed generalised abdominal tenderness, but no palpable mass. Bowel sounds were normal. She was apyrexic; blood pressure 140/110 mmHg. Haemoglobin concentration was 14.8 g/dl with normal indices, white cells 12,000 x 10^9/l. A routine biochemical profile showed values within the reference limits for sodium, potassium, urea, bilirubin and hepatic transaminases. Serum calcium was 2.46 mmol/l, glucose 8.3 mmol/l. Routine Stix testing of urine was positive for protein and blood. Urine culture for micro-organisms was negative. Ultrasound scan of the abdomen showed right-sided renal agenesis with hypertrophy of the left kidney. Gastroscopy was reported as normal.

Two days after admission she was found unconscious on the ward. Her blood pressure was 160/110 mmHg, and she had sinus tachycardia of 100 beats per minute. Fundoscopy revealed retinal artery spasm, but no haemorrhages or exudates. There were no lateralising signs. She was apyrexic and there was no neck stiffness. Over the next few hours blood pressure rose to 180/130 mmHg and pulse rate to 150 beats per minute. She then had a generalised tonic-clonic seizure lasting three minutes, which resolved spontaneously. Intravenous labetalol was commenced. Three further generalised seizures occurred over the next ten hours. The patient was then referred to our unit at the Royal Victoria Hospital.

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CT scan of the brain showed no focal lesion. A 24 hour urine collection for catecholamines was commenced and the specimen was noted to turn a dark red colour on standing. Catecholamille levels were later reported as normal. Urine tests for porphyrins revealed d-aminolaevulinic acid 209 μmol/l (normal=0.8-34.3) and porphobilinogen 316 μmol/l (normal < 8.8). An initial diagnosis of acute hepatic porphyria was made.

She was managed conservatively with a 10% dextrose infusion and oral glucose solutions.[1] Blood pressure fell to 130/100 mmHg on the labetalol infusion, which was discontinued when she became normotensive. The second day after transfer she complained of feeling weak. Physical examination showed proximal muscle power symmetrically reduced to Grade 4 in upper and lower limbs. Deep tendon reflexes were diminished. Serum sodium was 112 mmol/l, and plasma osmolality 236 mOsm/kg, with urinary sodium 27 mmol/l, and urine osmolality 542 mOsm/kg. These findings suggested the syndrome of inappropriate ADH secretion. Fluid restriction to 1000 ml per 24 hours returned the serum sodium level to normal within five days. Recovery over the next week included one episode of syncope, but was otherwise uneventful. She was discharged after sixteen days in hospital.

Subsequent results showed an excess of both coproporphyrin and protoporphyrin in a faecal sample, which confirmed a diagnosis of hereditary coproporphyria. Her parents and three younger brothers are currently being investigated in order to detect any latent cases of porphyria.

DISCUSSION

The porphyrias are a heterogeneous group of inborn errors of metabolism caused by enzyme defects in the biosynthetic pathway of haem. The acute clinical picture occurs secondary to the over-production of porphyrin precursors. These disorders are classified into acute and non-acute forms.

The acute porphyrias include acute intermittent porphyria, variegate porphyria, hereditary coproporphyria and plumboporphyria. All are inherited in an autosomal dominant fashion with the exception of plumboporphyria, which is autosomal recessive. Acute attacks may be precipitated by infection, alcohol, endogenous hormones and a variety of commonly used drugs, particularly those which are inducers of hepatic microsomal enzymes.[2,3] In plumboporphyria, heterozygotes are liable to illness after moderate exposure to lead.[7] Excretion of porphyrins in the urine is markedly increased in the acute phase, and can remain so for months afterwards. Faecal analysis is required to classify the biochemical subtype.

Hereditary coproporphyria was first described in 1955 by Berqer and Goldberg.[4] There is reduced coproporphyrinogen oxidase activity. The clinical manifestations are accompanied by the excretion of d-aminolaevulinate and porphobilinogen in urine and coproporphyrinogen III in faeces and urine. Genetic studies have shown the defect to be located on chromosome 9. A recent paper[5] reports an amino-acid substitution (Arg to Trp) resulting from a single base change, in a patient homozygous for hereditary coproporphyria. This is the first mutation that has been found in this condition. Similar methodology may be used in the future to determine the spectrum of mutations responsible for hereditary coproporphyria and could be used for diagnostic purposes.

The acute attack is managed with supportive therapy and a high carbohydrate intake. In severe cases infusions of haem arginate and tin protoporphyrin may be given. This induces a rapid biochemical remission.[6] They are thought to work by suppressing the rate-determining enzyme in haem biosynthesis as it repletes the hepatic pool of haem. During the recovery phase patient and family education are vital. It is necessary to supply the patient and their general practitioner with a list of drugs which must be avoided.[7] Essential too is the screening for latent cases amongst the relatives.

A large number of the classical clinical features found acutely with this disease were illustrated in this case. It is interesting that there was no evidence of any dermatological complaint. Approximately one-third of patients with hereditary coproporphyria will develop a photosensitive bullous skin eruption,[8] and these often have abnormal liver function tests, which were not found in this case. Our patient has nevertheless been counselled regarding possible problems on exposure to the sun; the use of a sun-block cream may be required.

We were unable to determine the precipitant leading to the development of this particular crisis. It is tentatively suggested that the preceding "'flu-like" illness or the initial trimethoprim therapy may have been responsible.

We acknowledge the assistance of the Department of Biochemistry at The University Hospital of Wales, Cardiff, with diagnostic faecal analysis, and are grateful to Dr L J E W Walker of the Mid-Ulster Hospital, Magherafelt, for referring the patient.

REFERENCES

1. Doss M, Sixel-Dietrich F, Verspohl F. "Glucose effect" and rate limiting function of uroporphyrinogen synthase on porphyrin metabolism in hepatocyte culture: relationship with human acute hepatic porphyrias. *J Clin Chem Clin Biochem* 1985; **23**: 505-13.
2. Moore M R, McColl K E L, Rimington C, Goldberg A. Disorders of porphyrin metabolism, 1977. New York: Plenum Press. 98-117.
3. Moore M R. Review: Biochemistry of porphyria. *Int J Biochem* 1993; **25**: 1353-68.
4. Goldberg A, Moore M R, McColl K E L, Brodie M J. Porphyrin metabolism and the porphyrias. In: Oxford Textbook of Medicine. Weatherall D J, Ledingham J G G, Warell D A. (eds). 2nd ed., 1987. Oxford University Press. 9: 136-9. 145.
5. Martasek P, Nordmann Y, Grandchamp B. Homozygous hereditary coproporphyria caused by an arginine to tryptophane substitution in coproporphyrinogen oxidase and common intragenic polymorphisms. *Human Mol Genet* 1994; **3**: 477-80
6. Dover S B, Moore M R, Fitzsimmons E J, Graham A, McColl K E L. Tin protoporphyrin prolongs the biochemical remission produced by heme arginate in acute hepatic porphyria. [See comments] *Gastroenterology* 1993; **105**: 500-6. Comments in: *Gastroenterology* **105**: 590-4.
7. Moore M R, McColl K E L. Porphyria – drug lists. University of Glasgow Porphyria Research Unit 1991.
8. Lip G Y H, McColl K E L, Moore M R. The acute porphyrias. *Br J Clin Pract* 1993; **47**: 38-43.

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Book review

To Cure and to Care. Memoirs of a Chief Medical Officer. James Deeny (pp 312). The Glendale Press. Dun Laoghaire. 1989.

Doctor James Deeny, whose autobiography this book is, died on 3rd April 1994, aged 87. A distinguished graduate of the Belfast Medical School, he was born into general practice in Lurgan, County Armagh, where his father practised, and where he was to practise himself for thirteen years. To his practice he brought extraordinary gifts of intellect, observation and heart. In the conditions of those times there were enormous opportunities in general practice for the study of disease, and Doctor Deeny threw himself into investigation and prevention. This reviewer remembers the sensation caused by his discovery of a family with inborn methaemoglobinaemia, and his successful treatment of them by ascorbic acid. His careful clinical method led to him finding cases of pellagra in Lurgan, and to diagnosing ratbite fever in a recurrently febrile "mountainy man". He cured the fever, he does not say how, but it must have been by organic arsenicals. It was fortunate for his development as a physician, that in those necessitous, straitened, malnourished and infected times he had the care and study of great numbers of sick people with all sorts of disorders. His publications did much to bring their unfortunate lot to notice.

In 1944 he became Chief Medical Adviser to the Department of Local Government and Public Health in Dublin. There followed eleven years of decisive administration of public health, and of reform and better provision of hospital care. These were years of great advances, and led to his appointment to the World Health Organisation. That took him the world over. This brief review cannot do justice to all Doctor Deeny's activity in Lurgan, Dublin, Geneva, Rome and the Asian and African world. Political opinions sometimes intrude in the book, sometimes painfully, but they are infrequent.

We recommend this book to all doctors. We offer our sympathy to Doctor Deeny's family, and congratulate them on his life, so well lived.

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