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REFERENCES

1. Wichterman K, Geha AS, Cahow CE, Baue AE. Giant paraesophageal hiatus hernia with intrathoracic stomach and colon: the case for early repair. *Surgery* 1979; **86**: 497-506.
2. Carter R, Brewer LA, Hinshaw DB. Acute gastric volvulus. *Am J Surg* 1980; **140**: 99-106.
3. Ball T, McCrory R, Smith JO, Clements JL. Traumatic diaphragmatic hernia: errors in diagnosis. *Am J Roentgenol* 1982; **138**: 633-7.
4. Welch FT, Reynolds SA. Diaphragmatic hernia simulating staphylococcal pneumonia. *Rocky Mountain Med J* 1972; **69**: 37-40.
5. Goldstein AI, Gazzaniga AB, Ackerman ES, Rajcher WJ, Kent DR, Campbell R. Strangulated diaphragmatic hernia in pregnancy presenting as an empyema. *J Reprod Med* 1972; **9**: 135-9.
6. Thind GS. Diaphragmatic hernia simulating pleural effusion and complicating puerperium. *Australas Radiol* 1977; **21**: 325-7.

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The Ulster Medical Journal, Volume 58, No. 1, April 1989.

THE ULSTER MEDICAL SOCIETY

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Training for general practice: meeting the challenge

A G McKnight

Presidential Address to the Ulster Medical Society, 27 October 1988

Many changes and advances have taken place in medicine from 1948, the beginning of the National Health Service, to the present time. One of the most noteworthy is in training for general practice and in particular how general practice has met this challenge.

In 1911 Sir William Osler in a letter to Ira Remsen, President of the Johns Hopkins University, said of the hospital specialist that 'he was cabined, cribbed, confined within the four walls of a hospital practising the fugitive and cloistered virtues of a clinical monk. How shall he, forsooth, train men for a race the dust and heat of which he knows nothing and — this is a possibility! — cares less? I cannot imagine anything more subversive to the highest ideal of a clinical school than to hand over young men who are to be our best practitioners to a group of teachers who, ex officio, are out of touch with the conditions under which these young men will live'.

The career preferences of house officers in Northern Ireland between 1982 and 1988 show that 40 – 50% or more had a first preference for general practice (Fig 1). This has been the case for many years during which the education of general practitioners, both as undergraduates and postgraduates, has been in the hands of colleagues who had little or no experience of general practice. In 1969 Sir Denis Hill said 'The family physician's role is a difficult one. If it is to be sustained and developed the general practitioner must become the best educated, the most comprehensively educated of all the doctors in the Health Service'.[1] It is the marriage of the views of Osler and Hill which forms the basis of this paper.

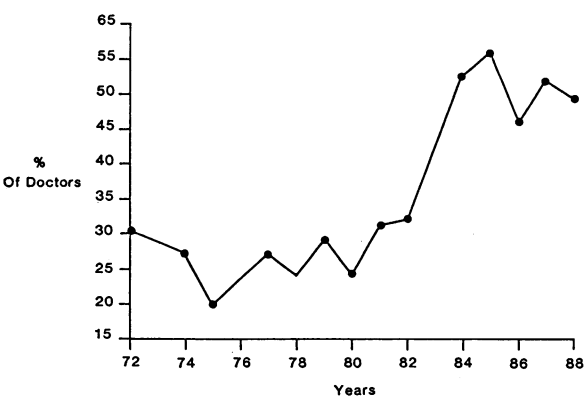

Fig 1. Career preferences of house officers in Northern Ireland 1972 – 1988, from data collected by the NI Council for Postgraduate Medical Education. The graph shows the proportion indicating general practice as first choice.

A G McKnight, MB, FRCGP, President, Ulster Medical Society, 1988 – 1989.

EARLY TRAINING ARRANGEMENTS

A history of training for general practice might begin in 1858 with the foundation of the General Medical Council. Its main purpose was to regulate the qualifications of practitioners in medicine and surgery. It was to keep a register so that persons requiring medical aid could distinguish qualified from unqualified practitioners. A Royal Commission in 1882 recommended that the qualifying examination should be in medicine, surgery and midwifery. The standard of proficiency should 'guarantee the possession of the knowledge and skill requisite for the efficient practice of medicine, surgery and midwifery' that is *a complete doctor* who could enter practice directly without further hospital experience after qualifying.

Sixty years later, the Goodenough Committee (1942 – 1944) recommended that after qualifying there should be one year of compulsory pre-registration hospital appointments in medicine and surgery.[2] This was introduced in 1953 and was the first step in the provision of postgraduate experience. It implied that undergraduate training no longer produced a fully trained doctor fit to enter general practice immediately after qualification as indeed did some of my contemporaries in 1950. (They had been preceded by many illustrious people, not least by Sir John Biggart, who was in general practice on the Shankill Road for five days between qualification at Queen's and graduation).

THE TRAINEE GENERAL PRACTITIONER SCHEME

The forerunner of vocational training for general practice was the Trainee General Practitioner Scheme which had been introduced in 1948 following the Spens Committee recommendations in 1946. The Committee recommended that the trainee should be a supernumerary member of the practice and spend one or preferably two years as an assistant after the completion of house appointments. Even those doctors who intended to become specialists would benefit from a year in general practice. Ten per cent of practitioners selected on the grounds of their success in practice and general suitability (whatever this might mean) should be encouraged to take a trainee. Trainees in the scheme were appointed by the trainers themselves and this has continued through to the present vocational training for general practice in Great Britain.

In 1957 there were 450 trainees in Great Britain in the scheme, but by 1966 the numbers had dropped to 150. The scheme became less and less attractive because of the ease in obtaining assistantships with a view to a partnership rather than because of faults in the scheme itself, though it was criticised on the grounds of exploitation of the trainee. This scheme was never introduced in Northern Ireland, which is one of the reasons for the present-day difference in the trainee entrance to vocational training in Northern Ireland compared with Great Britain. In 1950 the Cohen Committee of the British Medical Association considered the training of the general practitioner and concluded that the work of a general practitioner comprised a branch of medical practice for which an adequate period of training was necessary.[3] It suggested three years — the first as an assistant; the second in specially designated hospital posts, preferably residential; and the third year in supplementary training at the choice of the trainee. Flexibility would be required for the second and third years in order to meet the varying needs and aptitudes of the trainee. Little action appeared to follow this but the leaven of the report was working in the minds of many people including members of the College of General Practitioners which had been founded in 1953 amid opposition from the existing Royal Colleges.

That the leaven was working was shown in the College's first report on general practice in 1965 — *Special vocational training for general practice.*[4] In this, training for five years was recommended: three in hospital posts and two in general practice. The report commented specifically that trainers in the present Trainee General Practitioner Scheme, though worthy in the eyes of their fellow doctors, had no special gift for teaching.

The next advance in training was foreshadowed by the Royal Commission on Medical Education — the Todd Committee report in 1968[5] — which among other recommendations considered that after registration there should be a period of three years general professional training, followed by two years of further professional training which would be concluded by registration as a specialist or principal in general practice. The Commission stated that 'in our picture of the future pattern of medical service in Britain, all doctors — general practitioners as well as consultants — will be specialists in particular aspects of medicine who will be equally regarded as such and who will be fully trained for the work they undertake'.

The Todd Committee also stated — 'we do not think that the universal requirements for proper training will deter good doctors from entering general practice. In fact, we think that proper training will be an attraction in itself but *unless it is made compulsory* the few who put quick rewards before professional pride will have a financial advantage that will, we think, prevent any general advance in standards'.

TRAINING IN NORTHERN IRELAND

Northern Ireland was moving along a similar path. In 1962 Dr Frank Main, then Chief Medical Officer of the Ministry of Health and Social Services, convened a meeting to consider the immediate problem of continuing education for general practitioners. A steering committee chaired by the Dean of the Faculty of Medicine, Professor Sir John Biggart, recommended that the number of available refresher courses should be doubled to permit practitioners to attend a five-day course every five years. Dr Hunter, Medical Officer at the General Health Services Board — the forerunner of the Central Services Agency — was the organiser. The question of a postgraduate dean to direct the courses and appoint postgraduate tutors was also considered.

In July 1963 an important principle about the vital question of financing postgraduate education was considered. Professor Biggart was adamant that the grant for postgraduate medical education should not be merged with the block grant to the University, but that such funds should be 'earmarked' for postgraduate medical education only and that arrangements should be elastic — demand for courses would vary from year to year and therefore finance was going to change. The question of establishing a postgraduate education board was referred to the Ministry for its opinion. The steering committee met, for the last time, in 1964 to formalise representation on the Postgraduate Education Board and the decision to advertise for a postgraduate dean was taken. There is a gap from January 1964 until October 1965 when the first meeting of the Postgraduate Education Board took place. It can be surmised that those interested in general practice training were working in the background to obtain funds and set up the necessary administrative structure. Dr John McKnight was appointed Director of Postgraduate Medical Education from 1 October 1965 though he had been acting in a part-time capacity for some three months previously.

It should be remembered that these radical proposals for postgraduate education for general practitioners were taking place during the period between 1955 and 1965 when general practice was grossly underfunded and general practitioners were underpaid, overworked and very very unhappy. This was the time when general practitioners signed letters of resignation from the NHS and gave them to the BMA to hold; the time when over 100 general practitioners from Belfast wrote to the *British Medical Journal* about the pool system of payment. The unrest was halted by the 1965 Charter for General Practice which contained proposals for payment of rent and rates, ancillary staff, differential payments for elderly patients, group practice allowances, and so on. The Charter was accepted by Government in 1966 and proposals for promoting and financing postgraduate education for general practice were established.

A paper on vocational training had been considered by Professor Biggart's Committee in the spring of 1965. The relevant paragraph deals with the need for vocational training in general practice in Northern Ireland. 'The need for organised training for general practice is so self-evident that it hardly requires to be argued. Entry to practice by way of an ordinary assistantship may have sufficed as an adequate introduction to this branch of medicine before the 1939–45 war; but, more especially since the inception of the National Health Service in 1948, major changes have occurred both within general practice itself and in the context of general practice, all of which pointed to the desirability of systematic training for those who wished to take up general practice as a career. Among the internal changes in general practice were the growth of partnerships and groups, the employment of ancillary staff in large numbers, and the increasing importance of practice organisation. Among external changes were the development and increasing complexity of hospital services and the provision by local authorities of health visitors, social workers, district nurses, midwives and other domiciliary workers, which called for a greater awareness on the part of the general practitioner in the context of his branch of medicine and close collaboration with other agencies and Health Service colleagues in the interests of his patients. Furthermore, medical science itself was undergoing continual rapid change and development, and if the practitioner was to keep abreast of advances in knowledge he required more time for regular postgraduate study, for reading and for personal contact with professional colleagues. These changes all meant that the young doctor entering general practice was precipitated into a situation far more complicated, but at the same time affording far greater opportunity for the exercise of his skills, than ever before. If he was to make the fullest use of his medical knowledge in the different environment that confronted him on completion of his pre-registration year, some further period of systematic training seemed indispensable'.

In Northern Ireland it was decided to introduce a voluntary training scheme of two years' duration. One year would be spent in a training practice and one in hospital posts. The hospital posts would comprise six months obstetrics and gynaecology and the other six months in three of the following: dermatology, geriatric medicine, paediatrics or psychiatry. This was based on a scheme in Wessex. The Vocational Training Scheme which was to commence on 1 February 1966 was launched on 1 October 1965 and was given wide publicity in the news media. All senior house officers in post were informed about the scheme and a circular was sent to all general practitioners inviting them to apply to become trainers if interested.

There were about 740 general practitioners in Northern Ireland at that time. A surprising response resulted. Eighty general practitioners applied to become trainers and two senior house officers to become trainees. Of the latter both withdrew their applications — one to go into practice and the other to go back into hospital. This was a pattern to be repeated many times over the years until 1980, when mandatory vocational training for general practice came into being.

The initial criteria for appointment of the trainers were:— (1) Ten years a principal in general practice; (2) Less than 50 years old; (3) In a partnership; (4) List size was less than 2,500 in an urban and 2,000 in a rural practice; (5) Suitable practice premises and organisation; (6) Suitable geographical spread; (7) The practice had to practise midwifery.

Twelve trainers were appointed including Dr R E Hadden of Portadown, Dr N D Wright and Dr W G Irwin of Belfast. There were five in what is now the Eastern area, three in the Southern and Western areas and one in the Northern area. Twelve more were appointed in 1969 by the Trainer Selection Committee. It was decided that these, together with those appointed in 1966, should be interviewed in their practices before reappointment. This was a completely new concept and was the first example of peer review in Northern Ireland.

The criteria for the appointment of these new trainers had already advanced from the simplistic criteria used in the appointment of the first trainers. The new criteria showed the change in emphasis to teaching in the practices and were:-

(1) A desire to teach by the applicant, evidenced by past and present activities in the teaching of undergraduate or postgraduate students and paramedical staff, by interest in teaching methods, and attendance at teacher courses.

(2) The provision of time to teach or readiness to make time — at least one session per week.

(3) Attitudes towards patients, partners, colleagues, previous trainees (if any) and general practice itself.

(4) The trainer's and partners' special interests should be noted, particularly research.

(5) The ability to make available the necessary time for teaching by means of good organisation, — in particular the use of appointment systems, employment of appropriate ancillary staff and an efficient records system.

THE NORTHERN IRELAND COUNCIL FOR POSTGRADUATE MEDICAL EDUCATION

The Postgraduate Education Board was succeeded in 1970 by the Northern Ireland Council for Postgraduate Medical Education. The Council's responsibilities included the appointment of general practitioner trainers and the organisation of postgraduate education and training for doctors and dentists. Later it undertook the approval of selected hospital posts as suitable for general practice training in accordance with the Vocational Training regulations of 1979. These responsibilities were exercised through its General Practice Committee.

In Great Britain the standards of the various voluntary training schemes were being considered by the Education Committee of the College of General Practitioners. As part of the Education Committee's work in 1970–1973 it carried out studies on the accreditation of training schemes. Its main aim was to demonstrate that

visiting, preceded by written evidence, was an effective measurement of examining the quality of practices and of stimulating improvement. The recommendations following the study included (1) A national system of accreditation for training programmes should be established; (2) The views of trainees should be sought directly on a completely confidential basis; (3) Regional advisers should serve on visiting panels in regions other than their own — a very valuable exercise; (4) The visiting team should contain one member who had special knowledge of the training content, one with special knowledge of educational methods and one experienced in educational and health service administration.

An increasing administrative load was arising from recruiting trainers, training trainers, organising the day release course and giving career advice to potential trainees. More staff were required to deal with these matters. Dr Noel Wright was appointed as a course organiser in January 1971 and became Regional Adviser in December 1971. Dr Herbie Baird and I were appointed Associate Advisers in 1973 and 1974 and Dr Ben Moran in 1982.

THE JOINT COMMITTEE FOR POSTGRADUATE TRAINING IN GENERAL PRACTICE

The end of the voluntary schemes was presaged by the formation of the Joint Committee for Postgraduate Training in General Practice in 1976 which took over the central administration of vocational training in the UK. This was a national body, set up by the profession, essentially to advise on the implementation of vocational training and later to monitor the performance of regions in providing this training. It also had the statutory duty of issuing certificates of prescribed or equivalent experience. The membership of the Joint Committee was divided equally between the British Medical Association and the Royal College of General Practitioners.

The vocational training regulations came into operation in 1980. Initially, vocational training was not required before becoming a principal in general practice, from February 1981 to August 1982 a year in a training practice was required, and thereafter the full three-year training was required. This was to be one year in a training practice and two years in hospital posts which must include two of the following: medicine, geriatric medicine, paediatrics, psychiatry, accident and emergency or surgery, obstetrics, gynaecology or obstetrics/gynaecology, and the remainder in educationally approved posts.

VISITS TO APPROVED TRAINING SCHEMES

The Joint Committee adopted the methods for visiting previously used by the College for the approval of vocational training schemes in Great Britain and Northern Ireland. Visits were and still are carried out every two years. The visit is undertaken by three general practitioners led by a Regional or Associate Adviser. They are in receipt of documentation which includes the region's criteria for the approval and reapproval of trainers and training practices and they use these as a measure of the scheme. They spend three days in the region, talking first to the Adviser and Associate Advisers, then to the trainees in general practice, to the consultants in the local hospitals and to the course organisers. The team visits eight to 10 training practices. Two visitors interview the trainer, exploring his attitudes to training, his aims, his preparation and his methods of assessment of the trainee. The third visitor talks to the trainee in post and inspects the records.

Minimal educational requirements have been established for medical records — correspondence in date order (1984), long term drug therapy and monitoring prescribing (1986), library (1987) and summary problem lists (1989).

A report of the visit and its recommendations is made to the Chairman, the Secretary of Council, the Regional Adviser, the General Practice Committee and to the Joint Committee. The report is always constructive and gives a fresh perspective of the scheme being visited. Of course it may result in the withdrawal of approval from a region.

CRITERIA FOR THE APPROVAL AND REAPPROVAL OF TRAINERS

From 1980 a potential trainer applied to the Northern Ireland Council to be considered for appointment as a trainer. The applicant received the aims of the Northern Ireland Vocational Training Scheme: to produce a doctor who, on completion of training, will

(1) be able to provide personal, primary and continuing care to individuals and families in their homes and in the community
(2) be able to provide preventive care and health education
(3) have management interprofessional skills relative to general practice
(4) audit his work with a view to improving his performance
(5) understand the importance of continuing medical education and be prepared to participate
(6) be aware of personal and family needs in relation to his work
(7) be competent to pass the membership examination of the Royal College of General Practitioners
(8) hold a Certificate of Prescribed or Equivalent Experience.

He also received proformas to furnish details of his and his partners' curricula vitae, of his practice premises, records, staff, equipment etc, together with the criteria for the approval and reapproval of trainers and the priority objectives of training — these set out the educational standards Council required of its trainers. These documents had been discussed and amended at trainer meetings and courses and approved by the General Practice Committee of Council and Council itself.

In the 1986 paper the trainer's eligibility is clearly stated. He or she must have been a principal for five years and be under 50 years of age. He must demonstrate clinical competence, professional and personal values, and availability and accessibility.

He must want to teach, have time to teach (2 – 2·5 hours per week), have aims for teaching and have attended trainers' courses. He should be able to discuss his use of educational techniques, methods of assessment and involvement with the Primary Health Care Team. The practice should have adequate premises and staff with appropriate records up to the Joint Committee standards.

When the potential trainer considers he fulfils the criteria he is visited by a team consisting of the Chairman of the General Practice and Trainer Selection Committees, an established trainer and the Regional Adviser. If successful he will be appointed for two years with the proviso that he goes on a training course during this time. Subsequent appointments may be made for up to five years. The trainer is the lynchpin of the vocational training scheme. He is in contact with the

trainee daily and has the most direct influence on the trainee's education. The trainer is a very special person and Northern Ireland has been fortunate in bringing together the present cohort of 70 trainers in 62 training practices (ie 62 training places). The numbers of trainers and trainees are approaching one another. At present Council is looking for more trainers especially in the Western area (Fig 2).

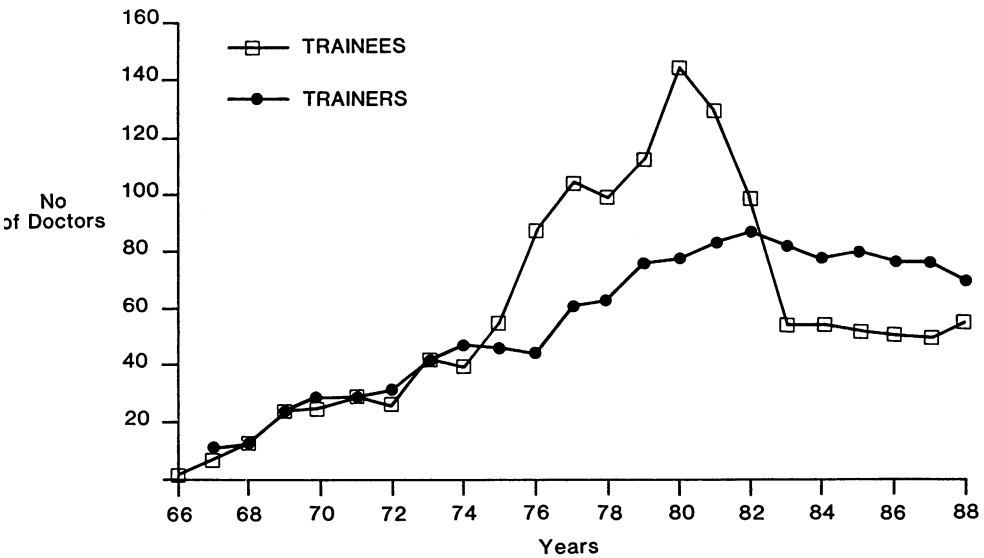

Fig 2. Numbers of trainers and of trainees in general practice in Northern Ireland 1966 – 88. Trainee numbers refer to those in both hospital and general practice posts until 1983.

TRAINER COURSES

Trainers needed instruction in teaching and educational techniques because they had little if any experience in these fields. The first courses were held in the early 1970s and have continued yearly. Trainers were introduced to the educational model which can be entered at any point but always produces a logical outcome and forms the basis of teaching in the practice and in the day release courses (Fig 3).

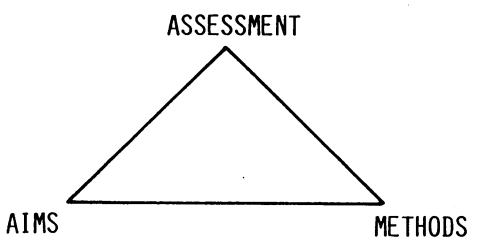

Fig 3. The educational model used in the trainer courses.

Educational techniques such as formal one-to-one tutorials with another trainer assessing the tutorial were practised at the courses. Random case analysis in which the trainer selects a particular patient for discussion, again with an assessor, proved a valuable tool. Role play in which the trainer acts out a patient's consultation was used occasionally and is coming back into vogue. Tape-recorded consultations have been used though they have drawbacks. Video-recorded consultations are really one of the best ways to assess the trainer and trainee; these have been encouraged in the practices and will be used more in the future.

Other subjects discussed over the years include assessment of the trainee, treatment protocols, prescribing and audit. Trainers were and are encouraged to go to Great Britain to attend trainer courses to obtain a different viewpoint of vocational training. These courses are not for the retiring personality as constructive criticism of a member of the course by other participants is both frank and free, and all trainers have to attend on a regular basis.

DAY RELEASE COURSES

The other main teaching activity provided for trainees are the day release courses held in Belfast, Bangor, Craigavon, Ballymena and Londonderry. These supplement the main learning process which takes place in the practice, and concentrate on group discussion and group activity rather than didactic lectures. The former is the most effective method of changing set attitudes which trainees may have absorbed already. The trainee is often deficient in problem-solving skills and in self-awareness.

The day release courses are autonomous and each reflects the ideas and expertise of the organiser and his group of trainees. The subjects discussed are wide-ranging, including an introduction to practice management, developmental screening, study of unruly adolescents and practice visits. The ideas behind the courses are considered and refined at meetings of course organisers. Professor George Irwin and his staff have an input to the courses and deal with subjects in which they have a special interest such as ethics, research, the consultation, and the dynamics of small group teaching. The present and past course organisers cannot be given enough credit for their work in the Northern Ireland Vocational Training Scheme.

Potential general practice trainees in hospital posts attend five or six evening meetings in Belfast. These concentrate on the interface between hospital and general practice under the leadership of trainers. The meetings are didactic in nature because of the numbers of young doctors attending — often 70 or 80. The meetings give the young doctors the opportunity of forming a group identity. In Londonderry potential trainees in hospital posts attend the day release course.

MANPOWER AND TRAINEE SELECTION

In 1981 and in 1986 the Medical Manpower Committee for General Practice, in considering the needs of general practice, decided that 50 trainees per year were required for Northern Ireland to replace those principals who died or retired each year. It was realised that there would be more applicants than trainee places and consequently selection would have to take place. The number of trainees completing their training from 1965 to 1988 is shown in Fig 4. Over the past six years almost all have completed and the numbers are close to 50 per year.

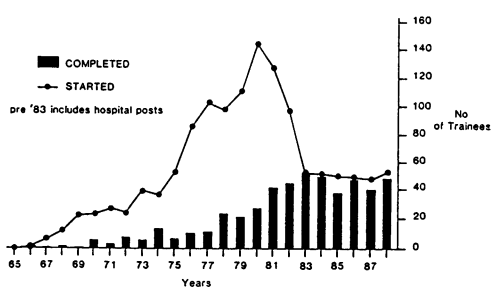

Fig 4.
Number of trainees who commenced and completed the course, 1965–1988. Before 1983 all trainees in hospital posts were counted. In 1983 and after, only those who completed a general practice year were counted.

Senior house officers who are potential general practice trainees gain their hospital experience in two ways — either by constructing their own rotations by applying for a post every six months, or by obtaining a post of two years' duration which rotates through the necessary specialties. The hospital experience obtained in Northern Ireland by trainees appears to be superior to the experience shown on the application forms submitted to the Joint Committee in the United Kingdom for Certificates of Prescribed Experience (Table).

TABLE

The distribution of Senior House Officer posts in Northern Ireland hospitals which are approved for general professional training and for vocational training for general practice compared with the Senior House Officer experience shown on the application forms submitted to the Joint Committee in the United Kingdom for Certificates of Prescribed Experience

	SHO posts shown on application forms to NIVTS August 1987 (total number of applications in brackets)	*SHO posts shown by UK Joint Committee certification 1987 (total number of certificates in brackets)*
Psychiatry	(33) 31·4%	(906) 40·5%
General medicine	(88) 83·8%	(1026) 45·8%
Geriatrics	(18) 17·4%	(878) 39·2%
Obstetrics and/or Gynaecology	(95) 90·5%	(1489) 66·5%
Paediatrics	(54) 51·4%	(1301) 58·1%
Accident and Emergency/Surgery	(75) 71·4%	(1507) 67·3%

It was decided that entry to the scheme would be for the general practice year only, both for doctors in self-constructed rotations and for those in two-year SHO rotations. The Central Services Agency is responsible for the appointments panel. Central selection has worked successfully over the years bearing in mind that the interview panel may have to spend three days in interviewing 100–110 candidates for 50 posts. Contrary to general belief, the composition of the panel does not change during the three days. The selection procedure is a great imposition on the time of the general practitioners and consultants who are on the panel.

ASSESSMENT OF THE TRAINER AND TRAINEE

Assessment of the trainer and trainee — the third apex of the educational model — has proved more difficult. The trainee assesses the trainer and training practice twice during the general practice year. The assessment is confidential to the trainee and the Adviser and cannot be used without the consent of the trainee. It is very rarely possible to obtain this. Even with this drawback the assessment gives valuable information. The trainer is also assessed by the trainer approval panels and the visitors from the Joint Committee (Fig 5).

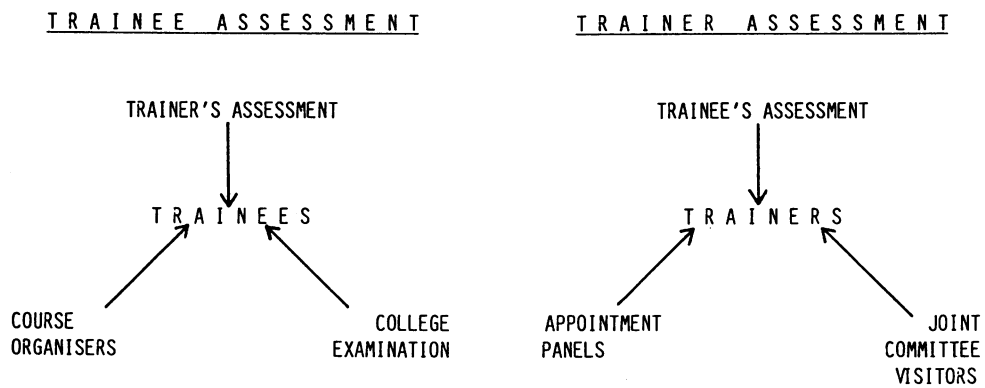

Fig 5. Models for trainee and trainer assessment.

Assessment of the trainee has been carried out over the years, but methods used in the past did not give reliable results. Recently the trainer has completed two assessments each year on the trainee, dealing with patient care; communication skills; knowledge of practice organisation, the statement of fees and allowances, and medical politics; personal values and personal and professional growth. The trainer discusses these assessments with the trainee, and this is the most valuable part of the exercise. The Course Organisers also assess the trainees at the day release course. At present the main assessment of the trainee is the membership examination of the Royal College of General Practitioners. Northern Ireland candidates have been extremely successful (Fig 6). Northern Ireland has a pass rate of about 95% for the past five years, and a very high distinction record. These excellent results are due to the teaching in the hospitals, the day release courses at the training practices, and the hard work of the trainees themselves.

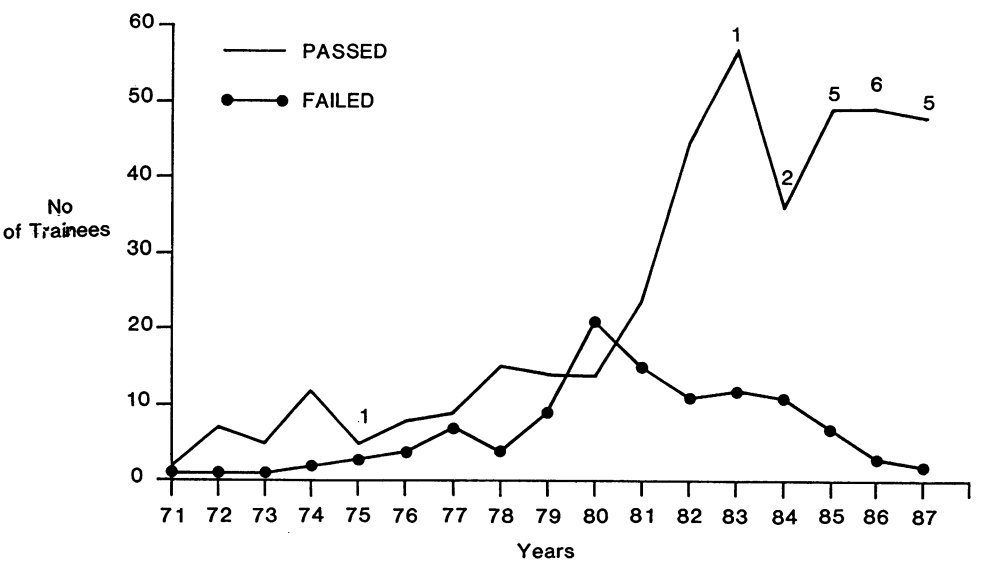

Fig 6. Number of trainees passing and failing the MRCGP examination in Northern Ireland, 1971 – 87. The numbers on the pass curve refer to those obtaining distinction.

A continuing change in education from hospital-based to general practice-based teaching of general practitioners is taking place.

WHAT OF THE FUTURE?

It is possible that the period of vocational training may be increased to four or five years with two years in hospital and two years in a training practice. The increasing number of computers in practices and their effect on audit and education has yet to be assessed but the indications are that they will make a significant impact. How will nurse/practitioners affect the role of the general practitioner, and how will day care in hospitals and the increase in community care affect the work of the health care team? The change to anticipatory care in general practice, prevention, health education, audit, and the emphasis on team work will pose new educational problems for vocational training and for general practice itself.

I would like to thank Miss Arlene Stockman, my secretary, for her work with this paper; the photographic department of the Belfast City Hospital and Mrs Gillian Stott of the Northern Ireland Council for Postgraduate Medical Education for preparing the figures.

REFERENCES

1. Hill D. Psychiatry in medicine — retrospect and prospect. London: Nuffield Provincial Hospitals Trust, 1969: 158.
2. Ministry of Health. Interdepartmental Committee on Medical Schools. Report. (Chairman, W Goodenough). London: HMSO, 1944: 202.
3. British Medical Association. General practice and the training of the general practitioner. (Chairman, H Cohen). London: BMA, 1950: paras 2, 24.
4. College of General Practitioners. Special vocational training for general practice. London: Council of College of General Practitioners, 1965: pts II, III.
5. Royal Commission on Medical Education. Report. (Chairman, Lord Todd). London: HMSO, 1968. (Cmnd 3569): paras 11, 117, 119, 121.

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Serving two masters

A H G Love

Annual Oration at the opening of the 1988–89 teaching session, Royal Victoria Hospital, delivered on 6 October 1988

The title that I have chosen for the 108th Oration, to open the 1988 teaching session, is not an excuse to indulge in a theological discourse — this is neither the time nor the place. Nor do I wish, as a joint appointment University staff member, to discuss the special arrangements in Belfast which accord such individuals a contract of employment with both the University and the Health Board. Such a discussion might suggest distinctions which do not exist in reality. There is a great unity of purpose shared by all members of staff in this Hospital. The three cardinal features of academic medicine — namely, patient care, teaching and research — are pursued with equal zeal and enthusiasm by both NHS and University employees alike. I am proud to say that University staff do not sit in isolated ivory towers but are busily involved in the day-to-day service provided by this Hospital. At the same time the NHS staff give generously of their time and energy in the education of each succeeding generation of students. The research profile of the hospital is not the prerogative of the academic. Much of the international reputation of this Hospital is based on a wide variety of excellent basic and clinical research pursued by Health Service and academic staff alike.

As a clinical scientist I might not be regarded as impartial in any examination of the difficulties posed by the pursuit of twin 'objectives of medicine' as a science and as an art. I have no doubt that we must continue to follow the true ideals of science in medicine as the quest after knowledge to conquer disease, and at the same time practise the art of medicine in the treatment of disease in our fellow men. It is perhaps paradoxical that it is the very success of science in medicine that should be one of the major causes of today's doctors' dilemma. Over the past 30 years or so the success of science in medicine has steadily expanded the scope for its practice. Large numbers of previously untreatable diseases have become treatable and this has inevitably led to a steadily rising demand for more and more forms of medical care. There is now emerging a conflict between the individual patient and the resources which are available for health care within a community. Society is now intensely interested in what we as professionals are doing — we are no longer only able to think of the individual patient — we must place our practice in the public arena. We have the patient and society as two masters to whom we must answer. It is these, apparently at times, conflicting perspectives of medicine that I wish to address.

Ideal medical care depends to a significant extent on the physician being the advocate of the patient. Yet there are many situations in which the physician must act as a double agent. For example, the physician may be forced to wear two hats

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when requested by an insurance company to judge the ability of one of his older patients to hold a driving licence. Assuming the patient is moderately impaired, and the answer could honestly be yes or no, should the physician's report favour the patient, or should the patient be forced to stop driving to protect the public and the insurance company? Of interest to us, in teaching hospitals such as this, is the balance between patient care and the interests of teaching and research. Patients, my own included, at times have been fatigued by too many undergraduate and postgraduate students and probably exposed to too many expensive tests and procedures. We must be grateful for the long-suffering attitude of our patients and strenuously preserve a proper balance of activity. It is, however, in the current state of cost containment as medical care rationing begins that we face the much more unpleasant prospect of addressing the potentially conflicting interests of the patient and society.

THE MEDICAL MODEL

Practitioners of medicine down the ages have subscribed to the Hippocratic Oath or one of its more recent Geneva or Sydney variants. The Oath embodies many of the best traditions of medicine and formulates how doctors should behave towards their patients. For the benefit of our students, I will refer to the following modern translation:[1]

> 'I swear by Apollo the healer, by Aesculapius, by Health and all the powers of healing, and call to witness all the gods and goddesses, that I may keep this Oath and Promise to the best of my ability and judgement.
>
> 'I will pay the same respect to my master in the Science as to my parents and share my life with him and pay all my debts to him. I will regard his sons as my brothers and teach them the Science, if they desire to learn it, without fee or contract.
>
> 'I will hand on precepts, lectures and all other learning to my sons, to those of my master and to those pupils duly apprenticed and sworn, and to none other.
>
> 'I will use my power to help the sick to the best of my ability and judgement; I will abstain from harming or wronging any man by it.
>
> 'I will not give a fatal draught to anyone if I am asked, nor will I suggest any such thing. Neither will I give a woman means to procure an abortion.
>
> 'I will be chaste and religious in my life and in my practice.
>
> 'I will not cut, even for the stone, but I will leave such procedure to the practitioners of that craft.
>
> 'Whenever I go into a house, I will go to help the sick and never with the intention of doing harm or injury. I will not abuse my position to indulge in sexual contacts with the bodies of women or of men, whether they be freemen or slaves.
>
> 'Whatever I see or hear, professionally or privately, which ought not to be divulged, I will keep secret and tell no one.
>
> 'If, therefore, I observe this Oath and do not violate it, may I prosper both in my life and in my profession, earning good repute among all men for all time. If I transgress and forswear this Oath, may my lot be otherwise'.

This encapsulates the concepts of the best possible practice of medicine for the individual patient. It hints at family but no further. The ancient seer had no doubt

that this behaviour would receive the blessing of deity. I am sure that we could agree that such service to our fellow men would be accorded divine approbation — in that sense we are servants of the Almighty. It is the present day resource dimension, Mammon, if you like, which represents one of the demands of our second master. It is now being suggested that the principle on which medical practice is based, that one should do everything possible for the individual patient, is a luxury that we as a society can no longer afford. As more and more practices that may be beneficial to the individual but not necessarily to the interests of society are pursued, we risk reaching a point where the marginal gains to the individual threaten the welfare of the whole community. How best then can we in the present and foreseeable future act as the patient's advocate? I believe we can perform this role only if we are aware of the current pressures, the public perception and even criticism of medical practice.

It is perhaps not sufficiently realised that medical practice and the public perception of it has changed rapidly in a relatively short period of man's history. Not much more than two to three generations ago there was no such thing as a public expectation for doctors to do, or to be, anything other than what doctors had always been: professionals who were called in to attach particular names to particular illnesses, to provide reliable information about the natural history of the illness, so that the patient and family could have a fairly clear idea about what was happening or was about to happen; then stand by and see that good nursing care was available.

Fifty years or so ago, treatment of disease had largely been given up. Medicine had been through several centuries of athletic efforts to change the course of disease by empirical remedies — mostly the figments of fertile imaginations — like bleeding patients to the verge of shock; like blistering or catharsis; even stupefaction by alcohol which was common in the nineteenth century. Finally everyone agreed that such things did not really work or were maybe positively harmful. We had then arrived at a state of therapeutic nihilism — led forcefully by that great physician and teacher Sir William Osler. But in the mid-1930s medicine suddenly changed into effective mode. Things could be cured. Antibacterial agents for infections, vaccination against childhood contagions, virtually eliminated many lethal, frightening and ubiquitous diseases. That is when expectations in the public mind began to rise; they have never since stopped rising, yet many feel that medicine has not continued to deliver with the same dramatic force.

It is, I believe, salutory to reflect on the state of medicine just 300 years ago. Medicine in these isles was at that time a combination of ignorance, superstition and downright charlatanism —

> 'At eight o'clock on Monday of February 2, 1685, King Charles II of England was being shaved in his bedroom. With a sudden cry he fell backward and had a violent convulsion. He became unconscious, rallied once or twice, and, after a few days, died. Doctor Scarburgh, one of 14 physicians called to treat the stricken king, recorded the efforts made to cure the patient.
>
> 'As the first step in treatment, the king was bled to the extent of a pint from a vein in his right arm. Next his shoulder was incised and the area 'cupped' to suck out an additional 8 ounces of blood. After this the drugging began. An emetic and purgative were administered, and soon after a second purgative. This was followed by an enema containing antimony, sacred bitters, rock salt, mallow leaves, violets, beet root, camomile flowers, fennel seed,

linseed, cinnamon, cardamon seed, saphron, cochineal, and aloes. The enema was repeated in two hours and a purgative given. The king's head was shaved and blister raised on his scalp. A sneezing powder of hellebore root was administered, and also a powder of cowslip flowers "to strengthen his brain". The cathartics were repeated at frequent intervals and interspersed with a soothing drink composed of barley water, licorice and sweet almond. Likewise white wine, absinth, and anise were given, as also were extracts of thistle leaves, mint, rue, and angelica. For external treatment, a plaster of Burgundy pitch and pigeon dung was applied to the king's feet. The bleeding and purging continued and the following medicaments were added: melon seeds, slippery elm, black cherry water, an extract of flowers of lime, lily of the valley, peony, lavender, and dissolved pearls. Still to come were gentian root, nutmeg, quinine, and cloves.

'The king's condition did not improve, in fact, it grew worse, and in the emergency 40 drops of extract of human skull were administered to allay convulsions. A rallying dose of Raleigh's antidote was forced down the king's throat; the antidote contained an enormous number of herbs and animal extracts. Finally bezoar stone was given. "Then, alas", said Scarburgh, "after an ill-fated night his serene majesty's strength seemed exhausted to such a degree that the whole assembly of physicians lost all hope and became despondent; still, so as not to appear to fail in doing their duty in any detail, they brought into play the most active cordial". As a sort of grand summary to this pharmaceutical debauch a mixture of Raleigh's antidote, pearl julep, and ammonia was forced down the throat of the dying king'.[2]

Such extravagances were not confined to this side of the Atlantic. George Washington, said to be a hale and hearty man in his late sixties, developed a severe sore throat, after a tiring horseback ride in the snow, on 12 December 1799. A blistering poultice was applied to his neck, he was required to gargle a mixture of vinegar and molasses, and he was bled for a total of five pints of blood. Among his last words were 'I thank you for your attentions, but I pray you to take no more trouble about me'.[3]

How have we reached today's state of art and knowledge from such a starting point? Arguably the most influential mind in promoting progress was that of Thomas Sydenham. He was alarmed by the confused and disorderly state of medicine and determined to improve it. The function of the physician, he claimed, is the industrious investigation of the history of diseases and of the effect of remedies, as shown by the only true teacher — experience. There must be a history of the disease, a description that shall be at once graphic and natural, and also a praxis or methodus medendi, which must be regular and exact, fixed, definite and consummate, by which the practice is based on a sufficient number of experiments and so proves competent to cure this or that disease.[4] In Sydenham's day, the main resource for reaching a diagnosis was the history of the illness. Examination was largely confined to inspection of the appearance of the patient. Sometimes touch was used to feel the temperature of the skin, the pulse and any obvious swellings. Occasionally, urine, stool or shed blood were examined. This provided a suspicious clientèle with an opportunity to test the doctor's fallibility by substituting someone else's urine or even a specimen from an animal. Medical textbooks of the time described how to distinguish animal from human urine by inspection.

The eighteenth and nineteenth centuries saw a great extension of the powers of the clinician as diagnostician. Augenbugger introduced percussion and Laennec the stethoscope. Then in the second half of the nineteenth century new technologies were developed which further extended the examination to inside the patient. Technology was moving into medicine. Today there is a bewildering array of possibilities. Fibre optics offer visulisation of the stomach, duodenum, bile ducts, even joints and blood vessels. Body imaging can be effected with isotope scans, ultrasound, CT scanners or magnetic resonance. We can measure the concentration of almost any body constituent and study almost any function. Modern DNA technology allows inspection of the genetic profile of the individual. Sir Douglas Black has stated — 'The essential difference between medicine today and the medicine of a hundred years ago is that our intellectual comprehension of disease has entered another dimension. In so far as medicine ever can be a success story, the success comes ultimately from science'.[5]

Despite such an impressive scientific base for clinical practice there are those who criticise modern medicine for its excessive reliance on science and its associated technology to the detriment, it is claimed, of those aspects of medicine which are variously described as holistic, human, emphatic or simply caring. The indictment is based on a number of strands — that the pursuit of science produces cold, detached people; that preoccupation with technology destroys compassion and distracts attention from the needs of the patient; that an awareness of biological systems precludes awareness of the whole person; that preoccupation with restoring functions, relieving symptoms, and concentrating on details of specific diseases may bring about neglect of basic problems of personality, environment or lifestyle; that the concentration of investigation in hospitals overemphasises their importance in relation to the total burden of illness; and that medicine based on scientific problem solving runs away with resources which could be better spent.

The Reith lecturer of 1980, the distinguished lawyer Ian Kennedy, now a member of the General Medical Council, stated the case forcibly — 'Modern medicine has taken the wrong path. An inappropriate form of medicine has been created, in large part by doctors and medical scientists, and eagerly accepted by a willing public. The nature of modern medicine makes it positively deleterious to the health and wellbeing of the population. Please understand that it is we, all of us, who have hitched our wagon to the wrong star, scientific medicine, as our guiding light'.[6]

The public concern relating to modern scientific and technological medicine is based on at least three anxieties. The fears and concerns about *technological developments* are myriad: annihilation by the bomb, dehumanisation, unemployment, pollution, urban blight, extinction of plants and animals, disposal of waste — to name but a few. The impact of technological change on industrial and urban society is blamed for the deterioration in family life, insecurity, anxiety, alienation, boredom, the escalation of drug abuse and the incidence of mental illness. Although technological development has not always been generated by scientific research, science is the major source for the knowledge required to generate technological change, and technology is often defined as the translation into practical use of the results of scientific research. Much of today's controversy over the role of technology in our society has to do with differing opinions as to whether a new product, service or process represents an improvement or a liability, progress or decline, benefit or burden. Medicine gets caught up in this

general discussion. Society and patients within it feel perhaps less able to exert influence in the field of medicine than in some other areas of life. The consumer of medical technology is strictly speaking not the patient, but the physician. The physician is the one who makes decisions about hospitalisation, diagnostic tests, operative procedures and the use of drugs. There is the impression in many minds that, in caring for the individual patient, the physician follows what many have called the 'technological imperative', the belief that every physician in every hospital should have available for that patient all the technologies of medicine, regardless of cost, questions of priority or the optimal allocation of resources.

This sense of remoteness from an impersonal technological world leads to the second anxiety of patients — that concerning *compassion.* Medicine was once the most respected of all the professions. Today, when it possesses an array of technologies for treating and curing diseases which were simply beyond comprehension a few years ago, medicine is under attack for all sorts of reasons. Doctors, the critics say, are applied scientists, concerned only with the disease at hand but never with the patient as an individual, whole person. They do not really listen. They are either unwilling to or cannot explain things to sick people or their families. They make mistakes in risky technology, hence the rapidly escalating cost of medical malpractice insurance.

What is it that people have always expected from the doctor? How, indeed, has the profession survived for so much of human history? Doctors as a class have always been criticised for their deficiencies. Shaw saw doctors 'just like other Englishmen: most of them have no honour and no conscience: what they commonly mistake for these is sentimentality and an intense dread of doing everything that everybody else does not do, or omitting to do anything that everybody else does. The medical profession has not a high character, it has an infamous character. I do not know a single thoughtful and well informed person who does not feel the tragedy of illness at present is that it delivers you helplessly into the hands of a profession which you deeply mistrust'.[7] What were the patients of physicians in the nineteenth century and before hoping for when they called for the doctor? In the years of the great plagues, when carts came through the streets each night to pick up the dead and carry them off for burial, what was the function of the doctor? Bubonic plague, typhus, tuberculosis and syphilis were rapidly progressive and usually fatal infections, killing off most victims no matter what was done by the doctor. What did the man do, when called out at night to visit the sick for whom he had nothing to offer in palliation, much less cure? There must have been compassion — and the oldest and most effective act of doctors — the touching. Some people don't like being handled by others. But that inhibition is not, or almost never, felt by sick people. They need to be touched, and part of the dismay in being very sick is the lack of close human contact. The doctor's oldest skill was to place his hands on the patient. That skill became more specialised and refined with time. The feeling of the pulse, the tip of the spleen, the edge of the liver. Percussion to elicit resonance or dullness over the lungs but at the same time, touch. Touching with the naked ear was one of the great advances in the history of medicine. Once it was learned that the heart and lungs made sounds of their own, and that the sounds were useful in diagnosis, physicians placed an ear over the heart and chest and listened. It is hard to imagine a friendlier human gesture. The stethoscope was invented in the nineteenth century, vastly enhancing the acoustics of the thorax, but removing the physician a certain distance from the patient. It was the earliest device of many still to come which would further distance patient from doctor. Today, the

doctor can perform many tasks from his office without ever seeing the patient. Some have gone so far as to have computer programs taking a history and a computer print-out listing diagnostic possibilities. Instead of spending time examining the chest, a slip of paper can take the patient to the X-ray department or scanning unit and there in great detail are revealed all the internal organs. Computerised devices reveal electronic intimacies of the flawed heart or malfunctioning brain with a precision far beyond the touch or reach, or even the imagination, of the physician at the bedside a few generations back. Medicine appears to many to be no longer the laying on of hands, but rather the reading of signals from machines. Scientific medicine is here to stay. The new medicine works. It is a vastly more complicated profession. The physician has the same obligations that he carried, overworked, and often despairingly, fifty years ago, but now he must add technological expertise and precision. He must still remain the patient's advocate in time of trouble and adversity.

One of the hard things to learn in medicine, even harder to teach, is what it feels like to be a patient. In days past when serious illness was a more common experience, the doctor had usually been through a few personal episodes and had a good idea of what it was like for his patient. Many physicians in pulmonary medicine were brought up in the early days of this century and first acquired their interest in the field from having had pulmonary tuberculosis themselves. Many of the leading figures in rehabilitation had been crippled personally by poliomyelitis. It is different today. The serious and life-threatening illnesses are largely reserved for one's advancing years. No one goes through the six perilous weeks of typhoid fever any more, coming within the sight of dying every day, getting through at the end a stronger character perhaps, certainly with a different outlook on life. The high technologies which are the norm today to cope with serious disease are matters to be mastered only from lecture notes and books, and then by actual practice on patients, but few doctors have more than an imagination of what it is actually like to go through such experiences. Even the childhood contagions are mostly gone, thanks to vaccines for measles, whooping cough, chickenpox and the like. You, as today's young physicians, probably do not know what it is to have earache let alone anything more sinister. The physician therefore must continually strive to conduct high quality scientific medicine in the context of patient sympathy and understanding.

We must address these issues if we are to serve our patients and the public conscience adequately. Sir Douglas Black has well said, 'The true antithesis of caring medicine is not scientific medicine, or high technlology medicine, or hospital medicine, or academic medicine or orthodox medicine, it is quite simply "bad medicine"'.[8]

If medicine has been caught up in the general fear of science and technological developments, patients feeling a sense of distance from their physician — the third problem is the perceived *cost of modern medicine.* Society is suspicious that the escalating cost of health care is largely related to the expansion of expensive technology. The high cost and dubious benefit of high technology medicine, together with its relevance to the relatively limited field of acutely ill patients, are seen in stark contrast to the low technology, perceived low cost needs of large numbers of chronically sick and the elderly. The last Royal Commission on the Health Services declared society's position: 'The emphasis on acute and high technology medicine is being challenged and more thought is being given to the chronic sector'.[9]

The concern of society that the balance of the allocation of health care resources should be adjusted away from high technology is widely voiced. In Kennedy's Reith Lectures he poses the major criticism that too much expensive technology is being used without due consideration of the resulting benefit, and that it often does more harm than good; that money, time and effort might be better spent on other activities. Public opinion is, however, a hard taskmaster because, as well as complaining about costs, society is expressing indignation that certain technologies are not more widely and readily available. Most often this plea relates to technologies that are generally accepted to be beneficial, such as renal dialysis and transplantation, and surgical joint replacement. Given the physician's dual role as advocate for the patient and at the same time spender of public resources, it is imperative that we examine closely the use of these resources and the costs of our activities.

THE SOCIAL MODEL

I would now like to turn your attention to another aspect of public perception of the role of the doctor in providing for the needs of society. The new terminology is interesting. The very word 'medicine', once used to describe the whole enterprise of looking after sick people, has been replaced by 'health care'. Patients are now formally known as 'consumers'; the doctors, along with hospitals and clinics, as 'providers'. The thing to be dealt with is no longer this or that disease, it is 'health'. The desired state is not just one in which disease is prevented or treated, it is a new situation called 'wellness', a destiny to which all are inalienably entitled; and this ideal condition is a lot more than freedom from disease. This is no longer the medical but the social model.

For a statement of these contrasting models, let me refer to the *Health and health policy* report of the Social Science Research Council.[10] This report lists different emphases or polarities: individual and population; treatment and prevention; cure and care; hospital and community; acute and chronic. The authors clearly imply that the physician is far too interested in individual patients who require treatment, meaning curing acute disease in hospital. The opposing and, they claim, society's demand is for population health by prevention and care of chronic problems in the community. You may think that stated in those terms the two positions are extravagant nonsense. Yet I quote from an official government report and it is now incorporated in the policy of transfer of resources from the acute sector to other activities.

Attempts to sort out the basic ideas and issues concerning the provision of health care are not new, they go back to the beginning of our Western civilisation. They are found in Plato's *Republic* where distinctions were made between acute treatment, chronic treatment and preventive medicine. Plato argued that a proper role for medicine was to care for wounds and some seasonal maladies, but that medicine which focused on the treatment of illnesses due to sloth, personal excesses, or poor personal regimen was an index of a society's disgraceful state of luxury or of poor education. He held that the ordinary reasonable person would in the ideal state expect treatment of his acute, not his chronic diseases. Socrates gave the example — 'A carpenter when he is sick expects his physician to give him a drug which will operate as an emetic on the disease, or to get rid of it by purging or the use of cautery or the knife. But if anyone prescribes for him a long course of treatment with swathings about the head and their accompaniments, he hastily says that he has no leisure to be sick, and that such a life of preoccupation

with his illness and neglect of work that lies before him is not worth living. And, thereupon, he bids farewell to that kind of physician, enters upon his customary way of life, regains his health, and lives attending to his affairs — or if his body is not equal to the strain, he dies and is freed from all his troubles'.

Individual and population medicine

The traditional image of the doctor is of someone who responds to appeals for help from those who are ill, or from their relatives. This must be a demand-led service, since only an individual can articulate a demand. Criticism of the conventional medical services comes mainly from two main sources — those who consider the present degree of emphasis on physical and social environmental factors to be inadequate, and those who believe that the preoccupation with individual medicine leads to the neglect of the particular needs of disadvantaged groups, such as the elderly and the physically and mentally handicapped. This latter area has promoted 'client-orientated medicine' — the term client to me depersonalises the individual member of the group and I think there are distinct dangers in segregation and loss of identity. This may be further compounded by unnecessary institutionalisation.

Acute or chronic sectors

Growth in the acute hospital services has been more rapid, since the inception of our health service, than growth in the provision for chronic disability. This is not bad planning, but rather a response, and an extremely effective response, to perceived need. To make good the shortfall in the chronic sector without 'new money' but rather depletion of the acute sector is an extremely doubtful course of action and has certainly no indication of being actually beneficial. It must not be forgotten that acute treatment can prevent chronic disability — for example, hip joint replacement and hernia repair. Such acute treatment is only effective if the resources are available, and that at the right time. The artificial gap between acute and chronic sectors is bridged by the patient. We must strive for good communication between different agencies rather than artificial separation of functions — otherwise, the patient wil suffer.

Hospital or community

Present-day conventional wisdom is that there should be a shift of resources towards community care which, in a time of financial constraint, means away from the hospital. A variety of reasons may be produced to support this position: compassion for the most disadvantaged members of the community, a belief that it will somehow be cheaper to look after people in the community than it is in hospital and a predilection for the use of less trained staff as compared with specialised professionals. These concepts have still to be tested and almost certainly are too simplistic — what of the cost to the community, borne as it is likely to be very largely by individual families at an unknown social cost? We are in danger of forgetting that the hospital is, in fact, part of and serves the community; the hospital and community services are not mutually exclusive: they are interdependent. Muir Gray has well described care as a four-box system.[11] We tend to forget box 1 — self care — and the informal care of family, friends and volunteers which is box 2. It is only when these cannot cope that box 3 — community care — and box 4 — hospital — are required. If cost were the only determinant of resource allocation, then we would opt for the cheapest: box 1 — self care. By deduction, we would need no health service and also stop

helping one another — absurd but, nevertheless, the logical conclusion using present-day wisdom in health service resource planning. No, in the real world, priority must be given to appropriateness of care, the cost being considered in context, not elevated to an absolute criterion of decision. The ultimate test of any health service lies in what it does for people. In a good service the actual patient may spend time in hospital, or being cared for in the community, according to need, not according to value judgements on the importance of hospital versus community. Easy transfer from one to the other is the proof of any integrated, patient-sensitive service.

Treatment or prevention

All would agree that the health of a community may be improved in many ways, not only by the activities of health professionals but also by social and economic changes that result in better nutrition, smaller families, less overcrowding and better education. It is now commonly argued that the burden of disease and the cost of services could both be reduced by redistribution of funds in favour of prevention. This opinion is based on many factors. One is the very success of curative medicine which has been so great that cures are taken for granted and clinical medicine is now blamed for the remaining failures. The increasing scope, complexity and cost of curative services has been so great that Government is on record as saying — 'Curative medicine may be increasingly subject to the law of diminishing returns'. The public experience of the hazards of drug treatment has also strengthened the lobby for prevention. Such is this enthusiasm that it is now fuelling an explosion of paperback bestsellers — *Diet for a strong heart, The cancer prevention diet, How to double your vital years, Healing with yoga therapy and nutrition* and, to go one better, *Creating health: beyond prevention toward perfection.* Before we become servants of this particular master it is important to ask some particular questions. If prevention is seen as an alternative to scientifically based treatment then it must also be capable of surviving the rigour of scientific scrutiny, not just acquiescence to the old adages, 'prevention is better than cure', 'an apple a day', etc.

The undoubted success story in preventive medicine must be immunisation. On a worldwide scale, smallpox has been eradicated. In 1940, 46,000 cases of diphtheria were reported in England and Wales with 200 deaths. In the past 10 years there have only been occasional cases and those in non-immunised individuals. In 1955 there were 4,000 cases of paralytic poliomyelitis; now we have less than 10 per year. For measles, the picture is more unsatisfactory. In 1982, over 100,000 cases were reported with 20 deaths; in 1984 there were still 60,000 cases, due largely to only 63% of children being immunised. For whooping cough the picture is very depressing. Because of adverse publicity in the 1970s relating to vaccine-induced encephalitis, vaccination rates have fallen alarmingly. Now we see 65,000 cases with 15 deaths per year from the disease. These risks far outweigh the most pessimistic estimates of vaccine damage — at most, one in 350,000 children vaccinated. The failure to exploit the benefits of immunisation raises immediately a fundamental question in any preventive programme — what is the degree of personal commitment in the community?

Leaving aside the confusing phenomenon that life expectancy is steadily increasing, there are certain areas that merit our attention. More than half of the lives lost under 85 years of age are now due to death between 55 and 74 years and only 11% to death in infancy and childhood. More than half at all ages are due to neoplasms and ischaemic heart disease. Clearly, therefore, any major advance

now requires an attack on these main killing diseases of middle age. If one looks at the major causes of increased mortality in the past 20 years, however, the reasons are fairly obvious — increasing use of alcohol, addictive drugs, road traffic accidents, cigarette smoking by women, industrial exposure to asbestos (particularly in men), and a greater number of sexual partners. Again, a question of human behaviour.

Human behaviour and health promotion

In any preventive programme then there must be evidence of the ability to modify human behaviour on a large scale — not just in a small sector of society who are already particularly dedicated. We do well to remember the words of Oscar Wilde — 'Man as a rule finds it easier to depend on healers than to attempt the much more difficult task of living wisely'. Any success, therefore, must take account of the public perception of risk and health priorities. The Active Health report recently published by the Canadian government makes interesting and, at the same time, depressing reading:[12] 88% of citizens surveyed assessed their health as good, very good or excellent. How then do you convince people to improve their health if they already feel that their health is good? Substantial numbers in this category smoked, drank excessively and used drugs. However, nearly two out of three respondents indicated that they had done something in the previous year to improve their health. Increasing exercise was the most frequent change, followed by improved eating habits. Weight loss and stopping smoking were cited by a small minority and reducing alcohol and drug use were cited by less than two per cent. It was clear that the public response was to undertake new positive activities rather than give up old bad habits. This is precisely the opposite of most health promotional and preventive programmes. The success therefore of any programme must account for these two types of human behaviour. When people are urged to avoid a health risk, we are usually asking them to give up some well established habit like smoking. But when they are urged to protect or improve their health, they more often than not are being asked to acquire some new behaviour like controlling weight. The strategies used to instigate new behaviour must often be very different from those used to break bad habits. Even when convinced of the need for action, few actually intended to take it. Of those who said they should improve eating habits, only half intended to do so; of those who said they should stop smoking, only half intended to do so. The gap between what people say they should do and what they actually intend to do becomes even more striking when we relate it to health risk behaviour. Of regular smokers, a third indicated that increasing exercise was the thing they intended to do to improve their health — not stop smoking. Yet the value of exercise is debatable while the dangers of smoking are incontrovertible. Chesterton was less than convinced on the matter of exercise — he claimed to get his exercise by attending the funerals of his more energetic friends!

In addition to the problems of behaviour modification, another major problem exists. It is perhaps not sufficiently realised that measures which might benefit a community, for instance by reducing blood lipid levels to lower the incidence of coronary artery disease, may offer little or no benefit to the individual. We are all familiar with the grieving relative whose departed never smoked or drank, took regular exercise, ate a healthy diet, was not overweight, had regular check-ups, yet died of heart disease. To the individual, therefore, there is no guarantee that a change in life-style will protect against heart disease, cancer or anything else. Conversely, we have all come across people who flouted all the rules of healthy

living and outlived their more cautious contemporaries. The centenarian celebrating his 100th birthday is reputed to have said, 'If I'd known I was going to live this long I'd have taken better care of myself'.

Regular screening of health individuals is often advocated, but the case for the annual check-up, so popular with the business executive, has little medical support. For selected age groups of women there is a good case to be made for regular checks for breast or cervical cancer. There is considerable controversy about screening at any age for high blood pressure, not only because of the logistical problem of the procedure but because of the indecision about what to do with the result. Some make extravagant claims for prevention — and I quote Kennedy again — 'We must concentrate much more on primary preventive medicine. If this means, as it inevitably must, that some aspects of modern curative care must be neglected or abandoned, so be it. The benefits to be gained outweigh any loss'.[6] I doubt, however, if anyone has a clear idea of the relative returns to be expected from investment in any of these fields.

CARE AND PREVENTION — TWO MASTERS

Rather than see curative procedures pitted against prevention, I think it is much more satisfactory to see the doctor's responsibility bridging both philosophies. The obstetrician has served both masters admirably for many years. In a case of eclampsia he would ask 'What went wrong?'. The occurrence of a preventable disaster is a threat to his professional reputation, for an obstetrician accepts prevention as an integral part of his normal professional responsibilities. Antenatal care is, in fact, largely preventive, and integration of prevention with treatment has led to a marked fall in maternal and perinatal mortality rates. Is this not a model for us all? Rather than separation, let us bring therapeutic and preventive roles together and enlarge our horizon from feeling that the care of the sick is our only responsibility.

Appreciation of the cost factor in medical care is not new. Sir William Petty (Professor of Anatomy at Oxford) in his plan of 7 October 1667 'of lessening ye plagues of London' estimated the cost-effectiveness of transporting people outside London for three months to be £84 for every £1 expended. We are in a dilemma because the compassionate physician and the citizen, when a patient, desire that everything be done and no expense spared to help the sick and suffering. On the other hand, the public who pay the bills through taxation or insurance demand that costs be contained.

There are those who would submit that cost considerations are unethical: E H Loewy has written: 'Of late an increasing number of papers in this and other journals have been concerned with "cost-effectiveness" of diagnostic and therapeutic procedures. Inherent in these articles is the view that choices will be predicted not only on the basis of strictly clinical considerations but also on the basis of economic considerations as they may affect the patient, the hospital, and society. It is my contention that such considerations are not germane to ethical medical practice, that they occupy space in journals that would be better occupied by substantive matter, and that they serve to orient physicians toward consideration of economics, which is not their legitimate problem . . . .

'It is incumbent on the physician (especially in a critical situation) to practice not "cost-effective" medicine but medicine that is as safe as possible for that patient under the particular circumstances. Optimization of survival and not optimization of cost effectiveness is the only ethical imperative. To select diagnostic tests on

the basis of cost effectiveness is a deliberate statistical gamble; to use diagnostic tests in an unthinking medical fashion is poor medicine, not because of cost but because unthinking medicine is dangerous for the patient. Ethical physicians do not base their practices on the patient's ability to pay or choose diagnostic and therapeutic procedures on the basis of their cost. It may be argued that the welfare of society is threatened by escalating medical costs; indeed, that argument at first appears to introduce a dilemma. Yet a large proportion of our ills are due to smoking, heavy drinking, and overeating, and the consequences of these indulgences consume a large portion of medical-care dollars. It is unfair to deprive those who have not been overindulgent of the best medical care while allowing the overindulgence of others to consume the available money, Furthermore, our society clearly has money to spend on luxuries and baubles. A physician who changes his or her way of practicing medicine because of cost rather than purely medical considerations has indeed embarked on the "slippery slope" of compromised ethics and waffled priorities'.[13]

This standpoint would be all very well if the citizen could determine the budget allocation to the NHS and had the ability to change behaviour. We, however, live in a different situation where neither is successful. To suggest withholding treatment from patients because of their misdemeanours is attractive but contrary to the basic ethos of doctoring. The medical profession in its more political mood tends to blame a lot of the financial problems on administration, where indeed there has been a great expansion that is of doubtful value. For example, the number of non-medical employees at regional, area and district level in England and Wales earning £5,000 per annum or more increased from 700 to 4,800 at the time of reorganisation in 1974.

There are obvious areas that contribute to the escalation in costs of the Health Service which are being addressed by the profession but much still needs to be done. The cost of drugs and appliances in England and Wales was £596 million 10 years ago, which exceeded the total cost of medical practitioners' salaries. Ten per cent of this went on slimming pills, tranquillisers and cough medicines, often prescribed because of patient pressure but probably totally ineffective, if not dangerous. New operations or techniques do not have to pass the Committee on Safety of Medicines; otherwise, for example, we would not have witnessed the adoption 15 years ago of gastric freezing for duodenal ulcer disease. It took seven years for properly controlled trials to show that it was useless. By that time £2·5 million had been spent on apparatus alone in the USA. We are just beginning to develop enough rigour in our practice properly to assess treatments, and in this the double blind clinical trial has been a milestone in methodological history.

QUALITY OF CARE

There is a new dimension which I believe needs to be brought onto the agenda — namely, quality of care. The supreme challenge today is to contain costs without lowering the quality of care. Some say this can't be done. We have already learnt that within limits this trade-off need not exist. In fact, the lower-cost way may be the high-quality way. A high volume of a product in a hospital not only yields economies of scale, it results in lower rates of post-surgical mortality and complications. We must grapple with the problem that the amount spent per case on a given condition often bears no relation to the outcome for the patient. What is quality care? Instinctively, I must suggest that it is about people. But quality in health care is an elusive entity and can mean all things to all observers. I believe

that it can be separated into numerous components. Some are measurable, some are not. The aspect which attracts most attention from economists and public policy makers has to do with technical, diagnostic and therapeutic procedures. We now live with performance indicators and norms for things like bed turnover and length of stay. This is all very impersonal and, while we strive for efficiency, we may further depersonalise the caring doctor/patient relationship. It is this aspect which is more difficult to study — the process of caring for the patient, the interpersonal, supportive and psychological aspects of the physician/patient relation — and which is the component of quality that most frequently separates the fulfilled physician, with a busy practice of satisfied patients, from others. It is the factor that gives rise to satisfaction on the part of the doctor and patient alike, accompanying and sometimes replacing the cure. There comes a point when increased efficiency or intensity of work becomes counterproductive in so human an activity as patient care, particularly in acute illness. The stress on staff can become intolerable if all the patients in the ward are seriously ill and morale may fall if patients are discharged so soon after treatment that their recovery cannot be appreciated by the staff. For patients and relatives the speed of passage may dehumanise their hospital experience, they have a sense of being processed. The physicians, having responded to the call for greater efficiency, are likely to end up being accused of not caring enough, of being concerned only with episodes of illness, and of having only a transitory relationship with patients. The quality of care as a measure in the doctor/patient relation is inversely proportional to the frequency of malpractice litigation. It is difficult to assess, most frequently ignored by health-care planners, economists, and theoreticians, and is not and cannot be addressed by mechanisms such as analysis of performance indicators. But it is not ignored by consumers, and is certainly the most visible, most easily perceived and most appreciated of the quality components by the patient. It is the loss of this component of quality that is most feared by doctors and by patients in the current social and economic struggles over the nature of the health care system of the future. Yet it is the most fragile and easily damaged characteristic of good medical care, and the characteristic that the medical profession, working with those responsible for formulating public policy, has the greatest responsibility to maintain.

Someone once said that variation in practice style is simply the evidence that good medical practice does not mean the same thing to different doctors! Nor, I would add, to different patients. And this is one very important reason that patients try to find a doctor who suits their own needs and style. Any system that truly aspires to high quality must accommodate personal preferences. They lie at the very heart of the doctor/patient relationship in an open society like ours. A purely data-driven approach to treatment that would take no account of patients' needs and wishes would indeed amount to cookbook medicine, and we would be right to reject it. We should put more, not less, emphasis on what patients expect as the outcome of their treatment.

In addition to introducing quality thinking as a dimension in our practice, we must also begin to understand the areas of real cost within the service. The public perception is that we spend excessively on technology. The finger is pointed at high cost techniques and procedures but they must be seen in perspective. It has been estimated that a 50% reduction in four major activities — CT scanning, renal dialysis, fetal monitoring and coronary artery bypass grafts — would save less than 1% of the medical budget. Far greater savings would be achieved if

doctors paid more attention to the wasteful use of apparently cheap and simple investigations. A study reported some years ago showed that the number of laboratory tests per case used in treating uncomplicated appendicitis rose from five in 1951 to 30 in 1971. Jan Brod wrote in 1977 about the explosion in the number of tests done on patients without any evidence of benefit in their management.[14] He estimated that biochemical screening of 200 consecutive medical outpatients had yielded no significant benefit but had cost DM12,400. You can find equally convincing reports on the wasteful use of X-rays, particularly of skull, spine and abdomen, for inadequate clinical reasons and for the geriatric 'ante-mortem' barium enema. One radiologist has entered the literature with the title *Department of inappropriate investigation.*[15] On the other hand, the CT scanner has been shown to be both accurate and cost-effective, particularly in neurological diagnosis. Balancing the cost of the machine and staff against the reduction in other invasive investigations such as arteriograms and air studies, and a reduction in waiting lists of patients, it has been shown that one unit saved £36,000 in one year.[16]

If we are to practise the best medicine for our patient and act as responsible custodians of the public purse we must look critically at how we use laboratory sciences, radiological tests and technical procedures. We are quick enough to develop and apply new techniques but not in any hurry to evaluate them rigorously; eager to add new procedures but slow to reject those of marginal value and sometimes reluctant even to eliminate those that are obsolete. We must maximise the important scientific advances for the benefit of our patients.

If we fail to use resources efficiently we will further deprive our society of health care. This is already seen as a result of political decisions but must not be compounded by professional profligacy. In the response to budget limits, we already see a perversion of provision. Lifesaving treatment is curtailed less than that which improves the quality of life. Because of the ease of deciding not to buy new equipment, as opposed to refusing to care for the sick, treatment dependent on costly equipment is being reduced more than that dependent on staff time and ordinary supplies. The special terror that cancer arouses means that treatment is curtailed relatively little. New treatments are slower in being developed since they have to compete with established services for available resources. Diagnostic procedures about which the public know little are supplied less than those about which the public are better informed. There is development of crisis management rather than planning. If we are to be serving both our patients and society we must engage in this dialogue.

In introducing another teaching session this morning my hope for our students is that they will acquire the knowledge and skills needed for the highest standards of medical practice. For, after all, the greatest unkindness to our patients is medical incompetence. I trust that they will also develop attitudes which will be responsive to the demands of both the individual patient and society. I wish you all success for this demanding but, I believe, exciting and rewarding future.

REFERENCES

1. Rhodes P. An outline history of medicine. London: Butterworth, 1985: 14
2. Silverman WA. The public character of scientific medicine. *Perspect Biol Med* 1983; **26**: 345. (Quotation from HW Haggard, *Devils, drugs and doctors.* New York: Harper, 1929).
3. Thomas L. The future of medicine. In: Austyn JM, ed. New prospects for medicine. Oxford: Oxford University Press, 1988: 119.
4. Newman G. Thomas Sydenham, reformer of English medicine. London: The British Periodicals, 1924.
5. Black DAK. The logic of medicine. Edinburgh: Oliver & Boyd, 1968.
6. Kennedy I. The unmasking of medicine. London: Allen & Unwin, 1981: 26-50.
7. Shaw GB. The doctor's dilemma. London: Constable, 1911: xxii.
8. Black DAK. An anthology of false antitheses. London: Nuffield Provincial Hospitals Trust, 1984: 17-30.
9. Royal Commission on the National Health Service. Report. London: HMSO, 1979: 51-70.
10. Health and health policy — priorities for research. Report to the Research Initiatives Board. London: Social Science Research Council, 1977.
11. Muir Gray JA. Four box health care: development in a time of zero growth. *Lancet* 1983; **2**: 1185-6.
12. Active health report — perspectives on Canada's health promotion survey. Ottawa: Department of National Health and Welfare, 1987.
13. Loewy EH. Cost should not be a factor in medical care. (Letter). *N Engl J Med* 1980; **302**: 697.
14. Brod J. The rational basis of diagnosis in internal medicine. *J R Coll Physicians Lond* 1977; **11**: 323-34.
15. Goldberg B. Department of inappropriate investigations. *Br Med J* 1977; **2**: 1273-5.
16. Thomson JLG. Cost effectiveness of an EMI brain scanner. A review of a 2-year experience. *Health Trends* 1977; **9**: 16-9.

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Primary medical care in a paediatric accident and emergency department

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SUMMARY

The characteristics of a random sample of 853 children who attended the accident and emergency department of the Royal Belfast Hospital for Sick Children were studied prospectively to determine the extent to which the department was being used to provide primary medical care. Direct parent referrals accounted for 69% of all attendances with a further 21% referred by the family doctor. Parental preference and accessibility were the main reasons given for choosing to attend the department with the latter significantly higher among out-of-hours attendances. However, only 37 of the 585 parent referrals had made an attempt to contact the family doctor.

Overall, 33·9% of children were felt to be inappropriate attenders, i.e. were neither accident nor emergency cases, and the proportion was highest among the parent-referred groups.

The present financial restraints facing the National Health Service make it uneconomical to provide primary medical care at both hospital and community level. However, the level of demand for the accident and emergency department, together with the attitudes of those who attend, make it unlikely that a more rational use of resources will be achieved in the foreseeable future.

INTRODUCTION

It has been estimated that approximately 2,000,000 children attend accident and emergency departments throughout the United Kingdom each year.[1] Although the characteristics of adults who present in this way have been scrutinised extensively since the 1950s, there is little comparable information about paediatric patients. In the United States, the recognition of the increasingly widespread demands for paediatric 'emergency rooms' has led to extensive research into the services which they provide and the characteristics of the families who use them.[2, 3, 4, 5] The Royal Belfast Hospital for Sick Children has the only specific paediatric accident and emergency department in Northern Ireland. The district around the hospital (North and West Belfast) has a total childhood population of 62,456. It is typical of many inner city areas with high unemployment, an over-representation of social classes IV and V, and a considerable proportion of single-parent families.

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A prospective study of the medical and social characteristics of a random sample of 22,617 children who presented at the accident and emergency department over a 12-month period from 2 December 1984 was undertaken. One of the objectives of the study was to assess the extent to which the department was being used to provide primary medical care and to determine the possible reasons.

MATERIALS AND METHODS

Data relating to new attendances at the accident and emergency department over the preceding five years were used to predict the number of children who might be expected to attend during the study. It was estimated that a random sample of 862 patients (4% of the predicted attendances) would provide statistically valid data. From 2 December 1984, patients were allocated a sequential research number on arrival at the department. Study patients were therefore easily identified. A questionnaire for each selected patient was completed by the first author with the consent and co-operation of the parents. The parents of children who attended when she was unavailable were informed of the study by the nursing staff, and consent obtained for subsequent contact at home by the same researcher.

Information about the child's social background, past medical history and the present attendance was included in the questionnaire. Each attendance was assessed as appropriate or inappropriate, using criteria given in Table I. Where there was difficulty in assigning an attendance to either the appropriate or inappropriate group the decision was made jointly with two consultant paediatricians, borderline cases being placed in the 'appropriate' category.

TABLE I

Appropriate and inappropriate attendances at the accident and emergency department

Appropriate		
Accidents	— injuries	
	— wounds	
	— poisoning	
Emergencies	— acute medical and surgical conditions referred by the family doctor (appendicitis, limp, meningitis, diabetes, etc.)	
	— respiratory difficulty	
	— altered level of consciousness	
	— non-accidental injury	
Inappropriate		
Family doctor unavailable		
Parent preference for the accident and emergency department		
Short-cut to out-patient clinics		
Family doctor referrals	(i) without examination of the patient	
	(ii) without contacting the accident and emergency department	
Rashes of long duration		
Duration of symptoms greater than 24 hours		
Infectious diseases referred by the family doctor. (These should go direct to the Northern Ireland Fever Hospital since no isolation facilities exist at the Children's Hospital).		

RESULTS

A random sample of 862 attendances was studied. The parents of two children refused to participate in the study; another three patients were excluded, being hospital staff, and a further four children arrived, were recorded in the attendance book but did not wait to be medically examined. The characteristics of the remaining 853 children were analysed.

The majority of children were referred by the family doctor or by their parents (Table II). Overall, more than two-thirds (68·6%) of the children were parent referrals arriving without any prior direct contact with primary medical care. In only five cases was this because the child was not registered with a family doctor. During the night, family doctor referrals accounted for only 8·8% of the children who attended, compared with 25·0% of day attendances and 18·2% of evening attendances. Conversely significantly ($p < 0·01$) more attendances from 5.00 pm to 9.00 am were direct parent referrals. The remaining patients were referred by other hospitals (2·2%), community medical officers (1·3%), or other sources such as health visitors, pharmacists etc (6·6%).

TABLE II

Time of arrival of patients attending the accident and emergency department analysed by source of referral

Source of referral	*Time of arrival*			
	9 am–5 pm	*5 pm–1 am*	*1 am–9 am*	*Total*
Family doctor	110 (25·0%)	69 (18·2%)	3 (8·8%)	182 (21·3%)
Parent	282 (64·1%)	274 (72·3%)	29 (85·3%)	585 (68·6%)
Other	48 (10·9%)	36 (9·5%)	2 (5·9%)	86 (10·1%)
Total	440	379	34	853

($X^2 = 10·18$, DF = 2, $p < 0·01$ between family doctor and parent referrals)

One-third (33·9%) of all attendances were considered to be inappropriate (Table III). Although significantly more parent referrals ($p < 0·05$) fell into this category, 28% of family doctor referrals were also thought to be inappropriate.

TABLE III

Number of appropriate and inappropriate attendances analysed by source of referral

	Source of referral			
	Family doctor	*Parent*	*Other*	*Total*
Appropriate	131 (72%)	374 (64%)	67 (78%)	572 (67%)
Inappropriate	51 (28%)	211 (36%)	19 (22%)	281 (33%)
Total	182	585	86	853

($X^2 = 4·00$, DF = 1, $p < 0·05$ between family doctor and parent referrals)

Parents of the children brought directly to the hospital were asked to say, in their own words, why they chose to attend the accident and emergency department rather than consult their family doctor. Overall, 20·9% did so because they thought the child would be referred to hospital by the family doctor, but the remaining 79·1% were brought for reasons other than a perceived need or likelihood of hospital referral. The answers given by parents varied significantly ($p < 0·05$), with the time of day, the accessibility of the hospital services accounting for a larger proportion of attendances between 5.00 pm and 9.00 am (39·3% compared with 31·6%) (Table IV).

TABLE IV

Reasons given by parents for choosing the accident and emergency department, by time of day

	Time of arrival			
	1 am–9 am	*9 am–5 pm*	*5 pm–1 am*	*Total*
Anticipated referral	2 (6·9%)	56 (19·9%)	64 (23·4%)	122 (20·9%)
Better treatment at hospital	1 (3·5%)	55 (19·9%)	39 (14·2%)	95 (16·2%)
Always come to hospital	4 (13·8%)	35 (12·4%)	28 (10·2%)	67 (11·5%)
Wanted second opinion	4 (13·8%)	22 (7·8%)	12 (4·4%)	38 (6·5%)
Hospital more convenient	1 (13·5%)	29 (10·3%)	30 (10·9%)	60 (10·3%)
Too long to wait for family doctor	3 (10·3%)	31 (11·0%)	22 (8·0%)	56 (9·6%)
Too difficult to contact family doctor	5 (17·2%)	13 (4·6%)	15 (5·5%)	33 (5·6%)
Hospital always open	2 (6·9%)	9 (3·2%)	14 (5·1%)	25 (4·3%)
Did not want deputising bureau doctor	1 (3·5%)	7 (2·5%)	26 (9·5%)	34 (5·9%)
Patient attending for this condition	6 (20·7%)	19 (6·7%)	19 (6·9%)	44 (7·5%)
Patient attending hospital for other condition	0	3 (1·1%)	1 (0·4%)	4 (0·7%)
Missing information	0	3 (1·1%)	4 (1·5%)	7 (1·2%)
Total	29	282	274	585

($X^2 = 9·62$, DF = 4, $p < 0·05$ between parental preference, accessibility and miscellaneous group)

Of the 122 children whose parents had anticipated referral, 68 (55·7%) did not require a specific hospital resource (admission, X-ray, plaster of Paris or emergency treatment) compared with 59·4% of all other attendances ($p > 0·1$, NS). In only 37 (6·3%) of the 585 cases where children were parent referrals had attempts been made to contact the family doctor. While significantly more ($p < 0·001$) parents who cited the main reason for direct attendance as difficulty in contacting their family doctor, or the delay in obtaining a consultation, had attempted to contact the doctor (25·8% compared with 2·8% of those citing other reasons) three-quarters (74·2%) of these parents had not made any attempt to make such contact (Table V).

DISCUSSION

The main function of an accident and emergency department should be, as the name suggests, the treatment of accident and emegency cases. However, it is well recognised that a considerable proportion of patients who attend, especially in urban areas, do so to obtain primary medical care. In studies of adults, the reported level of self-referral varies from 43%[6] to 78%.[7] Recognised factors influencing the choice of an accident and emergency attendance compared with

TABLE V

Relationship of attempted family doctor contact to the reason for parental referral to the accident and emergency department

	Reason for choosing the accident and emergency department		
Attempted to contact family doctor	*Inaccessibility* of family doctor*	*Other*	*Total*
Yes	23 (25·8%)	14 (2·8%)	37 (6·3%)
No	66 (74·2%)	481 (97%)	547 (93·5%)
Not known	0	1 (0·2%)	1 (0·2%)
Total	89	496	585

($X^2 = 67·33$, DF = 1, $p < 0·001$)

*Includes 'too long to wait' and 'too difficult to contact'.

a family doctor consultation include the perception of the role[8] and the accessibility of the department.[9] An earlier study carried out in Belfast by the Queen's University Department of General Practice found that the patient's perception of his illness and the treatment required also affected this decision.[10] The importance of accident and emergency departments as an unrestricted source of medical care for children is widely accepted. However, inappropriate utilisation diverts resources from those children who do require the facilities, and has disadvantages both for the child and the family doctor in terms of continuity of care.

It has been estimated that between 20% and 25% of the childhood population attend an accident and emergency department each year.[11] During the 12 months of the study, 22,617 children attended the accident and emergency department of the Royal Belfast Hospital for Sick Children, i.e. more than one-third of the 62,456 children who live in North and West Belfast. The recent trend in accident and emergency attendances in this hospital is upwards, with additional demands on staff and facilities. At a time of financial restriction it is a questionable use of resources to provide primary health care at both community and hospital level. In this study, more parent-referred children were considered inappropriate attenders, and, since this group was more than three times the size of the group referred by the family doctor, its contribution to the inappropriate use of the department was correspondingly greater.

An understanding of the factors influencing the parent-referred group in their choice might enable changes to be made in referral patterns. Parents who express a preference for a particular service may do so for one of two reasons. First, they have had previous contact both with the accident and emergency department and with other forms of primary care, and found the former more acceptable for a variety of reasons: included in this group are parents who say they always come to the hospital when their children are sick. This suggests a regular pattern of behaviour rather than a conscious preference but does imply that at an earlier stage the choice was made to use the hospital as a source of primary medical care. Second, parents may have made assumptions about the nature of investigations or treatment required, concluded that hospital facilities would be needed and therefore bypassed primary care. The largest single reason given by

parents for choosing the accident and emergency department was that the child would subsequently require referral to hospital. However, no more of this group did in fact receive a specific hospital service than those children in whom the parents did not anticipate referral. This suggests that although parents may be best at knowing when their child is sick[12] they are less proficient in determining the nature of the medical resources required and consequently the most appropriate place to find medical care. The greater proportion of direct parent referrals in the evening than at night is in keeping with the finding that more parents gave 'accessibility' as their reason at these times. Many parents were unaware that family doctors have 24-hour responsibility for their patients. Deputising services were also considered by some parents to be unacceptable substitutes for a family doctor consultation.

It may well be that there are actual as well as perceived problems in obtaining a consultation with a family doctor. A recent health profile carried out in one housing estate (Moyard), within the North and West Belfast district, found that there were no doctors' surgeries within the estate, and very limited telephone facilities.[13] Some parents may well find it more convenient to attend the hospital, rather than to contact their family doctor to arrange a visit or to make a future appointment. Even so, the finding that more than half of the parent-referred group did have their own telephone makes it unlikely that providing every home with a telephone would dramatically alter the pattern of referrals to hospital. Inappropriate utilisation is a difficult problem to resolve. The education of parents in the correct role of the accident and emergency department, and the co-operation of family doctors in improving their patients' knowledge about the nature and extent of the services which they provide, might reduce the number of direct parent referrals. However, the larger group who prefer to attend hospital are unlikely to have their attitudes changed, at least in the short term, by such measures. The real alternative to dissuading children from attending an inner city accident and emergency department may be to realise the demand for this form of health care and to allow for it when planning the best use of available hospital resources.

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REFERENCES

1. Jackson RH. Children in accident and emergency departments. *Br Med J* 1985; **291**: 991-2.
2. Bergman AB, Haggerty RJ. The emergency clinic. *Am J Dis Child* 1962; **104**: 36-44.
3. Halperin R, Meyers AR, Alpert JJ. Utilisation of pediatric emergency services. *Pediatr Clin North Am* 1979; **26**: 747-57.
4. Kahn L, Anderson M, Perkoff GT. Patients' perceptions and uses of pediatric emergency room. *Soc Sci Med* 1973; **7**: 155-60.
5. Wingert WA, Friedman DB, Larson WR. Pediatric emergency room patient. *Am J Dis Child* 1968; **115**: 48-56.
6. Crombie DL. A casualty survey. *J Coll Gen Pract* 1959; **2**: 346-56.
7. Morgan W, Walker JH, Holohan AM, Russell IT. Casual attenders: a socio-medical study of patients attending accident and emergency departments in Newcastle upon Tyne area. *Hosp Health Serv Rev* 1974; **70**: 189-94.

8. Davies T. Accident department or general practice? *Br Med J* 1986; **292**: 241-3.
9. Wilkinson A, Kazantzis G, Williams DJ, Dewar RAD, Bristow KM, Miller DL. Attendance at a London casualty department. *J R Coll Gen Pract* 1977; **27**: 727-33.
10. Reilly PM. Primary care and accident and emergency departments in an urban area. *J R Coll Gen Pract* 1981; **31**: 223-30.
11. Wilson DH. The epidemiology of childhood accidents. In: Proceedings of the Symposium on Accidents in Childhood. London: Child Accident Prevention Trust, 1985. (Occasional paper No 7).
12. Spencer NJ. Parents' recognition of the ill child. *Prog Child Health* 1984; **1**: 100-12.
13. Moyard Health Survey Group. Moyard, a health profile. Belfast: Eastern Health and Social Services Board, 1985; 34.

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An outbreak of salmonellosis amongst holidaymakers in Madeira, July 1988

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SUMMARY

In July 1988, 20 of 49 tourists from Northern Ireland became ill with gastrointestinal symptoms while on holiday in Madeira and four were admitted to hospital. Salmonella enteritidis *was subsequently isolated from eight members of the party. Epidemiological investigations implicated fried and/or scrambled eggs as the vehicle of infection*

INTRODUCTION

On 12 July, 49 people from Northern Ireland went on a package holiday via Gatwick to the same hotel in Madeira. On Sunday 24 July, approximately 70 hotel guests of varying nationalities became ill with abdominal cramps, vomiting and diarrhoea. At least 15 guests, including one from Northern Ireland, were admitted to the local hospital. Of the 48 holidaymakers who returned home to Belfast on 28 July three had severe gastrointestinal symptoms and had been advised by a doctor in Madeira 'to go straight to hospital' on arrival home. The sick passengers informed the flight crew on the Gatwick/Belfast flight of their illness. Therefore when the aeroplane landed it was met by a community physician and an environmental health officer. They arranged the transfer of the three passengers with severe gastrointestinal symptoms to Belvoir Park Hospital. In addition, an investigation was initiated to document the nature and extent of the outbreak as well as the vehicle of infection.

METHODS

Epidemiological

The three patients in hospital were visited by a community physician, and a detailed clinical and food history was elicited. The party leader was also visited and this combined information was used to construct a postal questionnaire which was sent to each member of the party. This sought information on symptoms arising while abroad, the hotel restaurant used, and details of food eaten in the hotel between 22 and 24 July. A case was defined as a person who

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had vomiting and/or diarrhoea associated with abdominal pain while staying in that hotel in Madeira. The hotel management was requested to supply details of all food served in the 72 hours preceding Sunday 24 July, but unfortunately this information did not become available until after the questionnaires had been distributed.

Microbiological

The names and addresses of the holidaymakers in this party were obtained at the airport. Home vists and the collection of faecal specimens were arranged by the Area Departments of Community Medicine.

Environmental

Information on the hotel and its menu were obtained from patients and the party leader.

RESULTS

Epidemiological

A total of 43 (88%) questionnaires were returned. Twenty (47%) admitted to having had some form of stomach upset while abroad; 19 had diarrhoea, 12 had abdominal pain, nine had nausea, seven had vomiting and five reported fever. Only 14 replies met the case definition. The cases were aged between 36 and 72 years (mean 60·3 years). The dates of onset of symptoms ranged from 18 to 25 July with a peak on 24 July. Of the 14 cases, nine had consulted a doctor while abroad, six received antibiotics and four were admitted to hospital with a length of stay of 8–13 days.

Food-specific attack rates based on food consumed between 22 and 24 July were calculated (Table). A statistically significant association was demonstrated between gastrointestinal illness and the consumption of fried and/or scrambled egg. No significant association was detected between illness and eating boiled eggs or omelette. Three cases developed symptoms before 22 July and these were excluded from the analysis. Gastrointestinal illness in the Northern Ireland party was not significantly associated with the use of any particular restaurant in the hotel.

TABLE

Food-specific attack rates for food eaten 22 – 24 July

	Ate			*Did not eat*			
Food	*Ill*	*Not ill*	*Attack rate %*	*Ill*	*Not ill*	*Attack rate %*	*p*
Fried egg	7	5	58	0	15	0	0·0009
Boiled egg	4	6	40	1	14	7	NS
Scrambled egg	9	5	64	0	16	0	0·0001
Omelette	7	11	39	2	11	15	NS
Mousse	3	8	27	4	10	29	NS
Chicken (cold)	4	14	22	3	6	33	NS
Chicken (hot)	4	18	18	2	2	50	NS

Microbiological

Faecal samples were obtained from 20 people who had been symptomatic and *S enteritidis* was isolated from eight. Of these all met the clinical case definition and included the four patients who were admitted to hospital. Five of the eight positive samples were subsequently phage-typed; four were phage type PT4 and the other PT7.

Environmental

The hotel had accommodation for more than 500 people. The party from Northern Ireland stayed on a half-board basis and tended to lunch away from the hotel. There were three main restaurants in the hotel but food was also served at some of the bars. The menu rotated on a fortnightly basis with a choice of about four foods per course. Eggs were always on the breakfast menu.

The outbreak was investigated by Madeira public health officials who visited the hotel on Monday 25 July. They interviewed cases and various food samples, including eggs, were obtained for analysis. Salmonella was not isolated from these samples. However, *Salmonella* group D was isolated from stool samples of some of the affected guests. Information on kitchen facilities and hygiene was not available. The local investigators suspected scrambled eggs as being the cause of the outbreak but this was not proven.

DISCUSSION

It is difficult to investigate an outbreak of food poisoning occurring in another country, but this incident provides an opportunity to document morbidity amongst a group of returning holidaymakers. This has been performed elsewhere, notably in Glasgow where 45% of passengers at Glasgow airport returning from package holidays stated that they had been ill while on holiday, or shortly after their return, gastrointestinal symptoms accounting for most of all illnesses.[1] Illness on holiday is common and, with the continued growth in air travel, particularly the number of holiday destinations served by Belfast International Airport, it becomes even more pertinent to enquire about a person's travel history.

The return flight was via Gatwick and although three passengers were obviously distressed, neither they nor their friends mentioned their condition to the cabin crew on the Madeira – Gatwick flight. Had they done so, it is likely that they would have been detained at Gatwick and medical attention sought, but they chose to wait until they were approaching Belfast. This aspect of human nature would have had more important implications for communicable disease control had they been suffering from a more serious communicable disease, or if it had been a long-haul flight. Modern aeroplanes can accommodate up to 500 passengers and some non-stop intercontinental flights may last 12 hours or more. If some passengers have an acute diarrhoeal illness it can impose enormous strain on sanitary facilities, making personal hygiene less easy and increasing the potential for person-to-person spread.

Although the hotel was asked to forward details of all food served in the hotel on 22 – 24 July the information was incomplete since it did not include breakfast items or food served at the numerous bars in the hotel. Thus it was impossible to elicit from the menu the exact nature of some of the food served and it provided little additional information to that already obtained from detailed case histories.

The epidemiological investigation showed an association between illness and the consumption of fried and/or scrambled eggs and the microbiological investigation

confirmed the organism as *S enteritidis* PT4 and PT7. In this outbreak a narrow clinical case definition was used to exclude the minor gastrointestinal illnesses which occur abroad. Nineteen (44%) of the passengers had diarrhoea while in Madeira. Holidaymakers involved in this epidemic appeared to have a severe infection as a high proportion were admitted to hospital.

Salmonella enteritidis was isolated from eight cases, all of whom had eaten scrambled egg in the three days prior to illness, and seven stated that they had definitely or probably eaten fried egg during the same period. Some of the party commented that the scrambled egg was often cold or lukewarm. However, their comments may have been biased following remarks made by a local doctor implicating scrambled egg. Approximately six weeks after this outbreak, two English tourists staying in the same hotel became unwell and *S enteritidis* was subsequently isolated from them.

This investigation again highlights the association between *S enteritidis* and eggs.[2] Although the public in the United Kingdom has been advised to eat well-cooked eggs, it may be prudent to extend this advice to those who holiday abroad. This study illustrates the methodology used in the investigation of an outbreak of gastrointestinal illness and also demonstrates the close co-operation that exists between community physicians, laboratories, clinicians and environmental health officers.

The authors wish to thank Dr D Canavan for permission to interview patients and examine hospital records; also environmental health officers in the various local authorities and colleagues who assisted in this investigation.

REFERENCES

1. Reid D, Dewar RD, Fallon RJ, Cossar JH, Grist NR. Infection and travel: the experience of package tourists and other travellers. *J Infect* 1980; **2**: 365-70.
2. Anonymous. Salmonella infection (editorial). *Lancet* 1988; **2**: 720-2.

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Perinatal mortality in Northern Ireland: where are we now?

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SUMMARY

Perinatal mortality in Northern Ireland has been declining over the last 30 years, but the factors which may account for this fall have not been clearly delineated. Crude perinatal mortality figures yield very little insight into the problem, and meaningful management statistics are urgently required if service performance is to be reasonably assessed. This paper sets out the case for birth-weight standardisation and explores the utility of a broad diagnostic taxonomy of causes of death.

INTRODUCTION

Since the early part of this century there has been a dramatic decline in perinatal mortality in this country. Over the last 25–30 years, crude perinatal death rates have fallen from 38 to 11 per 1,000 total births. The percentage improvement in Northern Ireland is higher over this period than in the other home countries, but there is debate about the factors to which this can be attributed (Fig 1).

Many perinatal deaths are determined by circumstances and events surrounding birth, and the perinatal mortality rate is widely regarded as a performance

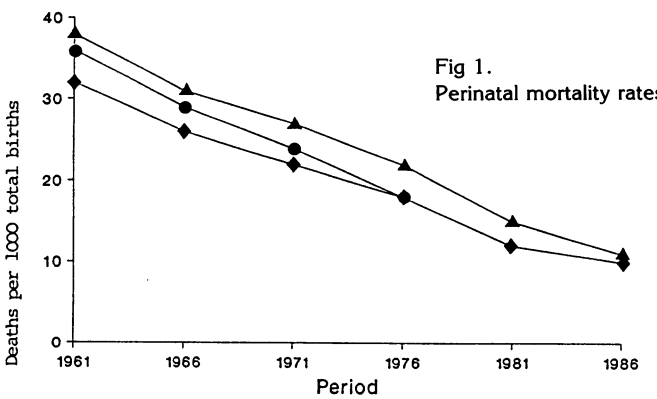

Fig 1.
Perinatal mortality rates for home countries.

Figure 1: The crude perinatal mortality rates for Northern Ireland ▲ Scotland ● , and England and Wales ◆ from 1961 to 1986.

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indicator for this aspect of the health services.[1-3] Health planners and other professionals, to say nothing of the public at large, have not been universally convinced that there are sufficient advantages in 'high technology' medicine in this area to compensate for the high cost, denial of free choice to mothers, and the possible impairment of development of mother-infant relationships. In contrast, the relationships between socio-economic status, low birthweight and mortality are consistently demonstrated[4] and have prompted some to recommend a closer liaison between social and medical interventions.[5-6]

Crude comparisons of overall perinatal mortality have very limited value in appraising the role of regional neonatal intensive care or the place of obstetric intervention in preterm labour, and there seems to be a consensus on the need for better indices of perinatal health. The routinely available statistics for Northern Ireland, which provide only crude death rates, contribute little to our understanding of how the prospects for low birthweight infants have changed in recent years. In view of the caution with which other crude mortality data are generally treated both by clinicians and epidemiologists, it is surprising how often circumspection is discarded when perinatal mortality rates are being compared.

One way of overcoming some of these criticisms is to use standardised perinatal mortality rates, analogous to the standardised mortality ratio, except that the standardisation is carried out according to the birthweight distribution instead of in terms of sex and age.[7, 8] It is clear that the major hope for improving perinatal mortality must depend upon measures which will shift the birthweight distribution to the right, whether through social or health care mechanisms, and, if we are to carry out realistic assessments of other components of care, then it is necessary to eliminate from the comparisons the effects of the birthweight distribution.[8]

A supplementary approach, as suggested by Wigglesworth, is to examine causes or modes of death within the various birthweight groups, as far as possible, selecting mutually exclusive groups of causes which carry implications for perinatal care.[9] He has suggested that the most pragmatic classification is that based on the following simplified pathological subgroupings to which most perinatal deaths can be provisionally assigned even if necropsy is not done:

1. Normally formed, macerated (stillbirth).
2. Congenital malformations (stillbirth or neonatal death).
3. Conditions associated with immaturity (neonatal death).
4. Asphyxial conditions developing in labour (fresh stillbirth or neonatal death).
5. Specific conditions other than above.

This paper explores the utility of such indicators in the Northern Ireland setting.

METHODS

The distribution of birthweights for all births in the province for the years 1984–86, and for the Northern Health and Social Services Board for the years 1976–78, was obtained from the Information Technology Unit of the Department of Health and Social Services, Northern Ireland. Birthweight was obtainable for 97·5% and 96·2% of all births during the respective periods. Birthweights were grouped into five categories; 0–999g, 1000–1499g, 1500–1999g, 2000–2499g and 2500g and greater. Calculation of the standardised perinatal mortality rate uses the technique of indirect standardisation, whereby perinatal mortality rates within specific weight bands in a reference population (Northern Ireland) are multiplied by the absolute numbers

of births in the same weight groups in a local population. The expected numbers within successive weight groups are added together to give an expected total. Finally, the observed total number of deaths and stillbirths is divided by the expected number and the result multiplied by 100.

Each health board receives a copy of the medical certificate of cause of death which enables an entry to be made in the birth register for each perinatal death. Wigglesworth's four main groups of causes of deaths present several minor problems of definition.[9] In some stillbirths with early maceration it may be difficult to decide whether death occurred before or after the onset of labour. Many congenital malformations which are fatal in the perinatal period are recognisable externally or clinically, but the total number of malformations may be underestimated unless post-mortem examination is performed. All normally formed fresh stillbirths of any birthweight and all early neonatal deaths in term infants are assigned to the asphyxial group, unless a specific condition, such as an inborn error of metabolism, has been diagnosed during life or at necropsy. Each perinatal death has been assigned to the place of residence of the mother.

RESULTS

The Table illustrates the crude and birthweight standardised perinatal mortality rates for the four health boards. Fig 2 elaborates this data by representing the contribution of each of Wigglesworth's cause of death groupings to the overall birthweight standardised mortality.

TABLE

Crude and birthweight standardised perinatal mortality rates (1984–1986) for the four health boards (weighed births only)

Board	*Crude rate per 1,000 total births*	*Birthweight standardised rate*	*(95% confidence limits)*
Eastern	11·45	99·41	(89–110)
Northern	10·78	90·78	(78–104)
Southern	13·23	115·98	(99–133)
Western	10·97	96·73	(82–112)

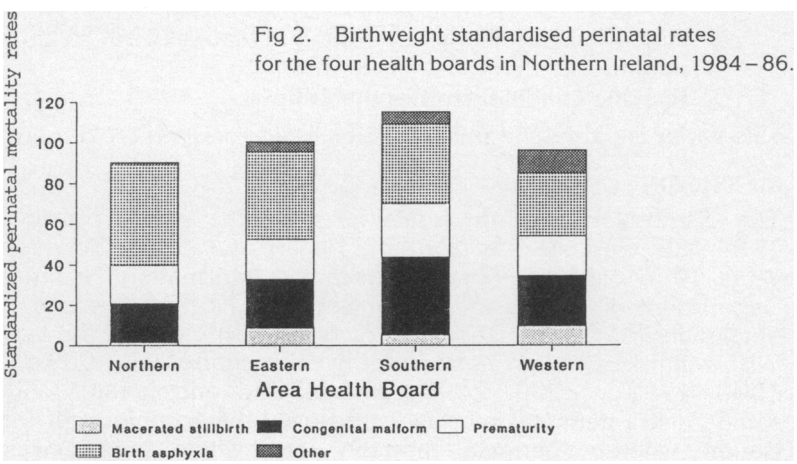

Fig 2. Birthweight standardised perinatal rates for the four health boards in Northern Ireland, 1984–86.

Fig 3 illustrates how the Northern Board has reached its present position since the earlier period of 1976–78. The weight-specific mortality rates of 1984–86 have been used as the standard for both periods shown. The fall in the standardised perinatal mortality rate has arisen from declines in all groups of causes.

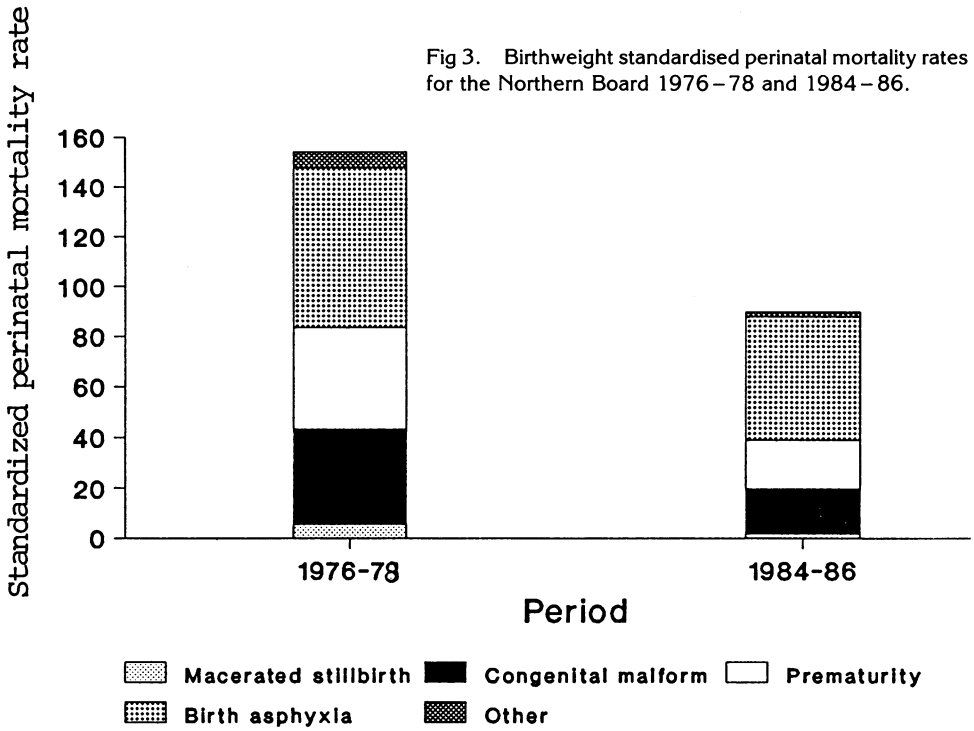

Fig 3. Birthweight standardised perinatal mortality rates for the Northern Board 1976–78 and 1984–86.

DISCUSSION

It is now generally accepted that, while crude perinatal mortality rates have been widely used as indicators of the quality of obstetric and early neonatal care, they provide little insight into the varying mortality experience across the birthweight distribution.[3] When different populations are being compared, birthweight standardisation can reduce the breadth of the distribution and consequently the chance of an area being labelled as aberrant.[7] There remain problems arising from small numbers and sampling variation, and all presentations relating to perinatal mortality should be accompanied by confidence limits.[10]

The statistical stability of small area rates can be enhanced by aggregating deaths over a number of years. and it is possible that, for small areas of the province like council districts or units of management, a three-year grouping is not sufficient. The confidence limits for some of these areas are wide but there is a real difficulty that aggregation over longer periods (five or six years) would mask the significant effects of other determinants of birthweight which could change during such an interval. An example might be the social class distribution — in the Northern Board, for 1976, 4·1% of social class I and II births were of low birthweight (<2500g) as compared with 6·3 of social class IV and V births. Although these percentages changed little for the later period (1984–86), the proportion of all births in social classes IV and V fell from 27·8% to 13·6% during the interval.

Gestational age is, of course, a further independent variable against which both birthweight and perinatal mortality might be assessed. However, because it is notoriously inaccurate in a substantial proportion of cases,[11] further standardisation by this variable was not thought worthwhile.

The purpose of breaking down the weight-standardised mortality into Wigglesworth's cause of death groupings was as far as possible to recognise groups with 'clear implications for clinical management'.[9] For instance, he suggests that a high rate of macerated stillbirths should prompt investigation related to antenatal care and background maternal factors. The frequency of fatal asphyxial conditions arising during labour may reflect provision of facilities for intra-partum monitoring or the availability of personnel trained in the resuscitation of the newborn. A high congenital malformation rate may raise questions about facilities for pre-natal screening or procedures for early neonatal diagnosis of potentially treatable conditions (such as transposition of the great vessels).

There are perhaps more problems of definition than Wigglesworth suggested in the use of his five broad categories. The quality of notifications of congenital malformations suggests considerable unreliability.[12] When looking for secular trends in perinatal mortality using a Wigglesworth type of taxonomy, the position is complicated because modern treatment attempts the salvage of an ever wider spectrum of conditions, such that a stable definition of 'lethal malformation' is elusive. There is the added difficulty that premature delivery itself can cause in this respect — certain conditions, such as a diaphragmatic hernia (which covers a wide spectrum of severity) could be labelled either as a developmental abnormality and thus be coded as a death attributable to prematurity, or as a malformation.

These caveats aside, it has been suggested that there may well be a limit to the degree by which perinatal mortality can be further reduced — a limit imposed by the 'unavoidable causes of death', predominantly congenital malformations. However, the experience of the Northern Board, which has shown a marked improvement over the last 10 years, would suggest that this improvement has not just been limited to those causes related to asphyxia and prematurity. During this interval, the Northern Board introduced a neonatal intensive care service, but a causal link with the improved outcome is not necessarily proven. When such patterns are investigated in future and districts are compared, a more detailed review of weight-specific mortality trends might be useful. The limited explanatory power of ordinary standardised mortality ratios has already been noted,[13] and so weight-specific mortality data may prove a useful supplement when districts are compared and when trends are sought. Although all weight groups in the Northern Board showed some improvement the reduction in mortality was greatest (70%) for babies between 2000 and 2499 g.

There can, however, be no grounds for complacency. Several groups have shown that the rapid decline in perinatal mortality over the last 20 or 30 years has not been matched by a proportionate decrease in post-neonatal deaths and have postulated that this may be due to a postponement of some neonatal deaths into the post-neonatal period.[14, 15] This hypothesis is supported by the anomalous pattern of unchanging (or increasing) post-neonatal mortality rates, which is more marked for very low birthweight babies.[14] Although this would merit further investigation in Northern Ireland, perhaps we should also re-examine the objectives of perinatal care. Mitchell has submitted that the prime objective should be 'to ensure that new individuals reach adult life in the best possible state' and that the final outcome is determined by many interacting factors 'from genetic

endowment to educational achievement, but above all by the qualities of the parents, eugenically before conception, physiologically during pregnancy and personally after birth'.[6] While prevention of low birthweight must be a prime objective, there is a growing belief that, to provide the best future for infants at risk, attention must be paid to the qualitative aspects of their childhood as well as to the standards of care at birth.

In our continued quest for better performance indicators it is essential to avoid a blinkered view, and also to appreciate the shortcomings of small area ecological analysis. While there may be significant associations between variables like the social class, maternal age or parity distributions of an area, and its perinatal mortality rates, such correlations provide little clarification of the processes involved at the individual level. We suggest that birthweight standardisation at least should be a first step towards fairer comparisons. Although it has some obvious shortcomings, not least a reliance on death certification,[16] the Wigglesworth taxonomy of perinatal mortality might provide a useful management tool to point the way to more detailed investigation.

REFERENCES

1. Black N, Macfarlane A. Methodological kit; monitoring perinatal mortality statistics in a health district. *Community Med* 1982; **4**: 25-33.
2. Alberman E. Prospects for better perinatal health. *Lancet* 1980; **1**: 89-92.
3. Knox EG, Lancashire R, Armstrong EH. Perinatal mortality standards: construction and uses of a health care performance indicator. *J Epidemiol Community Health* 1986; **40**: 193-204.
4. Chalmers I. Short, Black, Baird, Himsworth and social class differences in fetal and neonatal mortality rates. *Br Med J* 1985; **291**: 231-2.
5. Sunderland R, Greenfield AA. Declining mortality in the immature: medical or biologic effect? *Epidemiol Community Health* 1984; **38**: 326-30.
6. Mitchell RG. Objectives and outcome of perinatal care. *Lancet* 1985; **2**: 931-3.
7. Mallett R, Knox EG. Standardised perinatal mortality ratios: technique, utility and interpretation. *Community Med* 1979; **1**: 6-13.
8. Knox EG, Marshall I, Kane S, Green A, Mallett R. Social and health care determinants of area variation in perinatal mortality. *Community Med* 1980; **2**: 282-90.
9. Wigglesworth JS. Monitoring perinatal mortality. A pathophysiological approach. *Lancet* 1980; **2**: 684-6.
10. Diehr P. Small area statistics: large statistical problems. *Am J Publ Health* 1984; **74**: 313-4.
11. Scott M. (Personal communication).
12. Knox EG, Armstrong EH, Lancashire R. The quality of notifications of congenital malformations. *J Epidemiol Community Med* 1984; **38**: 296-305.
13. West RR. High death rates or earlier deaths? *J R Coll Phys (Lond)* 1987; **21**: 73-6.
14. Friede A, Rhodes PH, Guyer B, Binkin NJ, Hannon MT, Hogue CJ. The postponement of neonatal deaths into the postneonatal period: evidence from Massachusetts. *Am J Epidemiol* 1987; **127**: 161-70.
15. Zdeb MS. Differences in trends in postneonatal mortality by birthweight in upstate New York. *Am J Publ Health* 1982; **72**: 734-6.
16. Carr-Hill RA, Hardman GF, Russell IT. Variations in avoidable mortality and variations in health care resources. *Lancet* 1987; **1**: 789-91.

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Motivation in deliberate self-harm

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SUMMARY

Fifty adult patients presenting with deliberate self-harm at the Royal Victoria Hospital were given a choice of nine reasons for their actions. Most chose more than one reason and all but two of the 24 who said that they wished to die chose at least one other motive. There were no trends with respect to sex, past history, or method of deliberate self-harm. These results illustrate the complexity of this condition and show the importance of investigating motives beyond simply the intent to die.

INTRODUCTION

Since Stengel [1] observed that attempted suicides could usefully be distinguished from completed suicides in terms of clinical and demographic characteristics, motivation in attempted suicide has been an area of study. The most studied motive is usually the intent to die, probably because this is of greatest importance in the clinical effort aimed at prevention of future suicide. There is a danger, however, that, if too much emphasis is placed on the intent to die, then other related motives could be under-assessed. In extreme cases the degree to which other motives are important might be simply judged to be inversely proportional to the degree of suicidal intent. This dichotomous thinking is influential in determining the attitudes of professionals to individuals who have attempted suicide. Studying attitudes towards self-poisoning in a general hospital, Ramon et al [2] noted that 'doctors were more accepting of the "wish to die" motive than of the others, and tended to see behaviour as either suicidal or manipulative, being relatively unsympathetic to the latter'. This theory can be harmful to patient management in that it leads to lack of enthusiasm, to a relationship of mistrust and hostility between doctor and patient and to an inability to identify motives other than suicidal intent which are important in management.

Kreitman [3] described five themes which may be seen in attempted suicide: a blind reaction to escape immediately from stress or to relieve tension, attention-seeking, seeking to induce anxiety or guilt in others, testing the benevolence of fate, and true, but frustrated, suicidal intent. He says 'It would be mistaken to think that most patients show only one of the list just enumerated. In most parasuicides the motives are multiple and may be mutually contradictory'. This statement reflects the conclusion of a body of research beginning as early as the 1950s and most recently carried out by a group of researchers in Oxford.[4]

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The purpose of this study is to examine the motives for attempted suicide of patients admitted to the Royal Victoria Hospital, Belfast. It is part of a larger study aimed at the analysis of attempted suicide and social factors.

PATIENTS AND METHODS

One of the authors (GL) was informed of all patients admitted to the Royal Victoria Hospital with a diagnosis of deliberate self-harm. They had presented at the accident and emergency department which has a policy to admit all such cases. The patients had to fulfil the criterion of non-fatal deliberate self-harm,[5] that is, 'a deliberate non-fatal act, whether drug overdosage or poisoning, done in the knowledge that it was potentially harmful and in the case of drug overdosage that the amount taken was excessive'. Individuals had to be at least 16 years old at the time of the act. They were excluded if they were unable to remember the circumstances or reasons behind the act (one demented patient), if they were unco-operative at the time of the study (one), or if they were in hospital at the time of the act (one).

During the period of the study, 65 patients were admitted to the Royal Victoria Hospital fulfilling the study criterion. The 15 who were not studied had either signed themselves out of the Hospital before they could be seen (five), did not otherwise stay long enough to be seen (seven) or had been transferred and could not be traced (three). They did not differ in sex ratio, mean age or method of deliberate self-harm from the study population. No other demographic information was available from this group.

Fifty patients were therefore interviewed by one of us (GL) and the following information was obtained:–

Demographic details.

Method of deliberate self-harm.

The Beck Suicidal Intent Scale.[6]

Patients were shown a series of cards, each of which stated a possible motive. They were asked to select which card, or cards, best described how they felt at the time of the act. The range of motives was:–

(a) To make people understand how desperate you were feeling.
(b) To get relief from a terrible state of mind.
(c) To escape for a while from an impossible situation.
(d) To seek help from someone.
(e) To find out whether or not someone really loved you.
(f) To make people sorry for the way they have treated you;
to frighten or get your own back on someone.
(g) To try to influence some particular person or get them to change their mind.
(h) To show how much you loved someone.
(i) To die.

This method of choice is closely based on earlier work by the Oxford group of researchers. Our results are compared with the findings of this group in earlier studies.

RESULTS

The population consisted of 24 men and 26 women. The mean age was 34·4 years old (SD 13·9). The mean age for men was 37·2 years (SD 16·2), and, for women, 31·8 years (SD 12·0). There was a past history of deliberate self-harm in 19 cases and a past history of treatment by a psychiatrist in 27 cases. Forty-three patients had taken an overdose, three had both taken an overdose and injured themselves by self-cutting, and one had carried out self-cutting alone. One had jumped from a bridge, one ingested bleach and one undertook self-poisoning by domestic gas. Twenty-four patients were seen within 24 hours, a further 15 within 48 hours and the rest after a longer period of time. One was seen after 15, another after 22 days.

Twenty patients were unemployed, five were on invalidity benefit and two were retired. The remainder were either housewives or in employment.

The number of cards chosen is given in Table I. The percentage frequency for each card selected is shown in Table II, together with details of percentage frequencies of other groups studied in the same, or a very similar way.[7, 8, 9, 10] Although the numbers are small, there was no significant correlation between the choice of cards and age, sex, past history of deliberate self-harm or method. The card 'To die' was associated with a significantly higher score on the Beck Suicidal Intent Scale (t test, $p < 0{\cdot}02$).

TABLE I

Number of cards chosen per patient

Cards chosen	*Patients*	*Patients who had already chosen the card 'To die'*
0	1	3
1	8	10
2	18	1
3	8	3
4	8	4
5	4	3
6	3	—

TABLE II

Frequency of alternative motives in this and other studies

Motive	*Number*	*This study* 50	*Bancroft et al (1976)* 128	*Bancroft et al (1979)* 41	*Hawton et al (1983)** 50	*James et al (1985)* 34
Make people understand	22	44%	**	20%	42%	35%
Relief from state of mind	25	50%	52%	44%	42%	70%
Escape from impossible situation	20	40%	42%	32%	42%	44%
Seek help	14	28%	33%	15%	18%	29%
Find out whether loved	10	20%	**	10%	24%	17%
Make people sorry or frighten them	8	16%	15%	10%	32%	12%
Influence, change someone's mind	7	14%	19%	7%	26%	21%
Show love	9	18%	20%	24%	26%	24%
Die	24	48%	44%	**	**	65%

*Study of adolescents. **This motive not presented in the study.

DISCUSSION

With regard to the study population, any conclusions from a hospital sample can be cautiously interpreted only insofar as they apply to the entire population of those who attempt suicide. This study is, however, reasonably representative within this community of the adults who contact hospitals. Almost all patients who attempt suicide and present at the Royal Victoria Hospital accident and emergency department are admitted.

Any study of motivation has to contend with the possibility that what might be declared as a motive is, in fact, simply a justification. In addition, some motives may be falsely declared and others denied — consciously or unconsciously. Furthermore, there is conflicting evidence as to whether motives chosen by patients correspond with assessment of motives produced by psychiatrists or by the patient's close relatives.[8, 9, 10] The uncertainty over validity can be an objection to any study of motivation. This particular method may be criticised because it presents a limited range of alternatives. On the other hand, it reduces observer error, results can be compared across various populations and it appears to be acceptable to patients. Regarding the demographic details of the population, the approximately equal sex ratio and the relatively high mean age are of some interest. It may be that this reflects a trend to a more equal sex ratio and higher mean age of attempted suicides. The Beck Suicidal Intent Scale score corresponds closely to that found in other hospital-based studies in the United Kingdom.

Two studies have been reported from Northern Ireland. Lukianowicz,[11] reporting on 100 consecutive female admissions with attempted suicide in 1962 and 1963 to Holywell Hospital, Antrim, found that in 75 cases there was evidence of gain, while of the other 25 'some subtle gain was probably achieved in these cases also'. Robertson, Savage and Herron [12] reported a number of features in cases of attempted suicide admitted to the Ulster Hospital, Dundonald. They presented their clients with a list of reasons and asked 'Of these reasons which are the strongest?' In reply, 13·2 per cent reported 'A desire to end it all', 22·8 per cent were indifferent, 19·1 per cent reported despair or hopelessness, 17·6 per cent a wish to escape from problems, 11·7 per cent anger or frustration and 12·5 per cent another motive. In their study the intent to die seemed to be incompatible with any other motive. This contradicts much of what has been found elsewhere and is not consistent with clinical experience. It also perpetuates the false dichotomy referred to earlier, that either one intends to die or one has another motive but not both.

The comparison shown in Table II between various studies carried out using very closely related methodology shows a marked consistency in both the percentage frequency of each choice of motive and the rank order of motives, which supports the reliability of this method.

This study supports the conclusion that intent to die is not the most common motive, and that it is the exclusive motive in only a minority of cases. More hostile or manipulative motives are those least often selected. It is possible that this might be because these motives would be judged less socially acceptable and therefore less likely to be chosen on reflection. Similarly, the frequency of the intent to die may be exaggerated. The validity of this method may be strengthened by the fact that there was a statistically significant association between the choice of intent to die and a high score on the Beck Suicidal Intent Scale. It is perhaps surprising that no trend with age was found. It might be expected from clinical experience and from the study reported on adolescents,[9] that more manipulative

motives may be found in a younger age group. This may be due to the exclusion of the under-16-year-old patients. The motive associated with the highest mean age (40·0 years) was 'To try to influence some particular person or get them to change their mind', which is one of the most manipulative choices presented.

Most importantly, this study illustrates the complexity of motivation in attempted suicide. It argues strongly against the analysis of motivation as being either intent to die or some other motive. Enquiry about motivation should not stop at the first answer, and, since the patient might stop anyway unless strongly prompted, there is a case for the presentation of a wide range of alternatives. This is economical of time and in almost every case elicits a response. At the very least, clinicians should be aware of the wide range of alternative motives in deliberate self-harm, which can have an important bearing on the quality of patient management.

Thanks are due to the medical staff of the Royal Victoria Hospital for permission to interview their patients, in particular to Miss Christine Dearden, FRCSEd, and Dr P Nelson. Thanks are also due to all the staff of the accident and emergency department of the Royal Victoria Hospital for their courtesy and helpfulness.

REFERENCES

1. Stengel E. Enquiries into attempted suicide. *Proc R Soc Med* 1951; **45**: 613-20.
2. Ramon S, Bancroft JHJ, Skrimshire AM. Attitides towards self-poisoning among physicians and nurses in a general hospital. *Br J Psychiatry* 1975; **127**: 257-64.
3. Kreitman N. Suicide and parasuicide. In: Kendell R, Zealley A, eds. Companion to psychiatric studies. Edinburgh: Churchill Livingstone, 1982: 396-411.
4. Hawton K, Catalan J. Attempted suicide. 2nd ed. Oxford: Oxford University Press, 1987.
5. Morgan HG, Burns-Cox C, Pocock H, Pottle S. Deliberate self-harm: clinical and socio-economic characteristics of 368 patients. *Br J Psychiatry* 1975; **127**: 564-74.
6. Beck AT, Schuyler D, Herman I. Development of suicidal intent scales. In: Beck AT, Resnick HLP, Lettieri DS, eds. The prediction of suicide. Baltimore: Charles Press, 1974; 45-56.
7. Bancroft JHJ, Skrimshire AM, Simkin S. The reasons people give for taking overdoses. *Br J Psychiatry* 1976; **128**: 538-48.
8. Bancroft JHJ, Hawton K, Simkin S, Kingston B, Cumming C, Whitewell D. The reasons people give for taking overdoses: a further enquiry. *Br J Med Psychol* 1979; **52**: 353-65.
9. Hawton K, Cole D, O'Grady J, Osborn M. Motivational aspects of deliberate self-poisoning in adolescents. *Br J Psychiatry* 1982; **141**: 286-91.
10. James D, Hawton K. Overdoses: explanations and attitudes in self-poisoners and significant others. *Br J Psychiatry* 1985; **146**: 481-5.
11. Lukianowicz N. Suicidal behaviour: an attempt to modify the environment. *Br J Psychiatry* 1972; **121**: 387-90.
12. Robertson K, Savage M, Herron S. Some characteristics of attempted suicide admitted to a district general hospital. Belfast: HMSO, 1985.

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Acute pancreatitis as a cause of sudden or unexpected death in Northern Ireland

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SUMMARY

Utilising incomplete data supplied by the Hospital Inpatient Analysis, the annual incidence of acute pancreatitis in Northern Ireland was estimated to be about 170 cases per million population. The annual mortality rate for the years 1974–1983, using figures obtained from the Registrar-General for Northern Ireland, was 12·3 cases per million. An increase in both incidence and mortality from acute pancreatitis was demonstrated during the study.

There were 191 deaths from pancreatitis during the study period and in 27 of these the diagnosis was made only at postmortem examination. Of the undiagnosed fatalities, 10 occurred in individuals with a history of alcohol abuse. Eight of the 27 undiagnosed cases had not sought medical attention, five had presented with a systemic complication of acute pancreatitis, and a further five had only minor gastrointestinal tract symptoms prior to death.

The diagnosis of acute pancreatitis requires a high index of suspicion and should be considered in acutely ill patients, particularly those with a history of alcohol abuse, who fail to respond to appropriate therapy.

INTRODUCTION

Acute pancreatitis is a condition peculiar to the pancreas in which there is destruction of the gland by enzymes escaping into its substance. It is characterised pathologically by haemorrhagic necrosis and inflammation of the pancreatic tissue and clinically by abdominal pain, vomiting and shock, which may cause death. The clinical diagnosis is usually based on the history, examination and investigations including serum and urinary amylase and ultrasonic and CT scans.

The reported incidence of acute pancreatitis depends on the diagnostic criteria used by the investigator. Graham found an incidence of 238 cases per million using a serum amylase concentration in excess of 2000 iu/l, but with a serum amylase of 1200 iu/l the incidence was 331 per million.[1] Even when similar diagnostic criteria are employed, the incidence of the condition appears to vary in different parts of the United Kingdom,[1, 2, 3, 4, 5, 6] and variations have even been described in different parts of the same city.[7]

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Several studies have described an increase in the annual incidence of acute pancreatitis,[3, 6, 8] although the number of deaths per year has remained static.[3, 6, 9] Part of the apparent increase in incidence may be due to improved diagnostic techniques and increased awareness of the condition amongst clinicians.[6, 9] On the other hand, comparison of years with high and low incidences show similar proportions of mild and severe disease,[2] and a real increase in the incidence of acute pancreatitis due to chronic alcoholism has been described.[6] In a number of cases, the diagnosis of acute pancreatitis is unsuspected during life and is only made at autopsy. In two series, such cases accounted for 35% and 41% of all fatal cases.[2, 3] Some of these may represent patients with mild pancreatic disease which is only fatal if it occurs in combination with other systemic disorders;[8] in others, the symptoms may be due to one of the complications of acute pancreatitis which mislead the clinician.

In this paper we describe a group of 27 individuals who died with acute pancreatitis in the years 1974–1983, but in whom the diagnosis was unsuspected during life and made only at postmortem. In addition we estimate the incidence and mortality rate of acute pancreatitis in Northern Ireland.

METHODS

The autopsy reports of fatal cases of acute pancreatitis in which a postmortem examination was carried out by staff of the Departments of Pathology and Forensic Medicine at the Queen's University of Belfast were scrutinised, and cases of acute pancreatitis in which the diagnosis was not made prior to death were identified. Twenty-seven such cases were found. The diagnosis of acute pancreatitis was confirmed by reviewing the available histological sections of pancreas. Clinical information was available in the clinical summary of the autopsy report, and in 10 cases the patients' hospital case notes were also reviewed.

The number of cases of acute pancreatitis for each year between 1974 and 1983 were calculated using the Hospital Inpatient Analysis (HIA), which records the final diagnosis of patients admitted to most hospitals in Northern Ireland. Unfortunately not all the hospitals provide the relevant data. For each year of the study period, the number of cases of acute pancreatitis treated in hospitals supplying data, and an estimate of the proportion of the population of Northern Ireland served by these hospitals were obtained. For example, in 1974 the Hospital Inpatient Analysis received notification of 84 cases of acute pancreatitis. In that year 40% of the population was served by hospitals providing the data. We thus estimated that a total of 210 cases had occurred amongst the total population of Northern Ireland.

The reported annual mortality from acute pancreatitis for the ten-year period was obtained from the Office of the Registrar-General for Northern Ireland. Both the Hospital Inpatient Analysis and the Registrar-General's Office provided figures based on the number of subjects indexed using Code 557–0 of the International Classification of Disease, which also codes for acute relapsing pancreatitis. Incidence figures therefore include these patients, some of whom will have been admitted on several occasions.

RESULTS

Making allowance for the inadequacies of the Hospital Activity Analysis (the data available represented only 40–65% of the population), the estimated annual

incidence of acute pancreatitis in Northern Ireland varied from 131 per million in 1975 to 246 per million in 1982 (mean annual incidence for 1974–83 was 169 per million).

During the study period there were 191 fatal cases of acute pancreatitis, giving a mean annual mortality rate of 12·3 per million of population (11·9 per million in the years 1974–78 and 12·7 in the years 1979–84) (Table).

TABLE

Fatal cases of acute pancreatitis occurring in Northern Ireland between 1974 and 1983

Year	*No. of deaths per year*	*Mortality per million population*	*No. diagnosed at autopsy per year*	*Percentage of fatal cases diagnosed at autopsy*
1974	17	11·0	—	—
1975	23	15·0	1	4·3%
1976	21	13·7	2	9·5%
1977	18	11·7	4	22·0%
1978	13	8·4	7	53·8%
1979	9	5·8	4	44·0%
1980	18	11·6	2	11·1%
1981	17	10·9	4	23·5%
1982	37	23·6	2	5·4%
1983	18	11·4	1	5·5%
Total	191		27	
Mean	19·1	12·3	2·7	14%

In 27 of these fatal cases (18 male, 9 female), the diagnosis of acute pancreatitis was unsuspected until postmortem examination. The underlying cause of the pancreatitis in these cases included alcohol abuse (10), biliary tract disease (2), trauma (4), hypothermia (2) and hyperparathyroidism (1). No definite cause was found in eight cases. Of these 27 cases, eight had not sought medical attention prior to death, four had consulted their family doctors but had not been admitted to hospital and 15 had been admitted to hospital.

Of the eight individuals who had not sought medical attention before death, two had no history of alcohol abuse and pancreatitis was unsuspected prior to death. These two subjects had complained of mild nausea and vomiting before death but this had not been sufficiently severe to necessitate their seeking medical attention. There was a history of chronic alcohol abuse in the remaining six. Two had complained of nausea and vomiting in the days before death; three were found dead, not having been seen for some days, and one collapsed suddenly and died.

Four patients had been examined by their general practitioner before death, although they had not been admitted to hospital. One had complained of heartburn for which he received an antacid, and a second appeared to have had a respiratory tract infection for which a broad spectrum antibiotic was prescribed. Two complained of acute dyspnoea and died from respiratory arrest.

Fifteen patients had been admitted to hospital prior to death. Three had presented with acute abdominal pain. In two of these a laparotomy revealed fat necrosis and pancreatitis apparently due to biliary tract disease; the third was thought to have intestinal obstruction but died before laparotomy could be carried out. In another two patients, the pancreatitis occurred as the result of hypothermia, and both died whilst being treated for this condition, without a diagnosis of acute pancreatitis being made. A further three patients presented with acute shortness of breath and were treated for congestive heart failure. Two of these had associated pain in the epigastrium and chest; all three died shortly after admission to hospital, and autopsy revealed acute pancreatitis but no cardiac pathology. In the remaining seven cases, conditions other than acute pancreatitis might have contributed to death. In three, the pancreatitis followed surgery, and death was related to the primary condition for which the operation was performed. One case occurred after a road traffic accident with severe head injury. Two chronic alcoholics sustained head injuries after having fallen whilst drunk; the pancreatitis in these two was more likely to be due to their alcohol ingestion than to abdominal trauma. One patient, an 85-year-old woman, had a long history of hypercalcaemia which was thought to be due to primary hyperparathyroidism. She presented with dehydration, chronic renal failure and chest signs. At autopsy, the parathyroids were not enlarged. The autopsy also showed acute pancreatitis and early bronchopneumonia but no cause for her hypercalcaemia was identified.

DISCUSSION

The average annual incidence of acute pancreatitis in Northern Ireland was estimated as being 169 cases per million. This estimate is greater than the incidence reported in other cities in the United Kingdom,[2, 3, 4, 5, 6] except in Glasgow where the incidence rate is estimated to be 238 cases per million.[1] During the study period, an apparent increase in the incidence of acute pancreatitis occurred. The mean annual incidence for the first five years of the study was 148 cases per million, and in the second five years 189 cases per million. This reflects the apparent increase reported in other studies.[3, 6, 8, 9]

The case records in each case were not scrutinised to identify patients suffering from acute relapsing pancreatitis. This condition is associated with recurrent episodes of abdominal pain necessitating hospital admission and has a low mortality rate. It is therefore likely that any such patients were admitted to hospital on more than one occasion. Additionally, the incidence depends on the diagnostic criteria used by the investigators. The figures in this paper were based on the frequency of the disease as diagnosed by clinicians and reported to the hospital index. The criteria used in diagnosing acute pancreatitis were reviewed in each case. Had strict diagnostic criteria been applied some cases might not have been included in this estimate of incidence. It is possible therefore that our figures are an over-estimate.

During the study period, no more than 65% of the population of Northern Ireland was covered by hospitals providing data to the HIA, and they tended to be those which served the larger towns and cities. It is recognised that the incidence of acute pancreatitis varies from area to area, even within a given population[7] and the figures which have been estimated only provide an approximation of the true incidence. The average mortality rate for the entire study period was 12·3 cases per million, which is similar to that seen in Great Britain.[3, 6] During the study period, there was a slight increase in the average annual mortality rate in Northern Ireland. In other studies the average annual mortality rate has stayed constant.[6, 9]

It has been recognised for many years that in 30 – 40 per cent of fatal cases the diagnosis is not made until autopsy.[2, 3, 10] Since a postmortem examination is not carried out on everyone who dies, it would be reasonable to assume that there are additional cases of acute pancreatitis where the condition is not suspected clinically, and where the diagnosis is never made. If these fatalities were included, the mortality rate expressed in terms of the population at risk would be even greater.

A number of patients with acute pancreatitis experience little or no pain.[3, 11] Nausea and vomiting may be the main complaint, and such symptoms, particularly in an alcoholic, may be attributed to a minor gastric condition.[11, 12] Three of the subjects in this study developed acute pancreatitis after an operation. In such cases the patient may not experience pain due to post-operative analgesia and there is a high mortality rate.[3, 5] Two cases presented with respiratory failure and in a further three a diagnosis of congestive heart failure was made. Respiratory failure, cardiac arrest and shock are amongst the most common complications in cases of acute pancreatitis which may be responsible for the patient's death. In these five cases the patients' symptoms misled the clinician, since they related to the systemic complications of acute pancreatitis rather than to the primary illness.

A review of the 27 fatal cases shows that potentially fatal pancreatitis can easily be missed due to mild symptoms and should be suspected, especially in chronic alcoholics who present with upper abdominal pain and vomiting. Further important diagnostic pitfalls include individuals presenting with complications of pancreatitis such as pulmonary oedema, and presentation in the post-operative state. Cases discovered at autopsy account for over one-third of fatal cases, and recognition of these before death, combined with appropriate therapy, might result in a further decrease in the mortality rate associated with this condition.[2, 3]

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REFERENCES

1. Graham DF. Incidence and mortality of acute pancreatitis. *Br Med J* 1977; **2**: 1603.
2. Bourke JB. Incidence and mortality of acute pancreatitis. *Br Med J* 1977; **2**: 1668-9.
3. Corfield AP, Cooper MJ, Williamson RCN. Acute pancreatitis: a lethal disease of increasing incidence. *Gut* 1985; **26**: 724-9.
4. McMahon MJ. Incidence and mortality rate of acute pancreatitis. *Br Med J* 1977; **2**: 1350.
5. Thomson HJ. Acute pancreatitis in North and North East Scotland. *J R Coll Surg Edin* 1985; **30**: 104-11.
6. Trapnell JE, Duncan EHL. Patterns of incidence in acute pancreatitis. *Br Med J* 1975; **2**: 179-83.
7. Bourke JB, Giggs JA, Ebdon DS. Variations in the incidence and the spatial distribution of patients with primary acute pancreatitis in Nottingham, 1969 – 76. *Gut* 1979; **20**: 366-71.
8. De Bolla AR, Obeid ML. Mortality in acute pancreatitis. *Ann R Coll Surg Engl* 1984; **66**: 184-6.
9. Trapnell JE. Incidence and mortality of acute pancreatitis. *Br Med J* 1977; **2**: 1603.
10. Williams G. Acute pancreatic necrosis as a cause of sudden death. *Br Med J* 1954; **1**: 1184-5.
11. Read G, Braganzan JM, Howat HT. Pancreatitis – a retrospective study. *Gut* 1976; **17**: 945-52.
12. The Copenhagen Pancreatitis Study Group. Copenhagen Pancreatitis Study – an interim report from a prospective epidemiological multicentre study. *Scand J Gastroenterol* 1981; **16**: 305-12.

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Treatment of pilonidal sinus by phenol injection

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SUMMARY

This report reviews the treatment of pilonidal sinus by phenol injection in 54 patients. Forty-four patients were treated initially by phenol injection and this was successful in 70%. The median number of injections per patient was one (range 1–5) with a median hospital stay per injection of two days (range 1–17 days). The median time to complete healing for patients treated by injection alone was two months (range 1–32 months). These results compare very favourably with more radical methods of treatment.

INTRODUCTION

The correct treatment of pilonidal sinus is still undecided. It is most often treated by radical methods which usually involve a lengthy hospital stay and considerable time off work. Many different techniques have been tried including excision,[1] excision with primary suture[2] and Z-plasty.[3] Goodall[4] and Verbeck[5] have reported a hospital stay of 18 days and 15 days respectively for excision with primary closure while Notaras[6] and Goodall[4] have reported a mean hospital stay of 17 days and 18 days respectively for excision without closure. Although these methods can be very effective, they are more painful and require repeated dressings. Lord and Millar[7] described a simple deroofing treatment for pilonidal sinus performed under local anaesthesia. However, this technique is not ideal since 40% of the patients failed to co-operate with the dressing régime. The aim of this study was to review the results of treatment of pilonidal sinus by phenol injection in terms of cure rate, time for complete healing, number of injections per patient and hospital stay.

PATIENTS AND METHODS

Between 1964 and 1986, 54 patients with pilonidal sinus were treated by phenol injection as either all or part of their treatment. Most of these cases were under the care of one of the authors (WJHG); some, however, were patients of other surgeons. Details of the patients were obtained from a retrospective study of case notes. Forty-four were male, and 10 were female, with a mean age of 25 years (range 16–53 years). The median length of symptoms was four months (range 3 days–13 years). The patients were followed up until their sinuses had completely healed.

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The method of phenol injection was similar to that first described by Maurice and Greenwood.[8] Eighty per cent phenol was injected without pressure into the main sinus tract in order to sterilise infected contents and remove embedded hair and débris. Following induction of general anaesthesia with endotracheal intubation, the patient was placed prone on the operating table. The table was then split in the middle to obtain the 'jack-knife' position. After previous shaving of the sacral area, the buttocks were held apart with 7·5 cm strapping to expose the sacral area and anal verge. The skin of the area was cleansed with cetrimide and chlorhexidine solutions, and then dried and towelled up in the usual manner. The skin around the sinus was protected by smearing liberally with vaseline and the anus was protected by covering it with vaseline gauze. After careful probing, any loose hairs were removed with forceps from the sinus and from any of its side tracts. The main sinus tract was injected with a solution of 80% phenol using a blunt-nosed needle which fitted snugly into the sinus opening. The injection was performed slowly using the minimum of pressure to avoid phenol being forced into the tissue surrounding the sinus and causing a local inflammatory reaction. The injection was stopped when phenol was seen coming from any of the side openings and any excess was quickly wiped away. After one minute, pressure applied around the sinus tract expressed the phenol and brought loose hairs to the surface, which were then picked out. The whole procedure was repeated twice, each time leaving the phenol *in situ* for one minute, thereby giving a total exposure time of three minutes. The sinus was then washed out with saline and curetted. Vaseline gauze and a light dressing were then applied to the injected area. The patients were allowed home the following day and were instructed to have frequent baths. Strict hygiene of the area was emphasised during the healing period. After the sinus has healed, it is advisable to wash the natal cleft after defaecation rather than using toilet paper. Special care must be taken with loose hairs, particularly after a visit to the barber.

RESULTS

Of the 54 patients, 31 (57%) were treated by phenol injection alone and 23 (43%) required an additional procedure. In all, 97 injections and 35 additional procedures were performed (Table). The majority of patients required only one injection procedure (range 1–5). The median hospital stay per injection was two

TABLE

Procedures performed in 54 patients with pilonidal sinus

	Total procedures	*Initial procedures*
Phenol injection	97	44 (81%)
Excision without closure	9	—
Excision with primary closure	5	1 (2%)
Excision with partial closure	1	—
Incision	4	3 (6%)
Curettage	6	—
Z-plasty	1	—
Drainage of abscess	9	6 (11%)
Total	132	54 (100%)

days (range 1–17 days) with more than 85% of patients staying for two days or less. Of those who stayed for longer than two days, most only stayed for 3–5 days. One patient stayed nine and another 17 days, both having had an abscess lanced shortly before injection. The patients treated by injection alone had a median time for complete healing of two months (range 1–32 months). Only four patients took over one year for complete healing to take place, having healed initially but requiring a further injection as the sinus had recurred. Therefore, of the 44 patients who were treated initially by phenol injection, 31 (70%) were successfully cured by injection alone, requiring from 1–4 injections per patient. Thirteen (30%) required an additional method of treatment, such as excision with or without primary closure.

DISCUSSION

Pilonidal sinus is a relatively minor condition, yet the customary treatment is fairly radical. Phenol injection is a simple procedure requiring only a short hospital stay. Ideally, the injection should be done at a quiescent phase and a pre-injection course of an appropriate antibiotic may be useful in some cases. Postoperative discomfort is minimal and patients can return to work almost immediately. Our cure rate of 70% was slightly lower than the 73–91% reported elsewhere.[9, 10] However, our hospital stay of two days compares favourably with the 2·9 days mentioned in an earlier report of this procedure in Northern Ireland,[9] and was much shorter than the 15–18 days reported for more radical forms of treatment.[2, 3] The time required for complete healing in our series (two months) was slightly longer than the 3–4 weeks reported by Stewart and Bell[9] but compares favourably with the 11 days to 6 months reported for more radical surgical methods.[11, 12, 13] Seventeen patients (39%) required repeat injections, but we believe that this may still be preferable to more radical surgery with the risk of delayed healing and subsequent wound breakdown. In summary, the very short hospital stay and early return to work together with a cure rate of 70% combine to make this method of treatment attractive for pilonidal sinus.

We would like to thank those consultant surgeons who gave us permission to include cases in this report.

REFERENCES

1. Marks J, Harding KG, Hughes LE, Ribeiro CD. Pilonidal sinus excision — healing by open granulation. *Br J Surg* 1985; **72**: 637-40.
2. Rains AJH. Treatment of pilonidal sinus by excision and primary closure. *Br Med J* 1959; **1**: 171-3.
3. Casten DF, Tan BY, Ayuyao A. A technique of radical excision of pilonidal disease with primary closure. *Surgery* 1973; **73**: 109.
4. Goodall P. The aetiology and treatment of pilonidal sinus: a review of 163 patients. *Br J Surg* 1961; **49**: 212-8.
5. Verbeck HO, Bender J. Results of treatment of pilonidal sinus by block excision and primary closure. *Arch Chir Neerl* 1974; **26**: 311-8.
6. Notaras MJ. A review of three popular methods of treatment of postanal (pilonidal) sinus disease. *Br J Surg* 1970; **57**: 886-90.
7. Lord PH, Millar DM. Pilonidal sinus: a simple treatment. *Br J Surg* 1965; **52**: 298-300.

8. Maurice BA, Greenwood RK. A conservative treatment of pilonidal sinus. *Br J Surg* 1964; **51**: 510-2.
9. Stewart TJ, Bell M. The treatment of pilonidal sinus by phenol injection. *Ulster Med J* 1969; **38**: 167-71.
10. Shorley BA. Pilonidal sinus treated by phenol injection. *Br J Surg* 1975; **62**: 407-8.
11. Kronborg O, Christensen K, Zimmermann-Nielsen C. Chronic pilonidal disease: a randomized trial with a complete 3-year follow-up. *Br J Surg* 1985; **72**: 303-4.
12. Rainsbury RM, Southam JA. Radical surgery for pilonidal sinus. *Ann R Coll Surg* 1982; **64**: 339-41.
13. McLaren CA. Partial closure and other techniques in pilonidal surgery: an assessment of 157 cases. *Br J Surg* 1984; **71**: 561-2.

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Coronary risk factor prevalence in a high incidence area: results from the Belfast MONICA Project

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SUMMARY

Northern Ireland remains at the top of the world mortality league for ischaemic heart disease. The Province is providing a centre for the World Health Organisation's MONICA Project. Registration of coronary heart disease events began in 1983 and the first of three population surveys took place in 1983–4. A total of 2,361 men and women aged 25–64 years was screened. Subjects were shorter and heavier than their fellow citizens in Great Britain. The estimated mean cholesterol levels in the 25–64-year-old population (5·80 mmol/l in males and 5·85 mmol/l in females) were similar to those reported from Great Britain. Although mean systolic blood pressures were lower, mild diastolic hypertension was considerably more common; cigarette smoking levels were similar. The results were consistent with those expected for an area with a high coronary heart disease mortality, with more than 80% of subjects being at increased risk in terms of the three major factors (cigarette smoking, hypertension and raised cholesterol).

Public concern about coronary heart disease has grown and recently the Department of Health and Social Services (NI) has launched a 10-year prevention programme which will primarily employ a population approach.

INTRODUCTION

Northern Ireland remains at the top of the world mortality league for ischaemic heart disease (ICD No 410–414). The age-standardised mortality rate per 100,000 (40–69 years) for 1986 was 557 in males and 169 in females.[1] The respective figures in Scotland were 542 and 180.[2] These countries now head the world mortality league as a continued decline has been reported

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from Finland.[3] Belfast and its environs form a centre for the World Health Organisation-co-ordinated MONICA (MONItoring of trends and determinants in CArdiovascular disease) Project.[4] The MONICA Project is being carried out in more than 30 centres throughout the world and will last for a decade. It is in two parts: registration of coronary heart disease events (fatal and non-fatal) will establish trends in incidence, and screening of independent samples of the population on three occasions will monitor risk-factor levels. In this way, possible changes in incidence and case fatality may be analysed in relation to risk-factor changes within communities, and across the many centres. The study area comprises the Belfast, Castlereagh, North Down and Ards Health Districts of the Eastern Health and Social Services Board area with a total population of 499,111 and a target population, aged 25 – 64 years, of 223,575.[5] Registration began on 1 January 1983 and the first survey took place from October 1983 to September 1984.

METHODS

The MONICA Project requires that at least 200 persons of each sex should be screened for risk-factor levels in each 10-year age group between 25 and 64 years. Specific assumptions are made for each of the major risk-factors concerning the magnitude of the true change which should be detectable, and the sample size was estimated to give a high power to detect these changes as statistically significant. Screening is to be carried out on three well separated occasions during the study period. Various core (essential) items are: smoking (self-reported, validated by serum thiocyanate estimation), blood pressure, serum cholesterol, height and weight. Other factors strongly recommended for inclusion are high density lipoprotein cholesterol (HDL-cholesterol) and gamma glutamyl transferase. Each centre is free to study other factors and there are several MONICA optional studies, eg nutrition, exercise, medical care, drugs and psychosocial factors.

The sampling frame selected for the survey was the Northern Ireland Central Services Agency's general practitioner lists. A stratified random sample of 5,000 subjects was chosen with eight strata defined by age group and sex. This sample size was sufficient to give almost 400 subjects per stratum assuming a response rate of 75% and an ineligibility rate of 15%. Subjects were initially contacted by letter, with an introductory questionnaire seeking preferences for times and clinics. In the event of envelopes being returned undelivered or a non-response, general practitioners' receptionists were approached and asked to check addresses and, when necessary, subjects were visited at home. Subjects were seen in community health suites of health centres throughout the area. Arranging clinics in Belfast and certain other areas was a problem as subjects were sometimes reluctant to travel out of their localities. A strict protocol was adhered to for all screening procedures. Height and weight were measured in a standardised way, and blood pressure (diastolic phase V) was taken before venipuncture using the Hawksley random zero sphygmomanometer. Two readings were obtained when the subject was sitting and relaxed for five minutes. Where appropriate, an over-sized cuff was used. Periodic checks for digit preference on the uncorrected readings were made and recorders were retrained and recertified as necessary. As well as employing internal quality control procedures, total cholesterol, HDL-cholesterol and thiocyanate estimation were subject to external quality control through the World Health Organisation reference laboratory in Prague. Centrifuging and separation took place within three hours of venipuncture.

Cholesterol was estimated using the enzymatic CHOD-PAP method[6] and precipitation for HDL-cholesterol estimation employed phosphotungstic Mg^{++} reagents.[7] Thiocyanate was measured using Butts' method. Subjects were requested not to eat a heavy meal during the few hours before the examination (semi-fasting).

Estimates of means and proportions for the 25–64-year-old population were obtained by appropriate weighting of the estimates from the separate age strata.

RESULTS

Considerable difficulty was encountered in tracing subjects because of the large shifts of population which have occurred over the years of civil disturbance. Subjects were only classified as ineligible if at least two visits to the last known address and reference to other sources failed to establish that the person was alive and living in the study area: 1,620 (32%) were so classified.

A total of 2,361 subjects were screened giving an overall response rate of 70%; non-response records were completed for 82% of the 1,019 non-responders. Response rates were higher in the older age groups. The unemployed were under-represented in the sample whereas skilled workers were over-represented.[5]

The mean height of men in the population was estimated as 172·9 (SE 0·2) cm and of women, 159·3 (SE 0·2) cm. Younger subjects were taller than older subjects and this was more marked in males. The estimated mean weight in the male population was 76·6 (SE 0·4) kg and in the female population 63·7 (SE 0·3) kg; the weights were greatest in the 45–54 year age group in both sexes. The distribution of body mass index by age and sex is shown in Table I. In both sexes, the percentage carrying excess weight (body mass index [BMI] >25 kg/m^2) increased with age. In the three younger age groups a greater proportion of males than of females bore excess weight. These differences disappeared in the older age group. Over half of the men in the sample, 53·6%, were classified as carrying excess weight compared with 44·4% of females. A large proportion of men and women in the sample claimed to take no leisure time exercise: 43·5% of the males and 47·3% of the females.

TABLE I

Distribution of body mass index (kg/m^2)

	Male				*Female*			
Age (years)	*n*	*Over-weight* %	*Obese* %	*Excess weight* %	*n*	*Over-weight* %	*Obese* %	*Excess weight* %
25–34	241	34·0	6·2	40·2	267	17·2	9·4	26·6
35–44	276	40·9	12·0	52·9	294	30·3	10·9	41·2
45–54	334	48·8	11·7	60·5	322	35·1	15·8	50·9
55–64	314	48·1	8·9	57·0	308	38·6	17·5	56·1

Overweight:- BMI $>25 <=30$ kg/m^2
Obese:- BMI >30 kg/m^2
Excess weight:- BMI >25 kg/m^2
Missing values:- 3 male, 2 female

The distribution of total serum cholesterol is shown in Table II. Although females had a lower cholesterol than males in the two younger age groups, the converse was true in the older groups. The biggest increase between age groups was seen in males from the 25 – 34 to the 35 – 44 age group and in females from the 35 – 44 to the 45 – 54 year age group. In the 55 – 64 year age group, cholesterol levels were markedly higher in females than males. Across all age groups, however, the estimated mean levels for the population were similar in the sexes: 5·80 (SE 0·03) mmol/l in males and 5·85 (SE 0·03) mmol/l in females. Taking an upper limit of 6·70 mmol/l, 21 % of males and 24 % of females in the sample were identified as having abnormal levels. Applying an upper limit of 5·20 mmol/l, as recommended by one authority,[8] identified 71 % of males and 69 % of females to be at increased risk and in the 55 – 64 year age group, 80 % of males and 91 % of females were so identified.

TABLE II

Serum total cholesterol levels (mmol / l) *

	Male					*Female*				
Age (years)	*n*	*Mean*	*SD*	*Percentile* 5th	95th	*n*	*Mean*	*SD*	*Percentile* 5th	95th
25 – 34	233	5·22	0·97	3·79	6·72	264	4·99	0·91	3·59	6·48
35 – 44	276	5·95	1·10	4·32	7·58	291	5·44	0·93	4·04	7·05
45 – 54	328	6·02	1·08	4·34	7·89	320	6·26	1·18	4·49	8·05
55 – 64	312	6·12	1·05	4·63	7·83	303	6·74	1·14	4·84	8·71

*Conversion: SI to traditional units — 1 mmol/l = 38·6 mg/100 ml (approximately)
Missing values:- 19 male, 15 female

The distribution of HDL-cholesterol in men and women is shown in Table III. The mean levels remained fairly constant across the age groups, but were considerably lower in males than in females. The estimated population means were 1·14 (SE 0·01) mmol/l and 1·40 (SE 0·01) mmol/l, respectively.

TABLE III

HDL cholesterol levels (mmol / l) *

	Male					*Female*				
Age (years)	*n*	*Mean*	*SD*	*Percentile* 5th	95th	*n*	*Mean*	*SD*	*Percentile* 5th	95th
25 – 34	231	1·16	0·26	0·79	1·67	257	1·39	0·31	0·88	1·91
35 – 44	270	1·14	0·28	0·75	1·63	285	1·38	0·31	0·87	1·94
45 – 54	321	1·12	0·28	0·75	1·60	313	1·44	0·39	0·90	2·20
55 – 64	304	1·13	0·29	0·73	1·69	295	1·40	0·34	0·87	2·02

*Conversion: SI to traditional units — 1 mmol/l = 38·6 mg/100 ml (approximately)
Missing values:- 42 male, 43 female

The distribution of current cigarette smoking by age and sex is given in Table IV. Just over a third of men and women in the sample were cigarette smokers, even if only occasional. The estimated proportions of cigarette smokers in the 25–64-year-old male and female population were 35·8% (SE 1·4) and 36·3% (SE 1·4), respectively. A further 13·2% of males in the sample were cigar smokers and of these 70% had been cigarette smokers in the past. A higher proportion of females in the sample had never smoked cigarettes: 45·3% as opposed to 29·2% of males; and a higher proportion of males were ex-smokers: 35·4% as opposed to 18·6% of females. Male smokers smoked on average 18·8 cigarettes per day and female smokers 15·8. Only 16·0% of male smokers in the sample used low-tar-content cigarettes as opposed to 32·5% of female smokers. There were a further 131 individuals who denied smoking (5·5% of all subjects) but had a serum thiocyanate level > 50 mmol/l, of whom 37 (1·6% of all subjects) had a level in excess of 70 mmol/l.

TABLE IV

Cigarette smoking

	Male				*Female*			
Age (years)	*n*	*Current %*	*Ex- %*	*Never %*	*n*	*Current %*	*Ex- %*	*Never %*
25–34	241	36·9	23·7	39·4	268	39·9	20·6	39·6
35–44	277	39·0	27·8	33·2	295	35·9	17·0	47·1
45–54	336	33·0	39·3	27·7	322	33·2	18·6	48·1
55–64	314	33·8	46·8	19·4	308	36·0	18·5	45·5

Tables V and VI show the distribution of systolic and diastolic blood pressures. Although females had lower systolic blood pressure than males in the younger age groups, by middle age this difference had almost disappeared: this held to a lesser extent for diastolic blood pressure. The estimated mean systolic and diastolic blood pressures in the male population were 133·1 (SE 0·5) mmHg and 82·0 (SE 0·3) mmHg, respectively. For the female population, the corresponding figures were 128·6 (SE 0·5) mmHg and 77·5 (SE 0·3) mmHg. Applying cut-off

TABLE V

Systolic blood pressure (mmHg)

	Male					*Female*				
Age (years)	*n*	*Mean*	*SD*	*Percentile 5th*	*95th*	*n*	*Mean*	*SD*	*Percentile 5th*	*95th*
25–34	240	126·6	12·8	106·5	147·0	268	114·5	12·7	94·0	134·7
35–44	277	127·2	15·5	104·0	153·0	294	120·9	14·9	100·0	145·0
45–54	336	135·0	21·0	106·5	171·5	322	134·4	21·7	104·0	173·0
55–64	314	145·5	22·6	111·5	187·0	307	144·9	23·7	108·5	184·5

Missing values:- 1 male, 2 female

TABLE VI

Diastolic blood pressure (mmHg)

	Male					*Female*				
Age (years)	*n*	*Mean*	*SD*	*Percentile* 5th	95th	*n*	*Mean*	*SD*	*Percentile* 5th	95th
25–34	240	77·4	10·6	59·5	94·0	268	70·9	9·5	54·7	87·5
35–44	277	81·8	11·7	63·5	102·0	294	75·1	10·3	61·0	94·0
45–54	336	85·0	12·3	65·7	106·0	322	81·8	11·7	64·5	104·0
55–64	314	84·7	12·3	67·0	105·0	307	82·6	12·0	65·5	102·0

Missing values:- 1 male, 2 female

points of systolic $\geq$ 140 mmHg and diastolic $\geq$ 90 mmHg identified 213 (18·2%) males and 141 (11·8%) females in the sample as having abnormal blood pressure. Of these, 33 males (15·5%) and 37 females (26·2%) were on antihypertensive drugs. A further 33 males and 56 females were on antihypertensive drugs and were normotensive.

Taking risk factors in combination (cholesterol $\geq$ 5·2 mmol/l, cigarette smoking, systolic blood pressure $\geq$ 140 mmHg and diastolic $\geq$ 90 mmHg, or controlled on antihypertensives), 82·5% of men and 80·6% of women in the sample had at least one factor present; 39·4% of men and 37·0% of women had two or more factors.

DISCUSSION

The response rate achieved in the present study was 70% compared with 74% in the Medical Research Council trial of treatment of mild hypertension[9] and 78% in the British Regional Heart Study.[10] The latter studies, however, were based on selected general practices.

Both male and female subjects were shorter than their counterparts in Great Britain:[11] males on average were 0·5cm and females 1·3cm shorter. The difference was most marked in the younger age groups, possibly suggesting a difference in the rate of nutritional change between the two populations. Our subjects were also heavier: males on average by 1·6kg and females by 0·7kg. The differences were greatest in the middle (35–54) age groups. The body mass indices, which are largely uncorrelated with height,[12] revealed that 53·6% of males carried excess weight as opposed to 44·9% in Great Britain. In females the respective figures were 44·4% and 35·5%. In comparison with Great Britain, the higher proportion of subjects with excess weight was most striking in the 35–54 year olds. Increased weight contributes to coronary heart disease and this may affect serum cholesterol levels and blood pressure. Obesity has been reported to be associated with an increased production of low density lipoproteins.[13]

The mean total cholesterol level of 6·10 mmol/l for males aged 40–59 in our study was similar to that in the British Regional Heart Study (6·30 mmol/l).[14] There were differences in the biochemical methodology, and the Regional Heart Study samples were taken non-fasting, as opposed to semi-fasting in our study. Moreover, the British Regional Heart Study included northern regions in which the incidence of coronary heart disease is similar to our own. In the 40–59 year

age group in the British Risk Factor Prevalence Study, a mean of 6·0 mmol/l was found.[15] The latter study, using cut-off points of >6·5, >5·7 and >5·2 mmol/l, identified 23%, 45% and 63%, respectively, of the sample aged 25–59 years as exceeding these limits. The respective proportions in the present study were 24%, 51% and 67%. It is likely that the estimates made by Lewis and his colleagues[15] for achieving a satisfactory fall in cholesterol levels are relevant in the context of our population. The mean serum cholesterol levels reported from North Karelia in 1982 in the age group 30–59 were 6·3 mmol/l for men and 6·2 mmol/l for women.[16] In the same age group in our sample, mean values of 5·92 and 5·87 mmol/l were found for men and women, respectively.

Although the role of HDL-cholesterol as a major independent risk factor has been disputed,[17] many studies testify to its importance as a predictor of coronary heart disease.[18] The mean level in 40–59-year-old males in the present study was 1·12 mmol/l, similar to that found in the British Regional Heart Study (1·15 mmol/l).[14] The methodology in that study, however, has been criticised.[18] Furthermore, the delays in preparation of samples incurred in that study by sending to a central laboratory may have affected the precipitability. In subjects aged over 50 years in the present study, the levels were similar to those found in Framingham.[19]

The proportion of males who smoked was comparable with that reported in other local surveys, but in females it was higher.[20] The proportion of females in Northern Ireland aged 16 years and over smoking cigarettes in 1984 was estimated as only 29%. It is possible that female smoking is more an urban phenomenon in Northern Ireland. However, there was evidence from the thiocyanate estimations that smoking was under-reported. The prevalence of cigarette smoking was similar to Great Britain (1982: males 38%, females 33%).[21] In North Karelia in 1982, 38% of males aged 25–59 years were cigarette smokers[16] as compared with 36% in the present study.

The mean systolic blood pressures in 40–59-year-old males were markedly lower than in the British Regional Heart Study:[10] 134·6 versus 145·3 mmHg — the difference was greatest in younger males. Conversely, the mean diastolic blood pressure was higher in our study: 84·0 versus 82·2 mmHg. The Medical Research Council's trial of treatment of mild hypertension[9] identified 9% of 35–64-year-old men and women as having diastolic pressures of 90–109 mmHg (with a systolic pressure of below 200 mmHg). In our sample 20% of those aged 35–64 years fulfilled these criteria: 25% of men and 15% of women. Diastolic blood pressure has been shown to be a better predictor than systolic, at least in younger Norwegian men.[22] The relationship between coronary heart disease risk and diastolic blood pressure has lately been reaffirmed.[23] The 'cross-over' phenomenon usually observed in cross-sectional studies, that above 50 years there are more hypertensive females than males, was not seen in this study although a trend in that direction was observed. Johannson et al have suggested that this phenomenon is an artefact caused by selective mortality in hypertensive males.[24]

Concern about Northern Ireland's position in terms of coronary heart disease has stimulated action. In 1984 the Northern Ireland Coronary Prevention Group published its strategy for the primary prevention of coronary heart disease.[25] The recent launch of the 'Change of Heart' Programme[26] by the Department of Health and Social Services is appropriate as more than 80% of the population have been shown to have at least one risk factor present.

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REFERENCES

1. Registrar-General for Northern Ireland. [Unpublished data].
2. Registrar-General for Scotland. Report, 1986. Edinburgh: HMSO, 1987.
3. Tuomilehto J, Geboers J, Salonen JT, Nissinen A, Kuulasmaa K, Puska P. Decline in cardiovascular mortality in North Karelia and other parts of Finland. *Br Med J 1986;* **293**: 1068-71.
4. World Health Organisation. Proposal for the multinational MONItoring of trends and determinants in CArdiovascular disease and protocol (MONICA Project). Geneva: WHO, 1983.
5. Northern Ireland 1981 Census (1985 revision) — Source: Registrar-General, Northern Ireland, and Policy Planning and Research Unit, Department of Finance and Personnel, Northern Ireland. [Unpublished data].
6. Boehringer (CF) & Söhne, GmbH, Mannheim. Test combination cat. no. 704121.
7. Lopes-Virella MF, Stone P, Ellis S. Cholesterol determination in high-density lipoproteins separated by three different methods. *Clin Chem* 1977; **23**: 882-4.
8. European Atherosclerosis Society. Strategies for the prevention of coronary heart disease: a policy statement of the European Atherosclerosis Society. *Eur Heart J* 1987; **8**: 77-88.
9. Medical Research Council Working Party. MRC trial of treatment of mild hypertension: principal results. *Br Med J* 1985; **291**: 97-104.
10. Shaper AG, Pocock SJ, Walker M, Cohen NM, Wale CJ, Thomson G. British Regional Heart Study: cardiovascular risk factors in middle-aged men in 24 towns. *Br Med J* 1981; **283**: 179-86.
11. Knight I. Heights and weights of adults in Great Britain. London: HMSO, 1984. (Issued by the Office of Population Censuses & Surveys, Social Survey Division).
12. Goldbourt U, Medalie JH. Weight-height indices. *Br J Prev Soc Med* 1974; **28**: 110-26.
13. Kesaniemi YA, Grundy SM. Increased low density lipoprotein production associated with obesity. *Arteriosclerosis* 1983; **3**: 170-7.
14. Thelle DS, Shaper AG, Whitehead TP, Bullock DG, Ashby D, Patel I. Blood lipids in middle-aged British men. *Br Heart J* 1983; **49**: 205-13.
15. Lewis B, Mann JI, Mancini M. Reducing the risks of coronary heart disease in individuals and in the population. *Lancet* 1986; **1**: 956-9.
16. Puska P, Nissinen A, Salonen JT, et al. The community-based strategy to prevent coronary heart disease: conclusions from the 10 years of the North Karelia Project. *Ann Rev Publ Health* 1985; **6**: 147-93.
17. Pocock SJ, Shaper AG, Phillips AN, Walker M, Whitehead TP. High density lipoprotein in cholesterol is not a major risk factor for ischaemic heart disease in British men. *Br Med J* 1986; **292**: 515-9.
18. Miller NE. High density lipoprotein cholesterol is not a major risk factor for ischaemic heart disease in British men (letter). *Br Med J* 1986; **292**: 1012-3.
19. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. *Am J Med* 1977; **62**: 707-14.
20. Policy, Planning and Research Unit (Northern Ireland). Annual abstract of statistics no 4, 1985. Belfast: HMSO, 1986.
21. Central Statistical Office. Social trends. London: HMSO. 1984.

22. Tverdal A. Systolic and diastolic blood pressures as predictors of coronary heart disease in middle-aged Norwegian men. *Br Med J* 1987; **294**: 671-3.

23. Martin MJ, Hulley SB, Browner WS, Kuller LL, Wentworth D. Serum cholesterol, blood pressure and mortality: implications from a cohort of 361,662 men. *Lancet* 1986; **2**: 933-6.

24. Johannson S, Vedin A, Wilhelmsson C. Myocardial infarction in women. *Epidemiol Rev* 1983; **5**: 67-95.

25. Boyle DMc, Scott ME, Scally GJ, et al. A strategy for a community based programme for the primary prevention of coronary heart disease in Northern Ireland. Belfast: Northern Ireland Chest, Heart and Stroke Association, 1984.

26. Strategy for prevention of coronary heart disease in Northern Ireland (Northern Ireland's Change of Heart Programe). Belfast: Department of Health and Social Services (NI), 1986.

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Ice rink injuries: a new epidemic in Northern Ireland

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INTRODUCTION

A new Olympic-size ice rink (60 × 30m) opened in Dundonald in 1986 less than a mile from the Ulster Hospital. At about the same time a smaller 38 × 22m 'family fun rink' opened in Bangor within eight miles of the Hospital. These are the first ice rinks in Northern Ireland for nearly 20 years since the closure of the rink in the King's Hall, Balmoral, Belfast, and the majority of those using the facilities were novices. A large number of patients were referred to the Accident and Emergency Department of this hospital with injuries sustained while ice skating, and an initial analysis of the injuries received in those attending in the first six months was made by Freeland.[1] Some of these patients were referred to the Orthopaedic and Fracture Clinics at the Ulster Hospital for further treatment. This paper presents the types of injury referred to these two specialist clinics in the first six months of the opening of the rinks.

RESULTS

During the assessment period, 636 patients attended the Accident and Emergency Department following injury at the ice rink, and 78 (12%) were referred to the Orthopaedic and Fracture Clinics, or admitted to the Fracture Wards. There were 48 females and 30 males, aged 8–68 years. The most frequently injured age group was that of 16–25 years in both sexes. The injuries treated consisted of 70 fractures, two dislocations, five effusions into joints, and two soft tissue injuries. Two patients had double injuries. There were four injuries to bones in the hand. Wrist injuries were the most common with 37 (fracture of the distal radius in 27 and the scaphoid in 4). There were four Colles' fractures (fracture of the distal radius less than 2·5 cm. from the wrist often with fracture of the ulnar styloid with impaction, backward and radial angulation of the radius), six fractures at the elbow and one at the shoulder. In the leg there were 12 ankle fractures, three fractures tibia and fibulae, and three fractured patellae. The dislocations involved a shoulder and patella.

The fractures were all closed injuries, 19 involving the lower limb and 51 the upper limb. Fifteen required reduction, two by open methods, and five of these patients required hospital admission. The dislocations were reduced in outpatients under anaesthetic. The distribution of injuries by their anatomical sites, male compared with female, is shown in the Figure. Upper limb compared with lower limb injury ratios are similar; the anatomical site of the injuries varies in the lower limbs with a greater proportion of knee injuries in females and greater ankle and tibia and fibulae injuries in the males. This may reflect the fact that the

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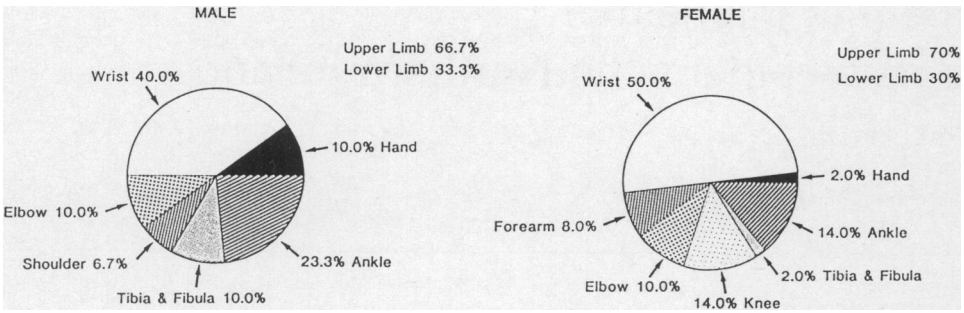

Figure. Anatomical distribution of injuries comparing male with female.

injuries in males tended to be high speed injuries. The number of injuries referred to the clinics was initially high, especially in the first four weeks. A second peak was noted after the Christmas holiday period, but the overall trend was downward as those skating gained more experience. There were over 355,000 attendances at the ice rinks during the assessment period, an average of 14,200 per week. The 636 injuries seen at the Accident and Emergency Department represent one injury per 550 ice rink attendances, or 0·2%. Of these, 78 were referred to the Orthopaedic and Fracture Clinics at the Ulster Hospital.

DISCUSSION

Ice skating has become very popular in Belfast since the opening of the new rinks, and skating appeals to a wide age range. The initial high number of injuries correlates well with the learning phase that must take place in any sport taken up for the first time by non-experienced participants. Most skaters are female, and more females are injured.[2, 3, 4]

The more severe injuries were seen in the males, tending to involve the lower limb and due to relatively high speed accidents, usually while playing ice hockey.[5, 6] The majority of injuries in both males and females involved the upper limb, falling on the outstretched hand. The nature of the sport requires considerable balance control, and inevitably leads to a large number of falls on to a very hard surface. Another major hazard is collision with another skater who is often out of control, leading to injury to one or both. Lower limb injuries requiring orthopaedic treatment were either high speed incidents, in the case of most of the fractured tibiae, or due to severe rotational forces at the ankle joint.

The severity of the injuries in those patients attending the Accident and Emergency Department were variable: only 12% received specialised orthopaedic management at this hospital. This is less than in other reports,[3, 4] since a number of patients with fractures were being referred to a hospital closer to their home, including some with lower limb injuries who required admission. Comparing the injury rate with that in some other sports, ice skating is relatively safe. Contact sports such as rugby union and rugby league have injury rates of 200 and 580 injuries per 10,000 playing hours respectively,[7] compared with 20 injuries per 10,000 playing hours on the ice, assuming that the skater remains on the ice for one hour each session.

Skating will remain a popular leisure activity, because of its social nature, and the various activities that can be pursued on the ice, such as hockey or curling. This

was confirmed when 355,000 skaters attended the rinks in the first six months. The nature of the sport makes falls inevitable. Suggestions have been made before on the use of protective clothing, and improving that now available, as well as adding more protection to the barriers around the ice rink. At the very least, wrist splints could be offered, but to date no controlled trial has been carried out to show if protective clothing would be of benefit. Other measures have been suggested, such as limiting the numbers on the ice at any one time, but this has not been supported.[1, 4] Segregation of beginners from those more competent may be more relevant in this respect.

These two ice rinks have had a major effect on the workload on both the Accident and Emergency Department and the Orthopaedic Department in the Ulster Hospital. Since the Hospital is so close to the rinks, liaison between the rink management and the hospital staff would be appropriate to discuss methods of prevention of injury; additional funding to the hospital from the Health Authority or even from the owners of the ice rink should be considered since more than 1,000 new patients each year may be expected as a result of injuries sustained on the ice.

I am grateful to all the consultant orthopaedic surgeons at the Ulster Hospital for allowing access to their patients, and to Professor B J Rowlands, Department of Surgery, The Queen's University of Belfast, for his help and encouragement in preparing this manuscript.

REFERENCES

1. Freeland P. Implications of two newly opened ice rinks on an accident and emergency department. *Br Med J* 1988; **296**: 96.
2. Horner C, McCabe MJ. Ice-skating and roller disco injuries in Dublin. *Br J Sports Med* 1984; **18**: 207-11.
3. Prescott MV. The effect of opening an ice-rink on the accident and emergency department of a district general hospital. *Arch Emergency Med* 1986; **3**: 107-10.
4. Williamson DM, Lowdon IMR. Ice-skating injuries. *Injury* 1986; **17**: 205-7.
5. Goodwin Gerberich S, Finke R, Madden M, Priest JP, Aamoth G. An epidemiological study of high school ice hockey injuries. *Childs Nerv Syst* 1987; **3**: 59-64.
6. Jorgensen U, Schmidt-Olsen S. The epidemiology of ice hockey injuries. *Br J Sports Med* 1986; **20**: 7-9.
7. Walker RD. Sports injuries: rugby league may be less dangerous than union. *Practitioner* 1985; **229**: 205-6.

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Hepatitis B virus infection in Northern Ireland 1970–1987

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SUMMARY

In the 18 years between 1970 and 1987, 504 patients were found to have hepatitis B surface antigen (HBsAg) in their blood. Acute hepatitis was present in 184 patients and six died (3·3%). The annual incidence of acute hepatitis B virus infection in Northern Ireland was about one-quarter that of England and Wales. A decrease in acute infection occurred in 1986–87, while in England and Wales acute infection has fallen by more than half since the peak in 1984. Hepatitis B virus infection in health care staff and patients in high risk groups were reviewed: 32% were in those of foreign origin or who had known foreign contacts. In blood donors there was a marked fall in incidence of hepatitis B surface antigen carriage from 1982 onwards: the incidence in antenatal patients and those screened for rubella antibody (mainly females) was half that of new blood donors in 1972–81. Carrier rates in blood donors and antenatal patients were less than those from other parts of the United Kingdom. All indices show that Northern Ireland has a lower incidence of hepatitis B virus infection than the rest of the United Kingdom.

INTRODUCTION

Hepatitis B virus infection is a large problem in the world. It has been estimated that there are in excess of 200 million human carriers, but the distribution of these carriers is uneven. The prevalence of carriers in northern Europe, North America and Australia is 0·1% or less, but rises to 20% or more in some parts of Africa, Asia and in the Pacific region. Hepatitis B vaccine is now available but it is expensive. A knowledge of local epidemiology is important to assess whether vaccine is required.

PATIENTS, MATERIALS & METHODS

Testing for hepatitis B surface antigen began in the Regional Virus Laboratory in 1970 in blood donors required for the Renal Unit, Belfast City Hospital. The immunodiffusion test was used initially; in 1974 this was replaced by the more sensitive reverse passive haemagglutination test (RPHA) (Hepatest-Wellcome) and from 1984 onwards the very sensitive enzyme-linked immunosorbent assay (ELISA) (Hepanostika-Organon Teknika) was used. The Regional Virus

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Laboratory receives specimens from patients with clinical or suspected hepatitis from hospitals and general practitioners throughout Northern Ireland.

Over 5,000 patients are screened for HBsAg each year. Patients and staff in the Renal Unit, Belfast City Hospital, are screened, as are all donors of organs and sperm, and certain population group surveys. In certain cases other markers of hepatitis B virus infection are tested.

The Northern Ireland Blood Transfusion Service began routine screening of all blood donors in 1972 using the immunoelectro-osmophoresis test. The RPHA test was introduced in 1975 and the radioimmunoassay test (Blood Products Laboratory, Elstree) in 1982. An ELISA test (Wellcozyme) has been used from July 1987. Screening of antenatal patients began in 1973 and of blood samples sent for rubella screening in 1982. Positive blood samples were referred to the Regional Virus Laboratory for confirmation. All hepatitis B surface antigen positive patients were reported confidentially to the Public Health Laboratory Service, Communicable Disease Surveillance Centre, in London.

RESULTS

HEPATITIS B SURFACE ANTIGEN IN PATIENTS AND CARRIERS

There were 504 patients and carriers identified in the 18 years from 1970 to 1987; 282 (56%) were detected by the Regional Virus Laboratory and 222 (44%) by the N I Blood Transfusion Service.

The annual incidence for each laboratory is shown in the Figure. Both showed a variable pattern; the peak years were 1983 and 1985. The age of blood donors is not usually recorded; however, the age distribution of 350 patients and carriers ranged from newborn to 74 years old. The highest incidence was in the age group 20–29 years and 60% of all patients were male.

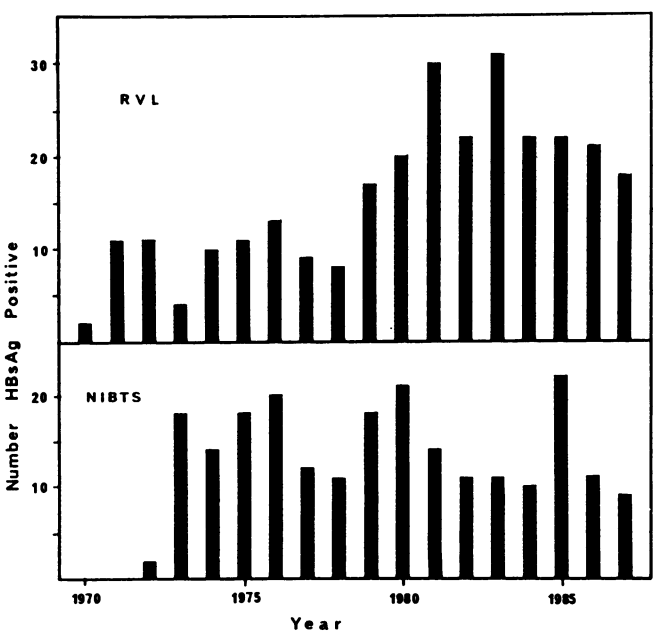

Figure.
Number of hepatitis B surface antigen (HBsAg) positive patients identified at the Regional Virus Laboratory (RVL) and the Northern Ireland Blood Transfusion Service (NIBTS) from 1970 to 1987.

The categories of patients who were HBsAg-positive and the number of patients who had acute hepatitis B virus infections are shown in Table I. There were 184 patients with symptoms or signs of acute hepatitis, accounting for 65% of the total diagnosed by the Regional Virus Laboratory (131 males and 53 females). The average annual incidence of acute infection per 100,000 population was 0·95 for males and 0·37 for females. There were six deaths, a mortality rate of 3·3%. In the age group 15–64 years there were 125 males and 50 females, an average annual incidence per 100,000 population of 1·48 for males and 0·58 for females. Three hundred and twenty patients were carriers in high risk groups or were detected on screening.

TABLE I

Categories of patients infected with hepatitis B virus

	Number with acute hepatitis (deaths in brackets)		*Total including carriers*	*%*
Regional Virus Laboratory				
Health care workers and spouses	18	(1)	25	5·0
Haemophiliacs	11	(1)	11	2·2
Received multiple transfusions	8		8	1·6
Recently tattooed	8		8	1·6
Post-surgery	20		24	4·8
Intravenous drug abusers	17		22	4·4
Renal unit patients	4		4	0·8
Mentally handicapped patient	0		1	0·2
Homosexual/bisexual contacts	8		17	3·3
Heterosexual contacts	12	(1)	16	3·2
Babies and children of known carrier mothers	1		16	3·2
Foreign-born children	0		18	3·5
Foreign-born adults or foreign contact	25		44	8·7
Donors of organs and tissues	0		3	0·6
Others	52	(3)	65	12·9
N I Blood Transfusion Service				
Blood donors	—		117	23·2
Antenatal patients	—		90	17·8
Patients screened for rubella antibody	—		15	3·0
Totals	184	(6)	504	100·0

HEALTH CARE WORKERS AND SPOUSES

Medical and dental: There were five surgeons or trainee surgeons with hepatitis B virus infection but none had been infected in Northern Ireland — India (one acute), Republic of Ireland (one acute), Africa (two carriers), and the Far East (one carrier). Two wives were infected: one had acute hepatitis and the other was a carrier. Three other doctors had been infected, one acutely in a Dublin neonatal unit, one in a London liver unit (carrier) and one in Africa (carrier). Only one

dentist was infected and died of acute hepatitis aged 63 years, but there had also been a history of receiving recent treatment from an unlicensed electrolysis parlour.

Nurses: Four nurses had acute hepatitis B virus infection while working in Northern Ireland. These were a midwife, a nurse who was vomited over by a known carrier, a nurse who received a needlestick injury from an acutely infected patient in the Renal Unit, and a male nurse who gave a history of needlestick injury. None had received prophylaxis with anti-hepatitis B immunoglobulin. In addition, a nurse with acute hepatitis had been infected in Saudi Arabia, and another nurse, a carrier, had been infected in Hong Kong. The average number of nurses in Northern Ireland per year between 1970 and 1987 was 14,980, giving an average annual incidence of acute hepatitis B virus infection of 1·48 per 100,000.

Laboratory staff: Four laboratory staff had acute infections: three worked in clinical chemistry and one in a haematology laboratory. Three became infected between 1971 and 1974 and the fourth in 1985. In addition, a laboratory worker in a Jamaican laboratory had acute hepatitis B virus infection. The mean number of laboratory staff in Northern Ireland between 1970 and 1987 was 542 per year, giving an average annual incidence of acute infection of 41 per 100,000.

Others: A hospital cleaner with a history of needlestick injuries developed acute hepatitis B virus infection. A hospital administrator in Oman and a hospital worker in Papua New Guinea also had acute infections while in Northern Ireland.

BLOOD AND BLOOD PRODUCTS TRANSMISSION

These high risk groups include those who within the previous six months had received blood or blood products, multiple transfusions, had been tattooed or had surgery, or were intravenous drug abusers.

Haemophiliacs: Acute infections occurred in 11 patients between 1972 and 1982 after receiving blood transfusions, cryoprecipitate or factor VIII, and one patient died aged 51 years.

Multiple transfusions: Acute infections took place between 1970 and 1980 in eight patients who had received multiple transfusions after surgery.

Tattoos: Seven males and one female had acute infections following tattooing. Two had been tattooed in Singapore and one in Hong Kong.

Post-surgery: There were two clusters of five patients each, and a third cluster of three patients who were associated with the use of an inadequately sterilised reusable piece of equipment. Five other patients had received dental treatment only but there was no evidence of clustering.

Intravenous drug abusers: Four patients had known contacts in London and one in Dublin.

RENAL UNIT

In July 1971 hepatitis B surface antigen was first discovered in a patient with chronic renal failure on haemodialysis. Two further patients were detected in September and a fourth patient in October. All patients carried the 'd' subtype of HBsAg. The first three patients were jaundiced. These patients did not share any equipment, but the first had received blood transfusions in a peripheral hospital and two others received blood transfusions in the Renal Unit before becoming

HBsAg positive. The first patient refused further haemodialysis and died, and the other three patients remained carriers for short periods (6–15 weeks) before becoming negative. While they were positive the four patients were isolated and dialysed in a separate Portakabin until they became negative. During the outbreak, staff and patients were screened weekly. A nurse received a needlestick injury from the fourth patient in October 1971 and became positive for 11 days only, two months later; she felt ill at the time, but liver function tests remained normal.

HOSPITAL FOR THE MENTALLY HANDICAPPED

Hepatitis B virus markers of infection were investigated in 720 patients in the largest hospital for the mentally handicapped in Northern Ireland during 1987. Only one HBsAg carrier was found (0·14%) who was hepatitis B 'e' antigen (HBeAg) negative and antibody to hepatitis B 'e' antigen (anti-HBe) positive which indicated low infectivity.

SEXUAL AND PERINATAL SPREAD

Of the 33 homosexual/bisexual and heterosexual contacts with hepatitis B virus infection, one heterosexual man died aged 32 years. Twelve infants and four young children were positive for HBsAg whose mothers were also known carriers. Twelve of the mothers were local, two Chinese, one African and one Afghan.

FOREIGN-BORN OR FOREIGN CONTACTS

There were 161 people (32%) infected with hepatitis B virus who were foreign-born or had foreign contacts. The two main foreign groups were Chinese (73) and Vietnamese (32). Some of these patients were classified in other high risk groups. Since 1982, about half of all hepatitis B virus infected persons diagnosed have been of foreign origin. Ninety were diagnosed by the Regional Virus Laboratory. The main groups were 21 Chinese and 23 Vietnamese, including 18 children under 18 years of age of whom 15 were Vietnamese and three were Chinese. These were additional to the 16 babies and young children infected perinatally. The NI Blood Transfusion Service detected 71 foreign-born people who were carriers. Twelve were blood donors, 48 antenatal patients and 11 were being screened for rubella antibody; 52 were Chinese and nine Vietnamese. In addition there were 37 HBsAg positive blood donors who were not indigenous Northern Ireland inhabitants.

DONORS OF ORGANS AND TISSUES

All organ and sperm donors are screened for hepatitis B surface antigen and antibody to human immunodeficiency virus. Two potential donors of ear ossicles and one potential kidney donor were HBsAg carriers.

OTHERS

There remained 65 (12·9%) patients investigated by the Regional Virus Laboratory who were infected with hepatitis B virus where a risk factor was not identified. There were three deaths in this group (two males aged 50 and a female aged 70 years).

BLOOD DONORS

The number of blood donors tested each year ranged from 50,006 in 1974 to 66,614 in 1986. The number of 'new' donors (previously untested for HBsAg) ranged from 9,302 in 1973 to 17,266 in 1987. Potential donors with a history of

hepatitis or jaundice during the previous 12 months were excluded, as were those with other risk factors for hepatitis B virus infection during the previous six months. Exclusion of those belonging to high risk groups for AIDS was introduced in 1983. The results of screening for hepatitis B surface antigen are shown in Table II.

TABLE II

Incidence of hepatitis B surface antigen (HBsAg) carriage in blood donors, antenatal patients and patients screened for rubella antibody

Category screened	*Years*	*Number tested*	*Number HBsAg positive*	*Incidence*
New blood donors	1972–81	130,459	82 (41)	1:1,591 (1:3,182)
	1982–87	79,195	16 (8)	1:4,950 (1:9,899)
Prior blood donors	1972–81	419,779	(17)	(1:24,693)
	1982–87	305,220	(2)	(1:152,610)
Antenatal patients and Patients screened for rubella antibody	1973–87 1979–87	375,760	105 (46)	1:3,759 (1:8,169)

Figures in brackets refer to indigenous Northern Ireland inhabitants.

One hundred and seventeen donors were carriers. In the new donors there was a threefold drop in incidence from 1972–81 to 1982–87. For the same two periods the incidence of HBsAg in donors found HBsAg negative on previous donations (prior donors) showed a sixfold drop.

Included in the new donors screening positive were 12 people of foreign origin and 37 transient residents from other parts of the United Kingdom. The number of these non-indigenous people tested was small in relation to the total number of new donors and, if these 49 positives are removed from the total, it halves the incidence, which corresponds more closely to the incidence in the indigenous population.

ANTENATAL PATIENTS AND PATIENTS SCREENED FOR RUBELLA ANTIBODY

All the positive antenatal patients were first-time positives and did not include those positive in a previous pregnancy. Ninety antenatal patients and 15 patients screened for rubella antibody were carriers. The incidence of hepatitis B surface antigen in this predominantly female group is less than half that of the new blood donors in the period 1972–81, and there was no drop in incidence between 1973–81 and 1982–87. Fifty-nine were of foreign origin; if these are excluded from the total the incidence again corresponds more closely to the incidence in the indigenous population.

PASSIVE AND ACTIVE IMMUNISATION

Human anti-HBs globulin was used in a Medical Research Council trial after inoculation accidents from 1973. It was available in limited amounts for clinical trial from the Blood Products Laboratory, Elstree, for accidental exposure from 1978. From 1983 it was obtained from the Scottish National Blood Transfusion

Service. To date, no one in Northern Ireland has developed hepatitis B virus infection who had received anti-HBs globulin following accidental exposure to infected patients. Plasma-derived hepatitis B vaccine (Merck, Sharp & Dohme Ltd) was available in 1982 but was expensive at £63.50 per course. Genetically engineered hepatitis B vaccine (Smith, Kline & French Ltd) became available in 1987 and was less expensive (£31.50 per course). At about this time the cost of plasma-derived vaccine was reduced to £36.22 per course. Very little vaccine was used in Northern Ireland up until 1987.

DISCUSSION

The main methods of hepatitis B virus spread are inoculation of infected blood and blood products, and sexual intercourse. During the period under study, there has been a continuous improvement in work practices to prevent infection in health care facilities, and more sensitive tests have been developed for the detection of hepatitis B surface antigen. Both these factors have contributed to greater safety. There was a steady increase in the number of positive patients in Northern Ireland until about 1985 since when the numbers have fallen by about 40%. There was an increase of acute cases in England and Wales which reached a peak of 2,000 cases in 1984 but then dropped sharply to about 800 cases in 1987.[1] Warnings about the risk of acquiring AIDS may have led to a decrease in drug abuse by injection and a modification of behaviour by homosexual men. The peak incidence in Northern Ireland was in the 20–29-year-old age group and males were more commonly infected than females. Other studies have shown similar results.[2]

Clinically only 184 patients (36%) had symptoms and signs of acute hepatitis B virus infection, the rest being carriers, which highlights the necessity of being aware of infection in high risk groups who are otherwise well. The average annual rate of acute infection in the 15–64-year-old age group per 100,000 population was 1·48 for males and 0·58 for females which is about one-quarter the incidence found in England and Wales in the same age group.[2]

The risk to health care staff was small. No surgeon or doctor was first infected while working in Northern Ireland. One dentist became infected and died, but there was strong epidemiological evidence that this infection was by means other than practising dentistry, which emphasises the danger of procedures which puncture the skin with instruments which may be inadequately sterilised. However, there was an increased risk when surgeons operated in areas of the world where the carrier rate was high, and patients who come from such countries to Northern Ireland for surgery present a greater hazard than the local population. Surgeons as a group in England had an annual incidence of 25/100,000 during 1980–84 which had doubled since 1975–79, while dentists had an annual rate of 17/100,000.[2]

Four nurses developed acute infection, two of them after needlestick injuries. Approximately 40% of self-inoculation accidents occur while resheathing needles using both hands and this should not be done unless there is a safe means available.[3] Needles may be resheathed safely using a one-handed method and the needle should remain safe once it has been disposed of into a 'sharps' container in the ward and thereafter until final disposal outside the hospital. Education plays a very important part in the safe disposal of contaminated 'sharps' and all members of the health care professions must be taught how to do it correctly. Anti-HBs globulin should be given to the person suffering the

needlestick injury if the source of the needle is known to be a hepatitis B positive person. The annual incidence of 1·48 nurses per 100,000 in Northern Ireland is less than that found in the female population aged 15–64 years in England and Wales, and is three to five times less than that found in nurses in England (7/100,000 in 1975–79 and 4/100,000 in 1980–84).[2] The fall in incidence in England was probably due to the adoption of safer working practices.

The case of a hospital cleaner who developed hepatitis after a needlestick injury emphasises that resheathing needles safely should prevent such injuries in people 'downstream' in the disposal chain from those who used the needles. The risk of acquiring hepatitis B virus infection following an accidental needlestick injury from a carrier ranges from 6% to 30%, which is far in excess of the risk of human immunodeficiency virus infection following a similar injury with an infected patient where the risk is $<1\%$.[4] Four laboratory staff became infected with hepatitis B virus. Statistics from England indicate that the annual rate in laboratory staff doubled from 18 to 37/100,000 between 1975–79 and 1980–84,[2] while in Northern Ireland three of the four cases occurred between 1971 and 1974. Nevertheless the overall incidence for Northern Ireland is similar to that of England in 1980–84.

Transmission of hepatitis B virus by blood and blood products such as cryoprecipitate and factor VIII has almost ceased following the screening of blood donors and the development of more sensitive and specific tests. There was no evidence that haemophilia patients became infected after 1982 and patients who received multiple transfusions after 1980 did not have acute hepatitis B virus infections. Tattooing is a high risk procedure, particularly if carried out abroad in a country where the incidence of hepatitis B virus infection is increased. Any procedure which uses unsterilised instruments such as ear-piercing, acupuncture, electrolysis, unregistered chiropody and accidental skin piercing in hairdressing and barbers carries a risk. The association of infection with previous surgical procedures is difficult to prove. There was probably only a chance association in those patients who gave a history of dental treatment in the previous six months, since dental treatment is common and there was no obvious 'clustering' of patients. There were three 'clusters' of cases identified involving a total of 13 patients. Advice was given to the staff involved, measures to preserve asepsis were instituted, and no further cases were indentified. It has been estimated that in England and Wales the average annual risk of a patient developing acute hepatitis B virus infection as part of a cluster caused by staff during surgical procedures was one in a million operations.[5]

Intravenous drug abuse is a small problem in Northern Ireland. The number of addicts notified was 13 in 1985 and 11 in 1986.[6] However, there are large numbers of intravenous drug abusers in Dublin, Edinburgh, Glasgow and London, and it is not known how many contacts are made with these cities. Drug abuse was the commonest high risk group in England and Wales and accounted for 23·9% of all cases of acute hepatitis B virus infection reported to the Public Health Laboratory Service in 1980–84.[2] Renal units are often cited as having a high risk of transmission to patients and staff, but while this may be true of other countries it does not now apply to the United Kingdom. Following a prevention and control programme instituted in 1971, the Renal Unit at the Belfast City Hospital has been free of hepatitis B virus infection since 1972. This programme included screening of blood donors, screening of patients monthly and staff every three months, and the isolation of infected patients in 1971 until they became

HBsAg-negative. Similar programmes were carried out throughout the United Kingdom which resulted in the almost complete elimination of hepatitis B from dialysis units.[7, 8]

Psychiatric hospitals are also regarded as having an increased risk of hepatitis B virus infection for patients and staff. In the largest hospital for the mentally handicapped in Northern Ireland only one out of 720 patients (0·14%) screened was found to be an HBsAg carrier of low infectivity. This contrasts markedly with 9% of patients found to be chronic carriers in a hospital for the mentally handicapped in the Republic of Ireland[9] and 7·6% in a hospital for the mentally subnormal in Wessex, Southern England.[10]

Hepatitis B can be spread sexually; male homosexual/bisexuals and heterosexual contacts with cases or carriers accounted for 6·5% of the total. In a study by the Public Health Laboratory Service male homosexuals alone were 7·8% of the total.[2]

Sixteen babies positive for HBsAg had mothers who were also positive antenatally. These children usually become lifelong carriers.[11] Transmission is mainly perinatal and intrauterine infection is rare. Transmission is likely if the mother has an acute infection in the last trimester of pregnancy or early in the puerperium. Mothers who are HBsAg carriers and are HBeAg-positive or who are both HBeAg- and anti-HBe-negative have an increased risk of transmitting hepatitis B virus infection to their babies. Transmission from mother to child is lower in Europe and North America than in the Chinese race. However, transmission has occurred from white women to their babies in Northern Ireland.[12]

One hundred and sixty-one (32%) of those found to be HBsAg-positive were foreign-born or had foreign contacts and 105 (78%) of these were of Chinese or Vietnamese origin. While both groups constitute a very small minority in the Northern Ireland population, they have contributed substantially to the total number of cases. Only three potential organ donors were found to be HBsAg-positive but it is essential to maintain an emergency laboratory service for testing organ donors for both HBsAg and anti-HIV. Since a hepatitis B virus infected donor could donate at least both corneas and both kidneys, four recipients could be infected.

All patients should be treated as if they were carriers of both the hepatitis B and the human immunodeficiency viruses and suitable precautionary measures taken when dealing with blood or other body fluids — wearing gloves, gowns or aprons and eye protection as well as avoiding skin puncture from contaminated 'sharps'.

Although blood donors are self-selected and not quite typical of the general population, new donors of the indigenous population in the early years of testing (1972–81) may be as close as one can get to the HBsAg carrier incidence in the population. The threefold fall in incidence between 1972–81 and 1982–87 may be caused by further self-selection of males due to publicity about AIDS, which started about that time. A similar drop in incidence was seen in the prior donors between the same two periods. The local incidence of HBsAg positive tests in new donors (1:1,590 or 1:3,180 for the indigenous population) is less than the 1:562 found by North London Blood Transfusion Centre, Edgeware,[13] or the 1:800 found by the Glasgow and West of Scotland Blood Transfusion Service,[14] but is similar to the 1:1,670 found in the Oxford region in 1973–84.[15] The 1:24,690 incidence in prior donors is, however, a measure of the very low incidence of HBsAg infection occurring in previously HBsAg-negative individuals

and is lower than the 1:16,000 found in the Glasgow and West of Scotland Blood Transfusion Service.[14]

A fall in number of positive HBsAg tests did not occur during 1982–87 in the antenatal patients or in the predominantly female patients screened for rubella antibody. This again suggests that the fall observed in the blood donors during this period was due to self-exclusion of male donors. The Northern Ireland incidence of positive tests in the antenatal patients and the patients screened for rubella antibody was less than half that found in new donors during 1972–81 and again confirms the two–threefold lower incidence of hepatitis B virus infection in females. The incidence is five times lower than that detected in Oxfordshire and Northamptonshire,[15] eight times lower than in the West Midlands,[16] and 20 times lower than in London.[13] In the indigenous Northern Ireland population the incidence is still lower.

The use of vaccine should be based on local epidemiology. The small groups who really are at increased risk might be offered vaccine, rather than the blanket coverage at present recommended. This strategy is logical and cheaper, but it is more important that safe working practices should be taught since good technique protects against all microbiological hazards.[17]

We thank the hospital consultants and general practitioners throughout Northern Ireland who provided epidemiological details on their patients, and Dr S N Donaldson, Department of Health and Social Services (Northern Ireland), for data on nurses and laboratory staff. The serological survey in the hospital for the mentally handicapped was part of a co-operative study with Dr F Kee, Registrar in Community Medicine, Northern Health & Social Services Board.

REFERENCES

1. Polakoff S. Decrease in acute hepatitis B incidence continued in 1987. *Lancet* 1988; **1**: 540.
2. Polakoff S. Acute viral hepatitis B: laboratory reports 1980–84. *Br Med J* 1986; **293**: 37-8.
3. Advisory Committee on Dangerous Pathogens. LAV/HTLV III, the causative agents of AIDS and related conditions — Revised guidelines. London: 1986.
4. Anonymous. Recommendations for preventing transmission of infection with human T-lymphotrophic virus type III/lymphadenopathy-associated virus in the workplace. *MMWR* 1985; **34**: 681-95.
5. Polakoff S. Acute hepatitis B in patients in Britain related to previous operations and dental treatment. *Br Med J* 1986; **293**: 33-6.
6. Advisory Council on the Misuse of Drugs. Aids & drug misuse. Part 1—Report by the Advisory Council on the Misuse of Drugs. London: HMSO, 1988.
7. Decrease in the incidence of hepatitis in dialysis units associated with prevention programme — Public Health Laboratory Service Survey. *Br Med J* 1974; **4**: 751-4.
8. Hepatitis B in retreat from dialysis units in United Kingdom in 1973 — Public Health Laboratory Service Survey. *Br Med J* 1976; **1**: 1579-81.
9. Lyons R, Kelly P, Hobdell M, Gavin G, Clancy L. Hepatitis B infection in the residential mentally handicapped population. *Ir Med J* 1987; **80**: 410-1.
10. Kingham JGC, McGuire M, Paine DHD, Wright R. Hepatitis B in a hospital for the mentally subnormal in southern England. *Br Med J* 1978; **2**: 594-6.
11. Wheeley SM, Boxall EH, Farlow MJ. Prognosis of children who are carriers of hepatitis B. *Br Med J* 1987; **294**: 211-3.

12. Bharucha C, Crowley D, McClelland M, Crawford RJ. Perinatal transmission of hepatitis B in Northern Ireland. *Br Med J* 1983; **286**: 439.

13. Cameron CH, Combridge BS, Howell DR, Barbara JAJ. A sensitive immunoradiometric assay for the detection of hepatitis B surface antigen. *J Virol Methods* 1980; **1**: 311-23.

14. Crawford RJ, Mitchell R. Hepatitis and blood donation. *Br Med J* 1980; **280**: 47.

15. Vickers MA, Puckett AY, Bowell PJ. Prevalence of HBsAg in UK population. *Br Med J* 1987; **294**: 57.

16. Boxall EH, Flewett TH. Prevalence of HBsAg in UK population. *Br Med J* 1987; **294**: 57.

17. Gatley MS. Time for action on hepatitis B vaccination. *Br Med J* 1987; **294**: 509.

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Trench fever in Belfast, and the nature of the 'relapsing fevers' in the United Kingdom in the nineteenth century

J S Logan

SUMMARY

Some evidence is assembled to suggest that trench fever, an infection with a strain of Rochalimaea, *if not* quintana, *then* vinsonii, *was present in Belfast in the first half of the nineteenth century in endemic and epidemic form. It may have amounted at times to one half or more of 'fever'. This may account for the comparatively low mortality in some years from 'fever'.*

The phrase 'relapsing fever' in the nineteenth and twentieth century medical literature of the United Kingdom should not be taken necessarily to mean infection with Borrelia recurrentis. *Much or most may have been infection with* Rochalimaea, quintana *or* vinsonii. *The newly discovered Irish vole should be examined to see if it carries a Rickettsia or Rochalimaea infection.*

Trench fever, a louse-borne disease due to infection with *Rochalimaea quintana,*[1] came to the notice of the modern medical profession in 1914–1918, when huge epidemics occurred in the Allied and German Armies in Europe.[2, 3, 4, 5] It was a major cause of loss of manpower. At one time in 1917 the rate of loss in the British Army was calculated to be 45,000 in a million men in a year.[4] In spite of the sharpness of the fever and some prolonged invalidism, it had no mortality. It had long existed in Europe and elsewhere and still has a world-wide distribution where lousiness persists.[6] In the early decades of the nineteenth century it was not distinguished from the mass of 'fever', and, when 'relapsing fever' was distinguished from 'continued fever', predominantly typhus, trench fever then was not distinguished from the relapsing fever caused by *Borrelia recurrentis* infection. Diagnosis was difficult because of the varying clinical pattern of trench fever.[5] The most common was a relapsing fever, but it also could be a five-day fever and occasionally a prolonged irregular fever. Only recently have cultural[7] and serological[8] methods of diagnosis been developed, and they are probably not available routinely where cases are still found. Moreover, when louse-borne diseases were epidemic, mixed or successive infections of typhus, Borrelian relapsing fever and trench fever must have been frequent. Reid observed such.[9] During the Great War, trench fever had a period of intense attention and research. When peace came it was forgotten in the United Kingdom. Complete recovery was usual and no case died. A few ex-soldiers had war pensions for disability attributed to trench fever. Byam remarks that 'many patients continue in ill-health for months after the acute febrile stage is past, some of them relapse with marked fever from time to time, whilst others pass into a condition which is

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variously called myalgia, neurasthenia or disorderly action of the heart'.[2] This opinion remained a matter of debate. Hurst disagreed.[10]

This note examines the possibility that trench fever or a trench fever-like illness was common in Belfast in the last century. Recent reports suggest that a strain of *Rochalimaea* other than *quintana* could have been responsible.[11]

Undifferentiated 'fever', endemic and epidemic, was the main health problem of the early nineteenth century. It was to meet this danger that the Belfast Fever Hospital was founded, and for years it was only reluctantly and infrequently that 'ordinary' medical and surgical cases were admitted. The Hospital Report of 1818–19 says 'Fever still is, and ought to be, the great and predominant subject of solicitude with the public'. In 1820–21 it calls fever 'our dreadful enemy'. In 1821–22 fever is 'this fearful assailant'. In 1819–20 estimates of those stricken with fever, in Belfast's population of 30,000, range from 4,000 to 10,000, and 7,000 was thought most near the truth. It is in this mass of 'fever' that one must look for trench fever.

One must remember what 'fever' meant for the physicians of the time, especially in the early decades. The term did not include the pyrexial illness of smallpox, measles, scarlatina, dysentery, tuberculosis, tonsillitis, abscess or wounds. In fever the patient was generally ill, affected with rigors, languor, stupor, headache, pains in the back and loins, giddiness, anorexia, thirst, burning heat over the body, a quick pulse and incapacity for exertion.[12] This was before the clinical thermometer came into regular use.[13] Neither clinically not post mortem could a focal lesion be found.[14] One cannot doubt from the clinical descriptions of the time that typhus, with its severe illness, its complications of vasculitis and gangrene, and its heavy mortality was the cause of many cases of 'fever'. Equally one does not doubt that many were cases of Borrelian relapsing fever, also with a heavy mortality and often complicated by hepatitis and jaundice. This was the 'yellow fever of Ireland', a term which has given rise to confusion with viral yellow fever of the Caribbean.[15] Brucellosis and Q fever must have been present but it is not in their nature to produce numerically large epidemics. It is impossible to identify typhoid fever in the Fever Hospital reports up to 1850, though that is not to say that it did not exist. 'Typhoid' is sometimes used as an adjective from typhus. The fever now known as typhoid had been described by Louis in 1829 (in the first edition of his book) together with the local pathology in the small intestine.[16] That is why Little looked so carefully at the small intestine when making his post-mortem examinations.[14] By 1860 J S Reid was satisfied that typhoid fever existed in Belfast and was certain that he had previously classified many cases of it as synochus.[17]

One begins to doubt, however, that typhus and Borrelian relapsing fever were the only or main causes of 'fever' when one notes the mortalities. The mortality of true, untreated typhus according to Osler varied from 10% to 20% and after middle age was as high as 50%.[18] Because much of the argument turns on death rates, it is right to quote Megaw at length on the mortality of untreated typhus.[19] The case fatality, he says, depends greatly on age. It is almost negligible in children. It is seldom more than 5% below age 20. At age 40 it is 10% to 15%; at age 50, 50%. Over 60, few recover. Persons debilitated by starvation bear the disease badly. Deaths are far more numerous in severe epidemics occurring in half-starved and crowded communities composed chiefly of adults. In such conditions the death rate may be as high as 40% or even 50%. In outbreaks in endemic areas the average rate may be as low as 5% to 10% if all the mild cases

are taken into account. Malcolm[20] tells us that in the early decades of the last century in Belfast 'the masses' lived 'in penury, in sickness, in wretchedness and woe, unpitied — nay unknown'. McCormac, the Belfast physician, said 'the physical condition of man, in these regions at least, so far as my own observation and that of travellers extends, is considerably inferior to that of the rude aborigines of Africa or America'.[12] McCormac had travelled in West Africa and in America. Belfast then could not expect a good recovery rate in typhus. Doberstyne reports the mortality in louse-borne Borrelian relapsing fever as from 10% to 50%.[21] In contrast, the mortality in the Belfast Fever Hospital in some years was so low as to exclude typhus and Borreliosis being the cause of the great majority of the cases. (Table). Good nursing will not account for the low mortality and there was no specific drug treatment. For instance in 1817–18 deaths from fever were 4·9% of 1,582 patients, in 1818–19 4·4% of 1,391 patients, in 1819–20 3·8% of 733 patients, in 1820–21 4·1% of 762 patients, 1827–28 4% of 709 patients, 1828–29 3·9% of 514 patients. The highest mortality was in 1847–48, 13·4% of 5,153 patients, when, the report says, there was a universal predominance of maculated typhus. McSkimin has recorded the mortality in the Carrickfergus Fever Hospital in 1817–18 as 5%.[22] When the Belfast Fever Hospital opened in 1797 in Berry Street there was only one death in the first 60 cases.[20] Whatever the skill of Doctor Stephenson and Doctor McDonnell, the attending physicians, it is impossible to believe that they were treating typhus or Borrelian relapsing fever.

Doctor S S Thomson, writing the Belfast Fever Hospital report for 1828–29, gives a clinical account indistinguishable from trench fever. 'The chief peculiarity observable in the cases which have come under treatment was the great tendency to come to a premature crisis. The ordinary duration of our fever is from 11 to 14 days and sometimes it runs out still longer. In many instances last year it was over in 5, 7 or 9 days; such cases were peculiarly liable to relapse. The slightest error on the part of the patient in making too free was sure to light up a new febrile accession, and this not once but repeatedly. Also relapses occurred without any ascertainable cause. Cases of this description proved extremely teazing but seldom or never fatal'. This was the year when the mortality was 3·8%.

J S Reid, the great physician and diagnostician, writing the Fever Hospital report in April 1844, records that, of 537 cases of fever in his own care, 60 were cases of typhus of whom 10 died (16%) and 477 were 'synocha', a 'relapsing fever' of whom 14 died (2·9%). Evidently Reid's 477 cases with the 'relapsing fever' were mostly not Borrelian relapsing fever. Evidently it has been an error to assume that all or most of the 'relapsing fever' in Ireland was Borrelian. If not that, what was it? The clinical features, the minimal or zero mortality, the epidemic character and the prevalence of lice are consistent with trench fever. It is true that some of these mild cases of fever might be Brill-Zinsser recrudescences of true typhus but these do not have a relapsing character.

The effect of the failure to distinguish typhus fever, Borrelian fever and trench fever from each other was to lower the total death rate from 'fever' by diluting the substantial death rates of typhus and Borrelian fever with the zero death rate of trench fever. And to produce an apparent death rate in trench fever which should not have one.

Further evidence lies in the work of Baker in Canada,[23] and later of Weiss. Grosse Isle is a small island, a mile long and half-a-mile wide, only 320 acres, in the St Lawrence river, 29 miles downstream from Quebec. In 1942 during the last

TABLE

Belfast Fever Hospital Reports

Year	*Fever patients*	*Fever deaths*	*% deaths*	*Remarks*
1817–1818	1,582	79	4·9	
1818–1819	1,391	62	4·4	
1819–1820	733	28	3·8	
1820–1821	762	32	4·1	
1821–1822	276	19	6·8	
1822–1823	337	27	8·0	
1823–1824	214	21	9·8	
1824–1825	415	21	5·0	
1825–1826	356	19	5·3	
1826–1827	907	52	5·7	
1827–1828	709	29	4·0	
1828–1829	514	20	3·9	
1829–1830	244	10	4·0	
1830–1831	582	40	6·8	
1831–1832	1,061	73	6·8	
1832–1833	598	53	8·8	
1833–1834	510	43	8·4	
1834–1835	691	62	8·9	
1835–1836	575	48	8·3	
1836–1837	1,941	181	9·3	Many fever patients 'fell victims to the influenza'
1837–1838	3,363	402	11·9	Erysipelas in the hospital increased deaths in fever patients
1838–1839	1,162	113	9·7	The figures are useless because in this year scarlatina, measles, smallpox, erysipelas were included in the list of fever patients
1839–1840	1,820	206	11·3	A few of the total fever patients were not 'fever' but the fever deaths are correct
1840–1841	1,822	166	9·0	
1841–1842	1,241	126	10·1	Some uncertainty about the diagnosis in some of the fever patients
1842–1843	707	70	9·9	
1843–1844	2,284	133	5·8	See text for Reid's analysis of his cases. Were most trench fever?
1844–1845	1,864	126	6·7	'The great mildness of the symptoms of the epidemic fever'
1845–1846	740	65	8·7	
1846–1847			No Annual report	
1847–1848	5,153	693	13·4	'The predominance of maculated typhus, the most fatal form'
1848–1849	The establishment has ceased to be a fever hospital			

war a new disease research station was established there. Baker of the United States Army Veterinary Corps was a staff member. There were voles (*Microtus pennsylvanicus*) on the island, and, with some idea of using them as experimental animals, several were captured and dissected. In some of them the spleen was seen to be enlarged. From these spleens Baker isolated an infective agent like a Rickettsia. In 1977 Weiss and his associates showed that the 'vole agent' was a 'strain' of the trench fever organism *Rochalimaea quintana.*[24] Ninety-five years earlier Grosse Isle had been the site of a great tragedy.[25, 26] It was the quarantine station for ships entering Canada by the St Lawrence river. The year 1847 saw remarkable emigration from Ireland, much of it to Canada, and much to the St Lawrence ports. Ship after ship arrived laden with the starved, the dead, and those ill and dying from the 'fever'. Five thousand three hundred Irish emigrants died of fever and were buried in those 320 acres of Grosse Isle. Either the voles acquired the *Rochalimaea* from the Irish immigrants or the voles had always been infected. If the first explanation is true, as it likely is, it supports the existence of trench fever in the Irish. In 1982 Weiss and Dasch demonstrated some cultural, metabolic and agglutinative differences between the 'vole agent' of Baker and *Rochalimaea quintana.*[11] They proposed that the vole agent of Grosse Isle (and presumptively of Ireland) be named *Rochalimaea vinsonii sp. nov.*

Rutherfurd in 1916, at the time of the great epidemic of trench fever in the army in France, reported that the trenches were swarming with long-tailed field voles.[27] He thought these might be the source of the infection. Zdrodovskii and Golinevich draw attention to a reported disease in Russia, 'paroxysmal rickettsiosis', which in some clinical respects resembles trench fever.[28] The animal reservoir was thought to be the 'red forest vole', *Clethrionomys glareolus,* Schreber, and the vector a tick.

Smal and Fairley describe the discovery of the 'bank' vole *Clethrionomys glareolus* in south-west Ireland in 1964.[29] Although there has been spread, this vole is still confined to the south and west. Fairley considers that this vole is a recent introduction to Ireland. It seems important this bank vole should be examined to see if it is infected with a Rickettsia or a Rochalimaea.

REFERENCES

The Annual Reports of the Belfast Fever Hospital are held in the Archives Office of the Royal Victoria Hospital, Belfast. Microfilm copies are in the Queen's University Medical Library and in the Public Record Office of Northern Ireland.

1. Bergey DH. Manual of systematic bacteriology. Vol 1; editor, NR Krieg. Baltimore: Williams & Williams, 1984: 698-701.
2. Byam W et al. Trench fever. London: Oxford University Press, 1919.
3. American Red Cross. Trench fever: report of Commission, Medical Research Committee. By RP Strong. Oxford: Oxford University Press, 1918.
4. History of the Great War. Medical services. Diseases of the War; ed. by WG Macpherson, WP Herringham, TR Elliott, A Balfour. Vol 1. London: HMSO, 1923.
5. McNee JW, Renshaw A. A relapsing fever occurring with the British forces in France. *Br Med J* 1916; **1**: 225-34.
6. Vinson JW. Louse-borne diseases world wide: trench fever. In: Proceedings of the International Symposium on the Control of Lice and Louse-borne Diseases. Washington (DC), 1972. Washington (DC): Pan American Health Organization, 1973: 76-9. (Pan American Sanitary Bureau. Scientific Publication no. 263).

7. Vinson JW. *In vitro* cultivation of the rickettsial agent of trench fever. *Bull WHO* 1966; **35**: 155-64.
8. Hollingdale MR, Herrman JE, Vinson JW. Enzyme immunoassay of antibody to *Rochalimaea quintana. J Infect Dis* 1978; **137**: 578-82.
9. Reid JS. Annual report of the Belfast Fever Hospital for 1843–44.
10. Hurst A. Trench fever. *Br Med J* 1942; **2**: 318-20.
11. Weiss E, Dasch GA. Differential characteristics of strains of Rochalimaea: *Rochalimaea vinsonii sp nov,* the Canadian vole agent. *Int J Syst Bacteriol* 1982; **32**: 305-14.
12. McCormac H. An exposition of the nature, treatment and prevention of continued fever. London: Longman, 1835.
13. Woodhead GS, Varrier-Jones PC. The clinical thermometer. *Lancet* 1916; **1**: 173-80.
14. Little R. Practical observations on fever particularly with reference to the plan of treatment which has been generally pursued in the Belfast Fever Hospital for several years. *Dub J Med Chem Sci* 1835; **7**: 35-65.
15. MacArthur W. Some pestilences of the past, ii: The Yellow Plague. *Trans R Soc Trop Med Hyg* 1959; **53**: 430-2.
16. Louis PCA. Recherches anatomiques, pathologiques et thérapeutiques sur la maladie connue sous les noms de fièvre typhoïde, putride etc. 2 éd. Paris: Baillière, 1841.
17. Reid JS. Annual report of the Belfast Clinical and Pathological Society for 1859–1860: 76-81.
18. Osler W. The principles and practice of medicine. 3rd ed. Edinburgh: Pentland, 1898.
19. Megaw JWD. Louse-borne typhus fever. *Br Med J* 1942; **2**: 401-3.
20. Malcolm AG. A history of the General Hospital, Belfast. Belfast: W & G Agnew, 1851.
21. Doberstyne EB. Studies of louse-borne relapsing fever in Ethiopia. In: Proceedings of the International Symposium on the Control of Lice and Louse-borne Diseases, Washington (DC), 1972. Washington (DC): Pan American Health Organization, 1973: 42. (Pan American Sanitary Bureau, Scientific Publication no. 263).
22. McSkimin S. The history and antiquities of the County of the Town of Carrickfergus. 3rd ed. Belfast: Printed for the author, 1829.
23. Baker JA. A rickettsial infection in Canadian voles. *J Exp Med* 1946; **84**: 37-51.
24. Weiss E, Dasch GA, Woodman DR, Williams JC. Vole agent identified as a strain of the trench fever rickettsia *Rochalimaea quintana. Infect Immun* 1978; **10**: 1013-20.
25. Woodham-Smith C. The great hunger. London: Hamish Hamilton, 1962.
26. Heagerty JJ. Four centuries of medical history in Canada. Vol 1. Bristol: Wright, 1928: 117-30.
27. Rutherfurd WJ. Trench fever: the field vole a possible origin. *Br Med J* 1916; **2**: 386-7.
28. Zdrodovskii PF, Golinevich HM. The rickettsial diseases. London: Pergamon, 1960.
29. Smal CM, Fairley JS. The spread of the bank vole *Clethrionomys glareolus* in Ireland. *Mammal Rev* 1984; **14**: 71-8.

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Historical Note

Robert Dick, MD, FRCS, IMS: a biographical note by his grand-nephew

Sir John Megaw

Dr John Weaver and Sir Peter Froggatt in their recent 'Wild geese' vignettes[1] included an appreciation of my uncle, Sir John Megaw, KCIE, MB, DSc (*hc*), whose career culminated as Director-General of the Indian Medical Service and as Medical Adviser to the Secretary of State for India. A note on his medical uncle, Robert Dick, who preceded him into the IMS, seems apposite.

Robert Dick, born in 1831, was a brother of Sir John's mother, Ellen Dick, the Dicks being long established in Garry, Ballymoney, County Antrim, where Robert's father, also Robert, was an extensive farmer. After schooling at Ballymoney, the young Robert entered Queen's College Belfast in October 1849 (one of the 195 students — 55 in medicine[2] — who constituted the first batch of entrants to the newly opened College) where he had an outstanding career.[3] He was a Scholar in each of his four years, was Class Prizeman in Practical Chemistry in 1851 and leading Class Prizeman in Anatomy and Physiology in 1852,[4–7] and graduated MD (the primary degree of the Queen's University in Ireland) in September 1853 with a prize medal.[8]

Dick took the MRCS (Engl) in 1854[9] and was appointed from 24 January 1855 Assistant Surgeon to the 'Queen's and [East India] Company Forces in the East Indies, etc., etc.'[10] He saw service in the British-Persian war of 1856–7 and the Indian Mutiny of 1857, and became a member of the IMS when it was formed in 1858, and for a while was stationed at Poona.[11] On 13 June 1867 he took the FRCS (Engl) by examination[12] and on 3 July was promoted 'Surgeon to HM Forces' retroactive to 24 January.[13] He retired in or about 1872 with the rank of Surgeon-Major in 'the Bombay Army'.[14] In 1874 he returned to Garry where he lived for some 40 years. He took a keen interest in the educational development of the children of his sister, Ellen Megaw, in Ballyboyland. He died in 1913 and was buried on 16 October in the Old Church Burying Ground in Ballymoney. He never married.

Dick was a shy and reserved man and these qualities, his campaign experience, particularly in the Indian Mutiny, and his deteriorating health probably determined his early retirement from the IMS after less than 20 years' service. His close attention to the education of his nephew, John, no doubt influenced John's decision to make medicine his career and to follow that career in the IMS of which he was to become in 1930 Director-General.

Sir John Megaw, PC, CBE, TD, Lord Justice of Appeal (retired).

NOTES

1. Weaver JA, Froggatt P. The 'wild geese'. *Ulster Med J* 1987; **56 (suppl)**: S31-S56.
2. Moody TW, Beckett JC. Queen's, Belfast 1845–1949. Vol ii. London: Faber & Faber, 1959: App II.
3. Among fellow entrants was James Cuming, later to be Professor of Medicine (1865–1899) and the first Catholic medical professor of QCB. Froggatt P. Medicine in Ulster: the Belfast School. In: O'Brien E, Crookshank A, Wolstenholme G, eds. A portrait of Irish medicine: an illustrated history of medicine in Ireland. Dublin: Ward River Press, 1984: 183-213.

   I am indebted to Sir Peter Froggatt for his research which has brought to light particulars of Dick's academic and service career.
4. The Belfast Queen's College Calendar 1850. Belfast: Henry Greer, 1850: 40.
5. Ibid, 1851. Belfast: Simms and McIntyre, 1851: 67, 72.
6. Ibid, 1852. Belfast: Alexander Mayne, 1852: 78, 79, 82.
7. Ibid, 1853: lxi, lxiv.
8. QUB Library, Miscellaneous Papers MS1/88, MD diploma. The medal is the property of his grand-niece, Dr Helen Dick Megaw, who has contributed much of the material in this memoir. MS1/88 contains five documents on Robert Dick which were lodged in 1938 by my father, Robert Dick Megaw.
9. The Medical Directory. London: J & A Churchill.
10. QUB Library, MS1/88, certificate of appointment. The certificate is in the name of Sir William Maynard Gomm, KBE, C-in-C Queen's and Company Forces in the East Indies, and is dated in Bengal 24 December 1855, i.e. 11 months after the operative date of appointment.
11. Obituary notice. *Lancet* 1913; **2**: 1219. This was supplied by the '*Lancet's* own correspondent in Ireland'.
12. QUB LIbrary, MS1/88, Fellowship certificate.
13. Ibid, certificate of appointment signed by George, Duke of Cambridge, Field-Marshal, and by Edward Cardwell, later (1868) Secretary for War.
14. The Medical Directory for 1876. London: J & A Churchill, 1876: under name in the Indian Medical Service and Mercantile Marine section.

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Case report

Extreme lateral lumbar disc herniation

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Lumbar disc herniations that project far laterally towards or lateral to the intervertebral foramen show minimal or no abnormalities on myelography and compress the nerve root one level above that of the classical posterolateral herniation. Computed tomography is the imaging method of choice, either as the first investigation or following a negative or equivocal myelogram.

CASE HISTORY

A 60-year-old man presented with a three-week history of low back pain and right sciatica after lifting a heavy weight. On examination, there was marked restriction of straight leg raising on the right side with a reduced knee jerk and impaired sensation in the right L3 and L4 dermatomes. The provisional diagnosis was L4 root compression by a herniated L3/4 disc. Myelography with water-soluble contrast medium showed no abnormality, and the following day a computed tomography scan was carried out at the L3/4, L4/5 and L5/S1 levels. This confirmed that the L3/4 and L5/S1 discs were normal. At L4/5 there was a large disc herniation on the right side lateral to the intervertebral foramen (Fig 1).

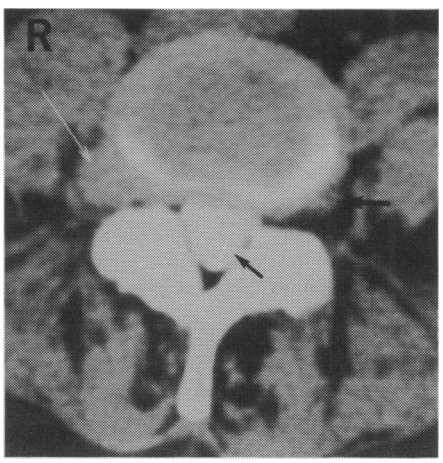

Fig 1. CT scan at level of L4/5 showing a large right-sided extreme lateral lumbar disc herniation (white arrow). Small black arrow = thecal sac containing residual contrast medium from myelogram. Large black arrow = left L4 root in paraspinal fat.

The herniated disc was surgically exposed via a modified lateral approach with removal of the superior and inferior facets at L4/5. Two large pieces of degenerate disc were removed with decompression of the L4 root laterally. His back pain and sciatica disappeared within a few days of operation, and at review six months later he remained symptom-free.

COMMENT

An extreme lateral lumbar disc herniation may be defined as prolapse of disc material such that it lies beyond the intervertebral foramen with or without extension into the foramen.[1] The reported incidence has ranged from

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0·7–11·7%.[2, 3, 4] In a recent series of 1,600 operated lumbar disc herniations, the overall incidence of this type of lesion was 6%, representing 6% of herniations at L5/S1, 4% at L4/5 and 18% at L3/4.[4] Characteristically, it occurs in the sixth decade. Reproduction of pain with lateral bending has been reported as a reliable sign of this condition.[2, 5] However, other authors have not found any characteristic clinical features which distinguish it from a classical posterolateral herniation.[4]

The classical posterolateral lumbar disc herniation compresses the lumbar nerve root within the spinal canal as it passes obliquely inferiorly towards the intervertebral foramen at the level below. Thus, a herniation at L3/4 will compress the L4 root (Fig 2a). Within the paraspinal tissues, the root passes over the lateral margin of the disc below (4/5); thus the L4 root will also be compressed laterally by an extreme lateral lumbar disc herniation at this level (Fig 2b). Therefore, this compression occurs at one disc space below that of a classical posterolateral herniation causing a similar clinical syndrome.

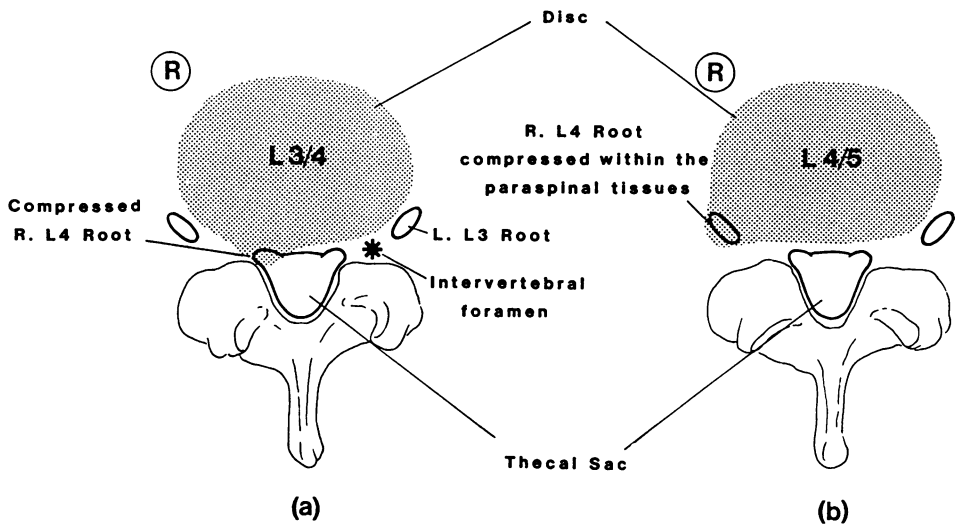

Fig 2. Diagrammatic representation of (a) a classical right posterolateral disc protrusion at L3/4 compressing right L4 root and (b) an extreme lateral lumbar disc herniation at L4/5 compressing right L4 root in the paraspinal tissues.

The radiological diagnosis of lumbar disc herniation depends on visualising the herniated material or deformity caused by it. Although myelography with water-soluble contrast medium is very reliable in detecting classical herniations,[6] only subtle changes are seen in extreme lateral lumbar disc herniations. These include incomplete filling of a nerve root distally with proximal swelling and loss of the delicate outline of the root: a significant proportion of cases show no abnormality.[4] Discography (with or without computed tomography) is invasive and time-consuming but may confirm the symptomatic level. Magnetic resonance may prove to be of value.

Computed tomography gives a direct demonstration of the disc and is non-invasive. It is highly accurate in the detection of extreme lateral lesions.[4, 5] It is the examination of choice when this diagnosis is considered,[6] and indeed probably for all suspected disc herniations. It is often difficult to distinguish clinically L5

and S1 root pain and L2, L3 and L4 root pain. In addition, the possibility of extreme lateral herniations may not be suspected and CT scanning in a patient with root compression therefore should include at least two disc levels and intervertebral foramina. In practice, it is usual to scan the L3/4, L4/5 and L5/S1 levels.

The diagnosis of extreme lateral lumbar disc herniations has important implications for the surgeon since a conventional approach to the disc may result in failure to locate the herniation. Techniques used include total facetectomy through a midline incision and a lateral approach via a paramedian incision with retraction or splitting of the paraspinal muscles.[4, 5]

I would like to thank Mr I C Bailey, FRCS, for permission to report this case, Dr C S McKinstry, FRCR, for his advice and Miss Adrienne Murphy for typing the manuscript.

REFERENCES

1. Novetsky GJ, Berlin L, Epstein AJ, Lobo N, Miller SH. The extraforaminal herniated disc: detection by computed tomography. *Am J Neuroradiol* 1982; **3**: 653.
2. Abdullah AF, Ditto EW, Byrd EB, Williams R. Extreme lateral lumbar disc herniations. *J Neurosurg* 1974; **41**: 229.
3. Kurobane Y, Takahashi T, Tajima T, et al. Extraforaminal disc herniations. *Spine* 1986; **11**: 260.
4. Fankhauser H, De Tribolet N. Extreme lateral lumbar disc herniation. *Br J Neurosurg* 1987; **1**: 111.
5. Abdullah AF, Wolber PGH, Warfield JR, Gunadi IK. Surgical management of extreme lateral lumbar disc herniations: review of 138 cases. *Neurosurgery* 1988; **22**: 648.
6. Schipper J, Kardaun JWPF, Braakman R, Van Dongen KJ, Blaauw G. Lumbar disc herniation: diagnosis with CT or myelography? *Radiology* 1987; **165**: 227.

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Case report

The Rapunzel syndrome: a rare presentation of trichobezoar

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Accepted 20 February 1989.

A trichobezoar is an infrequent occurrence and like most other gastrointestinal foreign bodies tends to occur in the stomach and duodenum. The Rapunzel syndrome is a rare form of bezoar that extends into the small, and sometimes the large, bowel. We describe the fourth reported case of this syndrome, with other unusual features.

CASE HISTORY

A five-year-old girl was admitted in 1985 with an eight-day history of epigastric pain and vomiting. The pain was occasional and not severe. She had lost weight, to 13·1 kg, and was pale and thin. There was a tender epigastric mass. The haemoglobin concentration was 12·8 g/dl. Her mother said the child regularly pulled and ate the sleeves of her cardigans. An ultrasound scan showed a foreign body in the stomach. At laparotomy through an upper midline incision there was a large ball of wool fibre in the stomach and duodenum. There was also an inter-loop abscess near the jejunum due to a localised perforation. A short segment of small bowel was excised and continuity restored by end-to-end anastomosis. Mesenteric glands were noted to be enlarged. The post-operative course was uneventful.

The child presented again in 1988 with a four-week history of poor appetite, lethargy and intermittent epigastric pain. Again she had lost weight, but there was no vomiting or constipation. Her mother had died four months prior to this admission. Clinically the child was thin, pale and apathetic. There was some tenderness in the epigastrium but no definite mass. The haemoglobin was 11·8 g/dl and the ESR 22 mm/hr. The barium meal (Fig 1) showed a large intragastric mass. At laparotomy a large foul-smelling mass which nearly filled the stomach was found, extending with a long string and short clumps of hair to the level of the ileocaecal valve (Fig 2). The small bowel was remarkably normal. The post-operative course was uneventful and she was eventually referred to a child psychologist.

DISCUSSION

Trichobezoars occur infrequently. They usually occur in females with long hair, and are uncommon under the age of ten. In the classic reviews by De Bakey and

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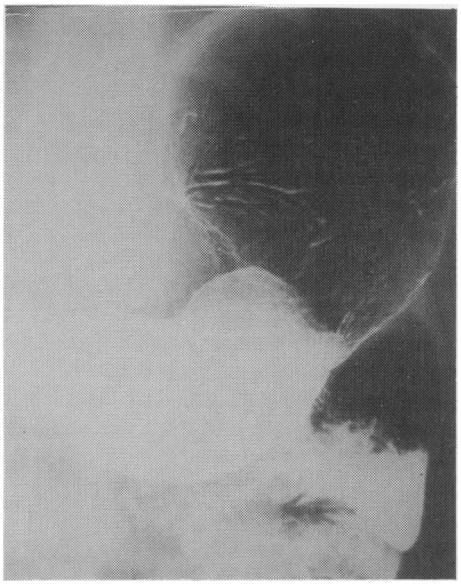

Fig 1 (above). Barium meal showing a large mass occupying the distal half of the stomach.

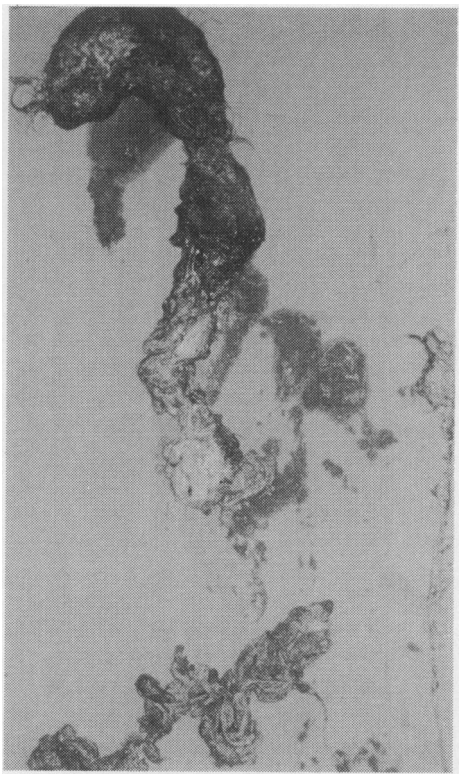

Fig 2 (right). The specimen after operative removal from the stomach and small intestine.

Ochsner,[1,2] only 31 out of 232 cases (13%) occurred in patients below the age of nine, 90% of whom were female. Many of the patients expressed personality maladjustment.

Bezoars occur in four forms: Trichobezoars (mainly hair), 50% of the total; phytobezoars (ingested plant fibre, skins, seeds, leaves or roots), 40% of the total; medication bezoars (antacids and resins), and concretions (imbibition by painters and furniture workers of furniture polish). Many patients remain asymptomatic; in some, only vague abdominal pain is experienced. More serious complications may occur including ulceration (duodenal, gastric), perforation, abscess formation or intestinal obstruction.

The symptomatology in 225 cases (131 trichobezoars and 94 phytobezoars)[1,2] was abdominal in 169, pain and tenderness in 172, nausea and vomiting in 155, weakness and weight loss in 81, constipation or diarrhoea in 72, haematemesis in 22, and a history of ingestion was obtained in 128. Intestinal obstruction occurred in 10 cases and the mortality was 19·1% for trichobezoars and 12·5 for phytobezoars. One patient in their series underwent six laparotomies. Intussusception has also been described secondary to bezoars.[3]

The term Rapunzel syndrome was first proposed by Vaughan et al.[4] They reported two patients with trichobezoars extending from the upper small bowel to the distal ileum and transverse colon respectively. They borrowed the name from a fairy tale: the charming maiden Rapunzel, held in captivity in a high tower, used to lower her long tresses to the ground for her prince to climb up to her window.

As far as we can ascertain, only one further case was reported.[5] This was a 14-year-old girl with a definite history of trichophagia (eating hair) and trichotillomania (pulling out her hair) in whom attempted medical treatment for the bezoar with papain and sodium bicarbonate unfortunately proved fatal. Our patient illustrates many of the features found in patients with trichobezoars. She has long fair hair, there was a history of trichophagia and there was a background of stress on the second occasion (mother's death). She was malnourished and a definite mass was palpable on the first admission.

Though some authors advocate multiple enterotomies to remove long trichobezoars, in our case only gastrostomy was necessary. The long string extending close to the ileocaecal valve was delivered intact with gentle, sustained traction. We do not feel that endoscopic removal would be possible except for very small trichobezoars. The whole bowel (large and small) should be palpated carefully at operation. Surgery should be preceded by correction of dehydration, anaemia and malnutrition where appropriate. In many patients, psychiatric help is needed.

REFERENCES

1. De Bakey M, Ochsner A. Bezoars and concretions. *Surgery* 1938; **4**: 934-63.
2. De Bakey M, Ochsner A. Bezoars and concretions. *Surgery* 1939; **5**: 132-60.
3. Both TW, Harries JT, Glaser R, Graham PJ. Trichobezoars and laparotomies. *J R Soc Med* 1981; **74**: 691-2.
4. Vaughan ED, Sawyers JL, Scott HW. The Rapunzel syndrome: an unusual complication of intestinal bezoar. *Surgery* 1968; **65**: 339-43.
5. Deslypere JP, Praet M, Verdonk G. An unusual case of trichobezoar: the Rapunzel syndrome. *Am J Gastroenterol* 1982; **77**: 467-70.
6. De Bakey M, Jordan GL Jr. Bezoars. In: Maingot R. Abdominal operations. 8th ed; eds SI Schwartz, HEllis. Norwalk (Conn): Appleton-Century-Crofts, 1985; **1**: 675-81.

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Case report

Fibromatosis of the breast: a benign lesion which simulates a carcinoma

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The fibromatoses are a group of benign proliferations of fibrous tissue which have an infiltrative growth pattern and a tendency to recur, but do not metastasise. Fibromatosis of the breast is rare, and less than 50 cases are described in the world literature.[1, 2, 3, 4]

CASE HISTORY

A 56-year-old woman presented to surgical out-patients having found a lump in her right breast seven weeks previously. She had 12 children, all breast-fed, was on no hormonal therapy, and reported no history of trauma. There was no family history of breast carcinoma or other breast disease. A 3×4 cm mass was palpable in the right lower inner quadrant, attached deeply and with some skin tethering. No lymph nodes were palpable. The clinical impression was that of a carcinoma. Mammography showed some bilateral breast thickening, but did not suggest carcinoma. Wide local surgical excision of the lesion with removal of a portion of chest wall fascia was carried out. The resected specimen was sent for frozen section examination.

The main portion of tissue weighed 106 g and contained a 2·5 cm diameter tumour, lying 1·5 cm deep to the skin. It was greyish-white and very firm, with a stellate infiltrative margin. The gross appearance was suggestive of carcinoma. Histology of the frozen section showed interwoven bundles of uniform spindle cells, with no pleomorphism and no epithelial component. A tentative diagnosis of fibromatosis was made. The differential diagnosis included spindle-cell sarcoma or part of a cystosarcoma phyllodes, although no epithelial clefts were present. The diagnosis of fibromatosis was confirmed on examination of paraffin sections. Bundles of uniform spindle cells were interlaced with collagen, and the edge was clearly infiltrative. Only occasional breast ducts were indentified. There was no cellular atypia and the mitotic count was less than 3 per 10 high power fields (Figs 1 and 2). The separate portion of chest wall fascia and muscle submitted was uninvolved. Immunoperoxidase staining indicated a tumour of mesenchymal origin, as vimentin was positive, while epithelial and muscle markers were negative. Electron microscopy showed the majority of the cells to be fibroblasts, with frequent myofibroblasts, in keeping with the histological diagnosis of fibromatosis.

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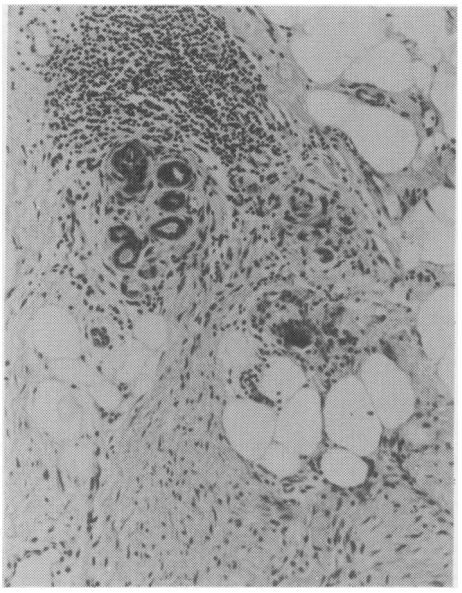

Fig 1. Infiltrative edge of lesion with involvement of breast lobule (× 300).

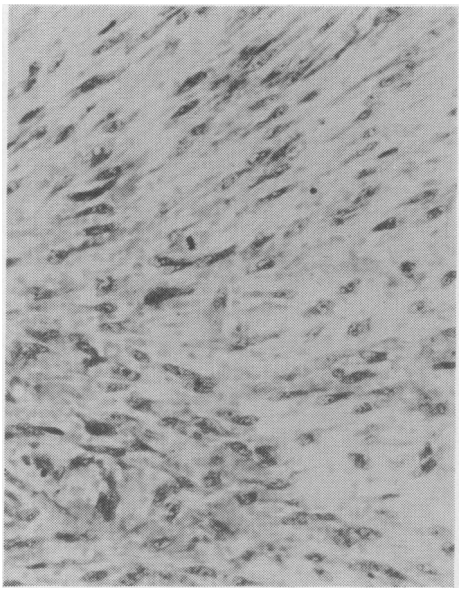

Fig 2. Representative high power field showing uniform fibroblasts. A normal mitotic figure is present (× 675).

DISCUSSION

Fibromatosis of the breast is a rare condition with less than 50 cases described in the world literature.[1, 2, 3, 4] Possible aetiological factors are trauma (30% of patients), hormonal influence (increased incidence of fibromatoses in pregnancy), and genetic predisposition (in association with Gardner's syndrome).[4] The clinical appearance of a hard fixed lump is often suggestive of carcinoma, and there seem to be no specific features which make pre-biopsy diagnosis possible. Mammography may also be misleading,[5] although in this case the mammogram showed no features of carcinoma. Several cases have been reported in which the clinical and radiographic findings have led to unnecessary mastectomy because of mistaken diagnosis,[1, 2] and in one reported case a cytological aspirate was also falsely positive for carcinoma.[2] However, the possibility of unnecessary mastectomy seems unlikely nowadays, as the trend in breast surgery has moved towards local resection.

The typical histological appearance is of interwoven bundles of uniform fibroblasts and collagen, with a characteristically infiltrative edge. The presence of breast ducts/lobules may be taken as an indication that the lesion has arisen in breast parenchyma rather than chest wall fascia.[3] In this case occasional breast ducts were present, and the separately submitted portion of muscle and chest wall fascia deep to the lesion was uninvolved.

Treatment advocated by most authors is wide local excision[1, 2] although some recommend mastectomy because of the high risk of recurrence.[4] The risk of recurrence appears to be related solely to the presence of fibromatosis at the surgical margins of excision, and it has been shown that other histological features such as cellularity, atypia and mitotic count do not predict the likelihood of recurrence.[3] The majority of recurrences manifest within 3 years.[4]

We would like to thank Mr R Curry, FRCS, for permission to publish this case.

REFERENCES

1. Rosen Y, Papasozomenos SC, Gardner B. Fibromatosis of the breast. *Cancer* 1978; **41**: 1409-13.
2. Bogomoletz WV, Boulenger E, Simatos A. Infiltrating fibromatosis of the breast. *J Clin Pathol* 1981; **34**: 30-4.
3. Wargotz ES, Norris HJ, Austin RM, Enzinger FM. Fibromatosis of the breast: a clinical and pathological study of 28 cases. *Am J Surg Pathol* 1987; **11**: 38-45.
4. Thomas T, Lorino C, Ferrara JJ. Fibromatosis of the breast: a case report and literature review. *J Surg Oncol* 1987; **35**: 70-4.
5. Cederlund CG, Gystavsson S, Linell F, Moquist-Olsson I, Andersson I. Fibromatosis of the breast mimicking carcinoma at mammography. *Br J Radiol* 1984; **57**: 98-101.

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Case report

Compulsive jogging: exercise dependence and associated disorder of eating

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A number of patients have been reported who have developed features of an eating disorder following a desire to increase their level of fitness.

CASE HISTORY

A 42-year-old male school teacher was referred to the psychiatric out-patient clinic by his general practitioner at the request of his brother who had become concerned by the patient's obsession with fitness and also his marked loss of weight. His weight had dropped from 218 lb to 112 lb in the space of two years. He had always been keen on sport, having played a number of games at a national level. He had also been an enthusiastic golfer from the age of six, at one stage reducing his handicap to two. In 1985 he decided suddenly to improve his physical health, having become markedly overweight at 218 lb and also because of a strong family history of ischaemic heart disease. The patient put himself on a strict diet and although he continued to eat mixed foodstuffs he limited his energy intake to 800–1,000 calories per day. He also took up regular running and ran at least five miles a day followed by one hour in a sauna. Running became his priority in life and as soon as his teaching duties finished he set out jogging, followed by a sauna, and later in the evening he would referee basketball. His weight fell to 112 lb which is considerably below the minimum desirable weight for his height of 5 ft 10 ins (144 lb), or the average UK weight for height and age (176 lb).[1] He was pleased with this loss of weight and did not consider himself to be abnormally thin.

As a result, his work as a teacher and, in particular, his marriage suffered. During the weekends he would referee hockey matches, but he would also indulge in binges of eating and taking excess alcohol. At these times he would put on up to 7 lb in weight, which led to marked feelings of guilt. The increase in weight would be quickly controlled by renewed strenuous dieting, exercise and spending time in the sauna. He complained of feeling depressed at times and became bored with his work and dissatisfied with his home life. He had loss of libido. When running he felt elated. His sleep pattern was disturbed, with both initial insomnia and also some early morning wakening. He became obsessed with his performance as a runner.

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He had earlier refused to turn up when a psychiatric out-patient appointment was made, and, when he eventually attended, accompanied by his brother, he showed some resentment about being brought to see a psychiatrist. He felt his actions were reasonable but admitted that his behaviour was putting strain on his marriage, partly due to the fact that he spent so little time at home with his wife and three children. At this attendance he looked thin, but was neat and tidy in his appearance. He appeared depressed with a constant worried and drawn expression on his face. He responded slowly to questions and demonstrated a flat affect with little variation in his speech.

He was seen six times as an out-patient over a three-month period and a number of joint interviews were carried out with the patient and his wife. Several psychogenic factors emerged and were discussed, such as his poliomyelitis as a child, his marital relationship (including long-standing sexual problems), his loss of ability to play certain sports competitively and his disappointment that his son was not successful at sport.

Although he continued to run he was able to do this in a more controlled fashion. His marital relationship improved and he spent more time with his wife and children and his weight increased to about 140 lb. Some clinical features initially were suggestive of a depressive illness, but in subsequent interviews any depression was considered to be reactive to the whole situation and drug therapy was not used. It was considered important not to stop running as this might lead to an increase in his depression.

DISCUSSION

Morgan described eight cases of 'running addiction' where commitment to running assumed a higher priority than commitment to work, family, interpersonal relations or medical advice.[2] A number of authors have suggested a similarity between exercise dependence, anorexia nervosa and bulimia nervosa. Katz reported two cases very similar to this patient, who developed features of anorexia nervosa associated with bingeing and vomiting following marked increase in exercise and weight loss.[3] Yates et al described three cases who were considered to be 'obligatory runners', and who had many features in common with anorexia nervosa, such as family background, socioeconomic class, personality factors with inhibition of anger, extraordinary high self-expectation, tolerance of physical discomfort, denial of potentially serious debility and a tendency towards depression.[4] Chalmers et al discussed a 30-year-old woman who developed anorexia nervosa which had presented initially as morbid exercising.[5] In their study of obligatory running and anorexia nervosa Blumenthal et al attempted to assess the similarity between 'obligatory running' and anorexia nervosa and concluded that the runners do not suffer from the same degree of psychopathology as do patients with anorexia nervosa.[6] Veale stated that exercise can become compulsive behaviour and harmful to an individual and lists a number of features present in patients suffering from 'exercise dependence' and commented on the similarity between these and other forms of dependency.[7]

In the present case, it seemed entirely appropriate for an overweight man of 42 to reduce his weight and increase his fitness, especially with the family history of ischaemic heart disease. It became abnormal behaviour because of excessive loss of weight, the amount of time he spent running and his preoccupation with energy intake, bingeing at the weekends with associated guilt feelings, the detrimental effect on his work and marital and family life, his feeling of depression when not running and his lack of insight into these problems.

In the past 20 years or so, jogging has become a fascination for a large number of people, and over the same period anorexia nervosa, previously thought to be a rare disorder, has increased to such an extent that it is now regarded as a public health problem. Is excessive running in the male an analogue of anorexia nervosa in the female? It has been suggested that exercise can play a role in contributing to an eating disorder via several routes, perhaps the most obvious of which is that exercise automatically causes increased energy expenditure and therefore weight loss, unless there is increased food intake.[2] Mood is also enhanced consequent to exercise which may be related to an elevation in endorphin production or opioid levels in the central nervous system. Exercise dependence is most likely to present at a sports clinic or casualty department as persistent physical injury, and it is important that this condition should be diagnosed to prevent continued exercise despite injury or illness.[7] Although there are many obvious benefits in a general increase in health awareness and fitness in the population, some few people develop problems with excessive exercise.

REFERENCES

1. Knight I, Elridge J. The heights and weights of adults in Great Britain. London: HMSO, 1984; 25-36.
2. Morgan WP. Negative addiction in runners. *Phys Sports Med* 1979; **7**: 57-70.
3. Katz JL. Long distance running, anorexia and bulimia. A report of two cases. *Compr Psychiatry* 1986; **27**: 74-8.
4. Yates A, Leehey K, Shisslak CM. Running, an analogue of anorexia? *N Engl J Med* 1982; **308**: 251-5.
5. Chalmers J, Catalan J, Day A, Fairburn C. Anorexia nervosa presenting as morbid exercising. *Lancet* 1985; **1**: 286-7.
6. Blumenthal JA, O'Toole LC, Chang JL. Is running an analogue of anorexia nervosa? *JAMA* 1984; **252**: 520-3.
7. Veale de Coverley DMW. Exercise dependence. *Br J Addict* 1987; **82**: 735-40.

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Case report

Community help for women with breast cancer after discharge from hospital

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Breast cancer is the most common malignancy in women. In Northern Ireland there are 600 new cases diagnosed each year. Approximately 80 of these patients are seen at the Ulster Hospital. It is now a well established fact that there is considerable psychiatric morbidity among women with breast carcinoma. Maguire found that 39% of women who had a mastectomy had mood disturbance and sexual problems serious enough to warrant psychiatric help.[1] Faulkner stated that women who have had a mastectomy are one group of patients who are likely to be neglected by the primary health care team.[2] The object of this study was to determine the extent of patients' problems and to assess the help available to them following discharge from hospital.

METHODS AND RESULTS

Thirty patients were sent a postal questionnaire and 28 returned it. The questionnaire included both closed and open questions. Closed questions sought factual information, such as age, change in sleep pattern, help given and by whom. Open questions such as 'What worried you most when you went home?' and 'Have you any suggestions as to how your care could be improved?' were included to obtain a personal individual perspective from each patient.

All patients in a one-year period who satisfied the following criteria were entered into the study: –

1. Females aged 30 to 60 years inclusive.
2. Awareness of their diagnosis before admission to hospital.
3. Performance of either simple or partial mastectomy.
4. Performance of surgery at least three months before questioning.

Twenty-three (82·1%) of patients felt they had had sufficient help in coming to terms with their illness while in hospital. This must be taken in the context that a breast advisory nurse was available to counsel patients prior to, during and after admission to hospital.

After discharge, 12 patients discussed their illness with their general practitioner. Of these, six felt he helped 'a lot' and six 'a little'. Three patients discussed their illness with a district nurse, one with a health visitor and two with a social worker. Those patients who were visited by a district nurse had required dressings and those seen by a health visitor or social worker were already being visited prior to

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the diagnosis of breast cancer. Thus, neither nurses nor health visitors attended in the role of counsellor. All patients were given some support following discharge, 85% from family and 75% from friends.

For 25 of the 28 patients the most traumatic time was either before admission to hospital or after discharge. Only three patients (10·7%) experienced their most difficult time while in hospital. In the latter half of the survey the study sought to establish the extent of problems experienced after discharge. Some 71% admitted to one or more symptoms of anxiety or depression, 53% had a change in sleep pattern, 25% had a change in sexual behaviour — either less interest or no interest at all — and 21% had more than occasional anxiety. In response to the question 'What worried you most when you went home?', the tone was one of fear and uncertainty regarding treatment, body image and prognosis.

The final question asked for suggestions as to how care could be improved. One patient stated 'I feel more medical support should be given to patients with breast cancer when they go home'. Another said 'A visit from a district nurse or health visitor would prove beneficial especially if the husband and family could be present'.

DISCUSSION

Although patients had help from family and friends, it would appear there is a need for more professional help after discharge. The results obtained in this study confirm this and are consistent with those of Anderson in her survey among patients in Scotland, who found that district nurses called mainly for suture removal or reasons other than to counsel the patient about her breast cancer.[3]

It is a worrying discovery that so many women experience such problems after breast surgery. The majority of patients are now treated by wide local excision of the tumour. With this conservative surgery, both the medical and nursing professions tend to assume that psychiatric morbidity has been reduced. Fallowfield found that there was still considerable psychiatric morbidity among patients treated conservatively and that these women need just as much support as patients who have a mastectomy.[4] Patients continue to have problems in coming to terms with their diagnosis and this study confirms that these problems are not being resolved nor are the patients given sufficient support.

I wish to thank Mr Hume Logan, FRCS, for his help and encouragement during this study.

REFERENCES

1. Maguire GP, et al. Psychiatric problems in the first year after mastectomy. *Br Med J* 1978; **1**: 963-5.
2. Faulkner A. Reclaiming a body image. *Community Outlook* 1985. (May).
3. Anderson J. Coming to terms with mastectomy. *Nurs Times* 1988; **84**: 4.
4. Fallowfield LJ, Baum M, Maguire GP. Effects of breast conservation on psychological morbidity associated with diagnosis and treatment of early breast cancer. *Br Med J* 1986; **293**: 1331-4.

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Case report

Acute gastric volvulus and strangulation of colon mimicking chest infection

W Dickey, G W McCullough, K R Logan

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Various gastrointestinal emergencies may present with signs and radiological abnormalities suggestive of pulmonary disease. Awareness is essential if early diagnosis is to be made, and appropriate, possibly life-saving, treatment given.

CASE HISTORY

A 63-year-old farmer presented with left-sided pleuritic pain, cough producing purulent sputum, and vomiting. Examination of the chest revealed diminished respiratory movement, dullness to percussion, coarse inspiratory crepitations and bronchial breath sounds at the left base. He was apyrexial. There was mild epigastric tenderness but no abdominal rigidity, guarding or masses, and bowel sounds were sparse but normal in character. Serum electrolytes and liver enzymes and haemoglobin concentration were normal. He had a neutrophil leucocytosis ($11{\cdot}5 \times 10^3/\mu l$) and ESR was 49 mm/hour. Chest X-ray (Fig) appeared to confirm the clinical impression of consolidation of the left lower lobe, and sputum grew coliforms. He was commenced on amoxycillin.

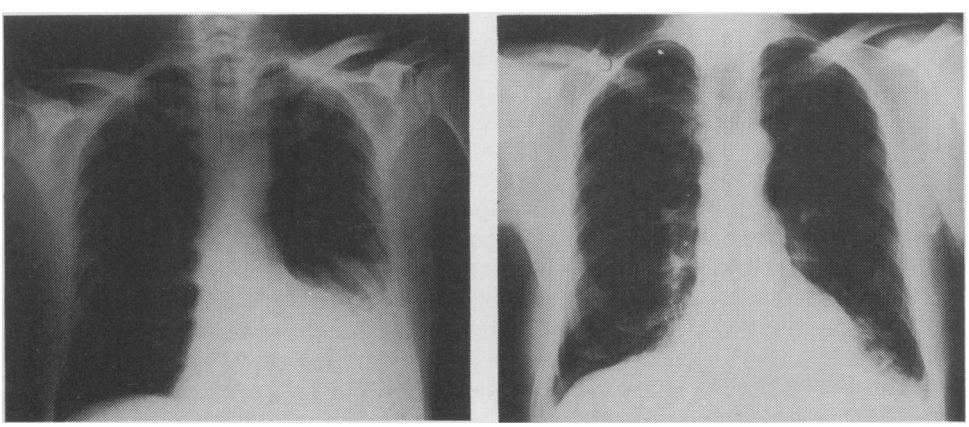

Figure. Chest X-rays taken on admission (left) and 10 days post-operatively (right).

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Thirteen years before, during investigation of iron deficiency anaemia, barium studies had shown a large hiatus hernia. He had a long history of chronic obstructive airways disease, and chest films from 1963 onwards had shown mild bronchiectatic changes in the left lower zone.

He gave no history of trauma to chest or abdomen. Following admission he had no further vomiting but had bouts of retching and despite complaining of no other symptoms was restless and agitated. Physical findings remained unchanged and plain abdominal films showed no evidence of obstruction.

Seventy-two hours after the onset of symptoms he developed severe abdominal pain, and examination revealed rigidity and absent bowel sounds. He proceeded to immediate laparotomy and was found to have a large paraoesophageal hiatus hernia containing stomach and an incarcerated, gangrenous loop of transverse colon. A gastric volvulus was present but there was no evidence of strangulation. Thirty centimetres of transverse colon were resected and an end-to-end anastomosis performed. The volvulus was corrected, stomach restored to the abdomen, and the hernia repaired. Examination of the resected bowel showed full thickness haemorrhagic infarction with ischaemia of the related omentum.

His subsequent course was uneventful and repeat chest films post-operatively showed dramatic resolution of the left lower zone shadowing. Nine months after operation he remained well.

DISCUSSION

Acute gastric volvulus and strangulation of colon are uncommon though well recognised complications of hiatus hernia, and simultaneous occurrence must be exceptional. Both are more common in patients with large paraoesophageal hernias. In a series of 22 patients requiring surgery, 10 had a portion of transverse colon in the chest, though none had strangulated, and six needed emergency operations for obstruction due to acute gastric volvulus.[1] As the stomach enters the chest through a large hernia, the more mobile body and greater curvature move relatively higher anteriorly, causing organoaxial rotation and drawing the greater omentum and transverse colon along. Torsion occurs at the relatively immobile pylorus and cardia. Borchadt's triad, comprising severe epigastric pain with distension, vomiting which gives way to violent retching as gastric obstruction ensues, and difficulty in passing a nasogastric tube, is said to be characteristic of acute volvulus.[2] However, as most cases are intrathoracic, it must be emphasised that abdominal findings may be minimal. Gangrene of the stomach complicates 5% of cases, but the great majority of these are related to diaphragmatic hernias of traumatic origin,[2] which can be notoriously difficult to diagnose and may present up to 60 years after the initial injury.[3]

The most notable aspect of this case was the initial predominance of respiratory findings. Large hernias may produce local collapse of the lung and pleural reaction, and further confuse the diagnosis. They have been mistaken for pneumonia, empyema and pleural effusion.[4, 5, 6] Should a complicated hernia be suspected, from unusual symptoms or previous history, barium studies will demonstrate viscera in the chest.

REFERENCES

1. Wichterman K, Geha AS, Cahow CE, Baue AE. Giant paraesophageal hiatus hernia with intrathoracic stomach and colon: the case for early repair. *Surgery* 1979; **86**: 497-506.
2. Carter R, Brewer LA, Hinshaw DB. Acute gastric volvulus. *Am J Surg* 1980; **140**: 99-106.
3. Ball T, McCrory R, Smith JO, Clements JL. Traumatic diaphragmatic hernia: errors in diagnosis. *Am J Roentgenol* 1982; **138**: 633-7.
4. Welch FT, Reynolds SA. Diaphragmatic hernia simulating staphylococcal pneumonia. *Rocky Mountain Med J* 1972; **69**: 37-40.
5. Goldstein AI, Gazzaniga AB, Ackerman ES, Rajcher WJ, Kent DR, Campbell R. Strangulated diaphragmatic hernia in pregnancy presenting as an empyema. *J Reprod Med* 1972; **9**: 135-9.
6. Thind GS. Diaphragmatic hernia simulating pleural effusion and complicating puerperium. *Australas Radiol* 1977; **21**: 325-7.

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Case report

Tetany in association with gentamicin therapy

O L Beatty, N P S Campbell, R D G Neely

Accepted 1 March 1989.

Magnesium plays a role in calcium metabolism and hypomagnesaemia can lead to hypocalcaemia.[1] Recently it has been recognised that prolonged amino-glycoside therapy can cause hypomagnesaemia.[2, 3] We have recently observed tetany associated with low serum levels of both calcium and magnesium in a patient receiving gentamicin, which resolved when the gentamicin was stopped and the serum magnesium was corrected.

CASE HISTORY

A 21-year-old male with a congenital ventricular septal defect was admitted with a ten-day history of rigors, malaise and anorexia. Three out of six blood cultures grew *Staphylococcus aureus.* A diagnosis of infective endocarditis was made and treatment with intravenous flucloxacillin and gentamicin was begun. Frequent peak and trough gentamicin levels were maintained within the therapeutic range. Eighteen days after treatment commenced, the pyrexia recurred and flucloxacillin was replaced by vancomycin. Three days later, he developed an allergic drug rash, and the vancomycin was changed to fusidic acid. Antibiotics were administered for a total of 56 days (Table).

Fifty-one days after treatment commenced, he complained of tingling around the lips and developed carpopedal spasm. The tetany responded to 30ml 10% calcium gluconate (6·69mmol). The uncorrected serum calcium was 1·36mmol/l (2·10–2·50) and the serum magnesium was 0·39mmol/l (0·7–1·03). Serum parathormone was 0·22μg/l which is inappropriately low for the serum calcium. Serum urea was 7·7mmol/l (3·3–8·8), creatinine 154mmol/l (40–110), and albumin 39g/l (30–45).

A magnesium load of 30mmol/l magnesium sulphate in 250ml of 5% dextrose was given intravenously over four hours, and urine collected over 24 hours for magnesium excretion. He excreted 68% of the total magnesium load in 24 hours (normal less than 50%) which shows excessive renal magnesium loss and excludes non-renal causes of magnesium deficiency.[1, 4] The calculation of the magnesium deficit is empirical as 99% of total body magnesium is intracellular and so serum magnesium does not accurately reflect total body magnesium. The deficit required to produce hypomagnesaemia is 0·5–1 mmol/kg of body weight. In our patient the deficit was 50mmol.

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TABLE

Gentamicin and other drug therapies in relation to serum calcium and magnesium

Day	*Drugs*	*Serum Ca mmol/l*	*Serum Mg mmol/l*
1	Gentamicin, flucloxacillin	2·11	0·84
18	Gentamicin, vancomycin	2·19	0·89
21	Drug rash	2·11	0·71
28	Gentamicin, fusidic acid	2·12	0·74
35	Gentamicin, fusidic acid	2·11	0·79
46	Gentamicin, fusidic acid	1·96	0·62
48	Gentamicin, fusidic acid	1·61	0·66
51	Gentamicin, fusidic acid (onset of tetany, gentamicin stopped)	1·36	0·45
52	Magnesium sulphate infusion	1·63	0·98
55	Magnesium sulphate infusion	1·62	0·86
56	Magnesium glycerophosphate orally	1·71	0·87
80	Magnesium glycerophosphate orally	2·29	0·79

However, to replace this deficit, excess magnesium had to be administered as our patient only retained 32% of a magnesium load. Therefore he required at least 150 mmol magnesium in order to retain 50 mmol magnesium. A total of 210 mmol magnesium sulphate was given intravenously over five days. No further tetany occurred and calcium supplements were not required. He was then given oral magnesium glycerophosphate for three months.

DISCUSSION

Magnesium deficiency can be the result of decreased intake (prolonged intravenous feeding, chronic alcoholism, intestinal malabsorption), increased non-renal loss (chronic diarrhoea, biliary fistula) or increased renal loss (diuretic therapy, renal tubular acidosis, drug-induced tubular injury [cisplatin, amphotericin, gentamicin], hyperaldosteronism).

In our patient, the evidence pointed to increased renal loss as a cause of magnesium deficiency as there was excessive renal excretion of magnesium, despite a low serum magnesium. The normal response of the kidney to hypomagnesaemia is to reabsorb the ion in the renal tubules. He had been on a diuretic from admission but this was later discontinued after a drug rash developed. Magnesium levels during administration of the diuretic were within the normal range. A small number of case reports show that prolonged aminoglycoside therapy is thought to cause a renal tubular leak of magnesium assessed by the renal response to a magnesium load.[2, 3, 5] Renal function may otherwise be entirely normal and toxic levels of the drug do not have to occur.

Hypomagnesaemia can cause hypocalcaemia by inhibiting the secretion of parathormone and by causing skeletal and renal resistance to the effects of parathormone.[1] Tetany in these patients will respond to intravenous magnesium sulphate and may not respond to intravenous calcium.[2] In our patient there was

an initial temporary response to intravenous calcium, but his hypocalcaemia was finally corrected by replacing magnesium, and oral calcium supplements were not required. It is suggested that all patients on prolonged aminoglycoside therapy should have regular measurements of serum calcium and magnesium. The renal tubular leak can persist up to three months and therefore prolonged oral magnesium therapy may be required.

REFERENCES

1. Berkelhammer C, Bear R. A clinical approach to common electrolyte problems: hypomagnesaemia. *Can Med Assoc J* 1985; **132**: 360-8.
2. Wilkinson R, Lucas G, Heath D, Franklin I, Boughton B. Hypomagnesaemia tetany associated with prolonged treatment with aminoglycoside. *Br Med J* 1986; **292**: 818-9.
3. Watson A, Coffey L, Keogh B, McCann S. Severe hypomagnesaemia and hypocalcaemia following gentamicin therapy. *Ir Med J* 1983; **79**: 381-3.
4. Bar RS, Wilson HE, Mazzaberi EL. Hypomagnesaemic hypocalcaemia secondary to renal magnesium wasting. *Ann Intern Med* 1975; **82**: 646-9.
5. Patel R, Savage A. Symptomatic hypomagnesaemia associated with gentamicin therapy. *Nephron* 1979; **23**: 50-2.

BOOK REVIEW

Insurance handbook for the medical office. By Marilyn Takahashi Fordney. 3rd ed. (pp 528. Illus, figs. £18.00, paperback). Philadelphia: Saunders, 1989.

I approached this book with interest because American-type medical insurance practices are rapidly approaching our shores. My curiosity was even further whetted on reading through the table of contents when I found Chapters 2 and 3 related to *Coding for professional services* and *Diagnostic coding* and Chapter 4 dealing with *Diagnosis related groups* (DRGs). In these there is an excellent 'Idiot's guide' to the coding systems ICD – 9 and ICD – 9 – CM, and a very full and general description of the rationale and working of DRGs.

These three chapters are, however, only 65 pages in a book of a total of 515, the remainder of which comprise a detailed and comprehensive exposition and explanation for the medical assistant, account's clerk and assistant treasurer in the North American medical domain. For them I am sure this is an update, easy to read and understand, which will enhance the skills necessary to handle medical insurance claims by physicians, surgeons and other doctors and their supporting staffs. It certainly will live up to its claim to help increase accuracy and efficiency on the job and thereby bring more revenue into the medical office. Nevertheless, interesting as it may be, it is not yet a required or recommended text for the British or Irish reader.

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2. Wilkinson R, Lucas G, Heath D, Franklin I, Boughton B. Hypomagnesaemia tetany associated with prolonged treatment with aminoglycoside. *Br Med J* 1986; **292**: 818-9.
3. Watson A, Coffey L, Keogh B, McCann S. Severe hypomagnesaemia and hypocalcaemia following gentamicin therapy. *Ir Med J* 1983; **79**: 381-3.
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Case report

Papillary carcinoma arising in a thyroglossal cyst

Claire M Thornton

Accepted 5 January 1989.

Cysts of the thyroglossal duct are common lesions, the majority of which present as a midline swelling in the anterior aspect of the neck. Infection and sinus formation are not uncommon complications, but the development of malignancy within a cyst is very rare. To date only 116 cases have been documented in the world literature.[1, 2, 3] This report describes a papillary carcinoma arising within a cyst which presented clinically as a simple cyst.

CASE HISTORY

A 65-year-old man presented with a painless midline swelling in the front of the neck, which had been present for two weeks and had been increasing in size. Examination revealed a mobile 2·5 cm diameter cystic mass in the anterior midline of the neck in the hyoid region, which moved on swallowing. The thyroid gland was normal and no regional lymph nodes were palpable. Aspiration of the mass yielded 4 ml straw-coloured fluid and reduced the size of the lesion. Cytological examination revealed poorly cellular fluid, the cell population being composed largely of histiocytes. No epithelial cells were seen.

At surgery the cyst was exposed and mobilised down to the level of the hyoid bone. The central portion of the hyoid bone was removed and the cyst widely excised up to the floor of the mouth between the hyoglossus muscles.

The specimen was a 2·5 cm diameter cystic structure. Histological examination showed a thick focally hyalinised fibrous wall, with foci of thyroid follicles and scattered aggregates of lymphocytes. The cyst lining was thrown into papillae, composed of fibrovascular cores covered by cuboidal epithelium (Fig 1). In areas the epithelium showed multilayering, nuclear overlapping and optically clear nuclei. Many of the stromal cores contained calcospherites or psammoma bodies (Fig 2). The features were of a papillary carcinoma arising within a thyroglossal cyst. Epithelial invaginations were present in the fibrous tissue but there was no evidence of invasion beyond the cyst wall. There were no metastases in a small draining lymph node.

A post-operative isotope thyoid scan showed normal and symmetrical uptake in both lobes of the thyroid gland. There was no evidence of a lesion within the thyroid gland and no ectopic thyroid tissue was identified.

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Claire M Thornton, MB, BCh, Registrar.

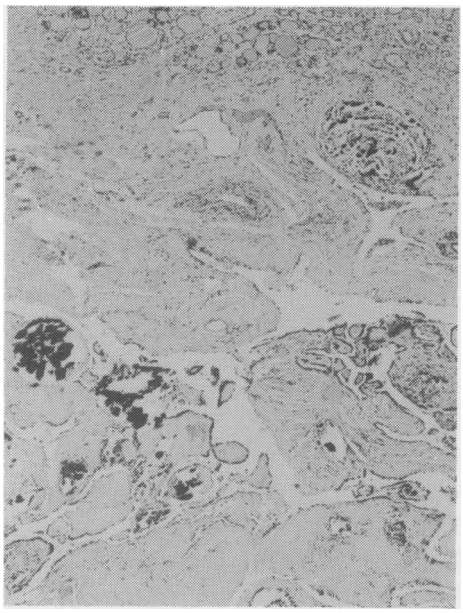

Fig 1. Thyroglossal cyst wall and papillary tumour in lumen (H&E, x 120)

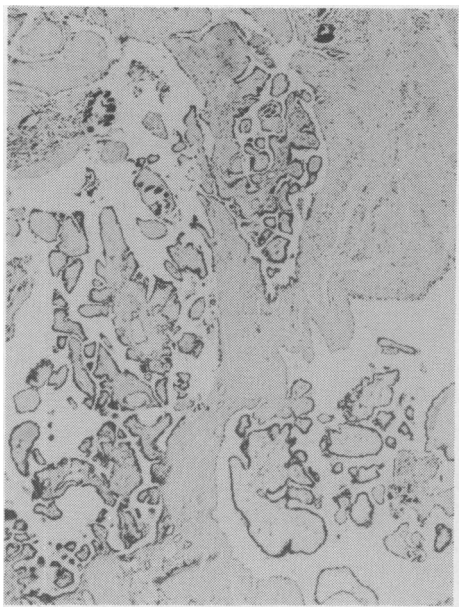

Fig 2. Papillary carcinoma with psammoma bodies (H&E, x 120)

DISCUSSION

Thyroglossal cysts are embryological abnormalities resulting from the persistence of the thyroglossal duct left by the descent of the thyroid gland. Cysts may form at any point along the line of descent from the base of the tongue to the retrosternal region, but most occur between the hyoid bone and the thyroid gland. The lining epithelium varies according to the site and may be squamous, cuboidal or columnar in type. The majority contain ectopic thyroid tissue and aggregates of lymphocytes deep to the epithelial lining.

While thyroglossal cysts are commonly complicated by infection and sinus formation, the development of malignancy is rare. An incidence of 1% is given by Allard.[4] In a review of the literature, 114 cases were identified[2] and more recently two further cases have been reported.[2,3] Malignancy may develop at any age but there is a predilection for females. Papillary carcinoma is the most usual histological variant, accounting for 83% of cases, and it has been suggested that the papillary carcinoma represents malignant metamorphosis in ectopic thyroid tissue.[2] Squamous cell carcinoma was found in seven of the 117 reported cases (6%). This arises in either a cyst high in the neck lined by squamous epithelium or in metaplastic squamous epithelium in an inflamed cyst situated lower in the neck. The remainder include adenocarcinoma, follicular carcinoma and mixed papillary-follicular carcinoma. Medullary carcinoma has never been reported in a thyroglossal cyst, perhaps because no parafollicular cells are found in ectopic thyroid tissue. Special stains were performed on this case and no parafollicular cells were identified. A pre-operative diagnosis of malignancy is rare and aspiration cytology proved unhelpful in this case.

The standard treatment for a benign thyroglossal cyst is the Sistrunk procedure. This remains adequate treatment if a papillary carcinoma is diagnosed on

histological examination and is found to be confined to the cyst, although some authors advocate further surgery if the tumour has breached the cyst wall.[4, 5] Metastatic spread is very uncommon. It is reported in 8% of cases,[4] with deposits occurring in the liver and lungs.

Joseph and Komorowski reviewed 52 cases of thyroglossal duct carcinoma, three of whom died as a result of extensive local recurrence or metastatic disease to the liver and lungs.[6] Two of these patients had a tissue diagnosis of papillary carcinoma and the third a diagnosis of squamous carcinoma. It is felt by some authors that the prognosis of papillary carcinoma in a thyroglossal cyst is similar to that of tumours arising in the thyroid gland.[1] Squamous carcinoma appears to have a considerably worse prognosis.

I am grateful to Mr M Bell and Mr V Loughlin for permission to report this case.

REFERENCES

1. Bosch JL, Kummer EW, Hohmann FR. Carcinoma of the thyroglossal duct. *Neth J Surg* 1986; **38**: 36-40.
2. Kimberley B, Cohen J, Posalsky S. Papillary thyroid carcinoma within a thyroglossal duct cyst. *Arch Otolaryngol Head Neck Surg* 1987; **113**: 206-8.
3. Banerjee SN, Ananthakrishnan N, Veliath AJ, Ratnakar C. Papillary carcinoma in a giant lateral thyroglossal cyst. *Postgrad Med J* 1986; **62**: 849-51.
4. Allard RHB. The thyroglossal duct cyst. *Head Neck Surg* 1982; **5**: 134-46.
5. Villet WT, Kemp CB, Thyroglossal duct carcinoma. *S Afr Med J* 1981; **60**: 795-6.
6. Joseph TJ, Komorowski RA. Thyroglossal duct carcinoma. *Hum Pathol* 1975; **6**: 717-29.

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Pages from the past

REMINISCENCES OF PARISIAN HOSPITALS

H Burden: Presidential Address to the Ulster Medical Society 1888. From the Transactions of the Ulster Medical Society, Session 1888–89; pp 50-59.

GENTLEMEN, — Few persons, I imagine, are disposed to deny that obvious and great advantages may be derived from a visit to one or more of the chief centres of civilisation in Europe. To become an eye-witness to the profound influence exercised over social and political life by modes of thought and action widely different from those to which we have become habituated is mental discipline of the highest order. Such experience tends to tone down rudeness, prejudice, and uncharitableness, the offspring of a too-restricted intercourse with our fellow-mortals; but, above all things, it impresses upon us with a force and vividness, otherwise unobtainable, that greatest discovery of modern civilisation — namely, the equality of all men. Nevertheless, while I am willing to admit all this, and much more, in favour of foreign travel, I candidly confess that, in my opinion, the professional culture resulting from attendance upon continental cliniques has been, and is, greatly over-rated. On the present occasion I shall not attempt to discuss the question, but shall merely point out that in the majority of cases imperfect knowledge of the language causes much waste of valuable time, together with erroneous interpretation of statements made by the clinical teacher, and that peculiarities in the organisation of foreigners, though real, affect the manner in which we treat our patients so immaterially that they may, without sensible error, be safely discounted by a British practitioner. A Frenchman or a French-woman — and I advisedly add woman, for a reason to which I shall hereafter refer — is, for all practical purposes, constituted on the same plan, is subject to the same accidents, suffers from the same diseases, and is amenable to the same treatment as one of our own countrymen. I do not ignore that fact, so properly and wisely stated by Professor Cuming in his Presidential Address to the British Medical Association, that differences exist in the proclivity of diverse races of mankind for certain specific diseases and morbid processes. My contention simply is that a continental course of medical study should be regarded as a luxury, not a necessity — a luxury which, nevertheless, I would cordially recommend to every one who can command the necessary time and money.

Such being my views, you will not be surprised to learn that though I have twice resided in the French Capital, during many weeks each time, yet my primary object in going there was not precisely to avail myself of the manifold facilities it offered for increasing professional knowledge. My first experience of the inner life in Parisian hospitals occurred during my honeymoon trip. On that occasion my wife and myself dwelt in the city for a period of about six weeks. Each morning, with few exceptions, I spent several hours in one or other of the great hospitals.

My second visit to Paris took place about eight years later, in the course of an extensive tour through France. During our three weeks' stay in Paris I renewed my acquaintanceship with hospital physicians and cliniques. The Hôtel Dieu, the Children's Hospital, the Hôpital Lariboisière, the Salpêtrière, claimed the larger share of my attention, but my favourite resort was the Hôtel Dieu. . . . .

. . . . In the Hôtel Dieu it was my good fortune to witness a feat in the art of percussion, which I had never previously, nor indeed have ever since, seen

equalled. A patient, suffering from kidney disease, was under examination by the physician in attendance. The name of the latter I am unable to recall to mind. Tall, spare, erect, his features were pale, sharply cut, and as impassive as though carved in marble; his eyes brilliant and piercing as a hawk's. A black frock coat, closely buttoned up to his throat, contributed to impart a thoroughly military air to his figure. He turned the patient over on his face and proceeded to percuss the region of the kidney. Then he called for pen and ink and drew, in outline on the skin of the man's loin, the form of the underlying organ. When completed, the figure represented a kidney of normal shape, but measuring about half an inch to an inch more than the healthy viscus in every diameter. A murmur of applause burst forth from the train of students in attendance, and the physician passed on in triumph to the next bed.

In the Children's Hospital Trousseau was now at the height of his glory. Here he continued to test to the utmost the efficacy of his special modes of treating croup, notable among which was his frequent recourse to section of the windpipe. Some years before my visits to the hospital he had reported the results of the operation in 215 cases, of which 47 recovered and 168 died. He concluded that there was little danger from tracheotomy, as he had performed the operation 121 times with only one mischance, so far as the operation was concerned.

If I went occasionally to the Salpêtrière it was not because of any extraordinary advantages it supplied for obtaining professional instruction. Charcot had not yet appeared upon the scene. Neuro-pathology in general, locomotor ataxy, scleroses of the nervous centres, and myo-trophic lesions in particular, were still involved in a cloud of misinterpretation and obscurity. What drew me to that ancient hospice was the historical associations connected with it and the weird old-world aspect it presented. Having recently quitted the fresh and handsome boulevards, peopled by a gay, frivolous, over-dressed, gesticulating, chattering throng, the latest development of an age too prone to drown all thought of yesterday and to-morrow in the enjoyment of to-day, on entering within the portals of the Saltpetre House one found oneself suddenly face to face with the surroundings of a period nearly three centuries old. Roughly paved, uneven courtyards and quadrangles, and buildings, whose grey, weather-beaten, time-worn walls proclaimed the antiquity of their origin, everywhere met the eye. The inmates themselves presented an appearance in consonance with their environment. For the most part aged and imbecile, they wore the aspect of a race who had survived the conditions of a long forgotten past. The dreamy quietude of the place subdued the mind to meditation on byegone events suggested by the scene. The nature of saltpetre, the invention of gunpowder, Louis XIII, the French Revolution, and the tragic fate of the illustrious chemist, Lavoisier, who once laboured here, were subjects that almost inevitably thrust themselves into the mental retrospect. How this slumbering abode of mental aberration has been roused from its lethargy! How Charcot has established in it a laboratory of research and a clinique of world-wide fame and resort! How it has, through his indomitable energy and zeal, been made to yield material for the elucidation of neurological problems that had baffled the efforts of all previous inquirers!

Before concluding, I must fulfil my promise to explain why, in my assertion that Frenchmen exhibit the same organic structure as Englishmen, I considered it necessary to make special reference to women. I did so for the following reason:– When Demonstrator in Queen's College, I was on one occasion asked by the Professor of Anatomy and Physiology to read and estimate the value of the

answers he had received from students to questions set by him. "Describe the collar bone?" was the substance of one query. When I came to the papers of a certain candidate, his answer proved extremely interesting. The young gentleman's style was graceful and flowing, yet somewhat light and discursive. The anatomy of the clavicle, to judge from the tenor of the earlier part of his communication, appeared to be a distasteful subject, since he merely used that bone as a peg upon which to hang some favourite theories and doctrines of his own, which had the slightest possible connection with it. It turned out, however, that he had been reserving his strength until he had arrived at the peroration, for on reaching the last line I read the astounding statement that Frenchwomen have longer clavicles than Englishwomen. The apparent magnitude of the discovery was artfully enhanced by the bald and simple language in which it was announced. The fact, if fact it were, being new to me, I was placed in a position of some difficulty. It it were true, to how many marks was the candidate entitled for his answer? But with the discussion of this knotty problem I shall not trouble you. On inquiry I found that our philosopher had enjoyed the advantage of an entire session's medical study in Paris. The knowledge revealed in his answer was, I have no doubt, a mere bagatelle compared with the stores of information of a like nature he had acquired during his residence in the French Capital. Whether he arrived at his discovery by the comparison of a series of careful measurements made in the Osteological Museums of Paris with others obtained in Great Britain, or in a less laborious but much more agreeable manner, I know not. While I am prepared to admit to the fullest extent the value of his discovery, should it be confirmed by other *savants,* I am, nevertheless, equally ready to make him or anyone else a present of it.

I would still hold to my belief that a Frenchwoman is constituted on essentially the same plan as an Englishwoman.

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Book reviews

Pauper to patient. A history of the Route Hospital, Ballymoney 1840–1987. By Cecil Burns, OBE, JP, DSc, MB, FRCGP. (pp 152, illus. £10.00 + £1.50 p&p). Coleraine: Impact Printing, 1988.

The hospitals of Belfast and Dublin, though few in the rest of Ireland, have been chronicled, some very thoroughly. Now Dr Cecil ('Joe') Burns tells the story of the Route Hospital, Ballymoney, and also something of its associated community services to which he himself has made a significant contribution. Though inevitably mainly of local interest — the Route worthies were devoted, some were colourful, but only one (Dr William Moore) was a national luminary — this readable book is also a serviceable prototype for any would-be local medical historian. Where Dr Burns has led others can (and should) follow.

The Route, like many Irish hospitals, descends from the Union workhouse and Infirmary, 130 of which were built in Ireland between 1840 and 1846 (40 per cent — including Ballymoney — with an isolation 'fever hospital') to the austere and uniform plans of George Wilkinson, poor law architect for England. The early days and personalities are described in crisply-written chapters chronologically structured, until the sale (for £3,000) of the workhouse in 1918 (by which time only ten inmates occupied accommodation planned for 700–1000), its repurchase in 1934 from the hapless vendors for £400, and its re-opening as the Route District Hospital on 25 July 1937. Thereafter the sheer pace of professional and administrative change and documentation, within the memory of many, call for a more discerning selection, and here the author's long involvement as local practitioner and member of the Northern Ireland Hospitals Authority (1955–62) and North Antrim Hospital Management Committee (1966–70) serve him well. The benign xenophobia of many non-metropolitan medicos is missing though very properly local pride is not: even when referring to the many perceived 'threats' to the future of the Route — which the author dates from as early as 1946 and were omnipresent when the book went to press — Dr Burns eschews the all-too-common litany of grievances, understands the issues, and preserves his innate commonsense. Queen's medical school is nowhere mentioned but then Queen's called little on the staff and repeatedly was unable to approve pre-registration posts at the hospital.

This book is essentially an *histoire* — a story — with no pretensions at analysis, deep discussion, or interpretations: 150 years are crowded into 131 pages competing with 58 illustrations (many full-page) and sundry in-text tables. Persons predominate, forming nearly half the entries in the general index. Yet the narrative has pace and the prose is forthright and lucid. Dr Burns has shown what deep attachment harnessed to energy and good sense can achieve using standard sources and local records.

The book is well produced, generously illustrated, has few misprints and those unimportant, and has 14 appendices of hospital staff and statistics. It is a pity that the referencing of so much material is inadequate: 100 superscript numerals and two extra pages of the bibliography would have made this book an important source of reference as well as readable. Dr Burns has worked hard, delved deeply, and read much: the fruits of his history and the needs of the serious reader require more rigorous referencing. Textual evidence of scholarship is not an inevitable recipe for boredom, least of all in Dr Burns's lively hands.

PF

Diabetes; scientific editor: R D G Leslie. (pp 316. £25.00). Edinburgh: Churchill, Livingstone, 1989. (British Medical Bulletin, vol 45, no 1).

For those interested in diabetes, the past decade has seen an explosion in the literature associated with the condition. In order to understand the pathogenesis, one needs to be geneticist, immunologist and physiologist. In order to treat patients, knowledge of biochemistry, pharmacology and metabolism is needed, as well as an expertise in endocrinology, neurology, nephrology, cardiology and psychology. Keeping up-to-date can be an obsessional chore but can be both effective and enjoyable by attendance at an international meeting. For those unable to attend the International Diabetes Federation in Sydney in November last year the publication of this book is timely. The editor has assembled authors who demonstrate the strength of interest in diabetes in the United Kingdom. The topics dealt with provide a comprehensive view of our understanding of the aetiology and pathogenesis of diabetes and its complications. Included in the science is practical advice on patient management. The challenge must now be to make this knowledge available to the patient through improvement in our organisation of diabetic care. I found reading this book valuable. It is cheaper but not so enjoyable as the meeting in Australia.

JRH

The Ulster Medical Journal, Volume 58, No. 1, pp. 117–118, April 1989.

Book reviews

Pauper to patient. A history of the Route Hospital, Ballymoney 1840–1987. By Cecil Burns, OBE, JP, DSc, MB, FRCGP. (pp 152, illus. £10.00 + £1.50 p&p). Coleraine: Impact Printing, 1988.

The hospitals of Belfast and Dublin, though few in the rest of Ireland, have been chronicled, some very thoroughly. Now Dr Cecil ('Joe') Burns tells the story of the Route Hospital, Ballymoney, and also something of its associated community services to which he himself has made a significant contribution. Though inevitably mainly of local interest — the Route worthies were devoted, some were colourful, but only one (Dr William Moore) was a national luminary — this readable book is also a serviceable prototype for any would-be local medical historian. Where Dr Burns has led others can (and should) follow.

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This book is part of a series of 'in depth' review texts which aim to give up-to-date and detailed insight into clinical and research developments in gastroenterological practice. In its foreword, the editor states that the objective of the book is to give a compilation of adverse drug reactions in the gastrointestinal tract and liver. An international team of contributors attempt to fulfil this aim.

Undoubtedly, iatrogenic disease is of great importance and deserves particular attention by all practising clinicians. To devote a volume of this eminent *Clinical gastroenterology* series to this problem is laudable but the end product is rather dry and uninspiring. The contributions vary greatly in their style but most oscillate between descriptions of the mechanisms of drug-induced damage and long lists of side effects attributable to individual drugs.

Chapters cover drug-induced damage in the oesophagus and stomach, malabsorption, diarrhoea, constipation, pancreatitis and a wide range of drug-related liver disorders. The coverage is extremely broad and well referenced with many 1987 papers quoted. Some controversial and topical issues such as the role of aspirin and non-steroidal anti-inflammatory agents in gastrointestinal bleeding are discussed in fair detail but other areas such as halothane and the liver are treated rather superficially.

It is attractive for the gastroenterologist to have close to hand a well referenced compact volume detailing adverse drug reactions in the gastrointestinal tract. Nonetheless, in clinical practice, most doctors will choose to consult their pharmacy department or drug information service if they suspect a drug problem and will likely obtain information which will be at least as up-to-date and probably more comprehensive. Thus, I question the value of a 'book of lists'. The more readable sections of the book deal with mechanisms of drug injury and yet this is peripheral to the stated aims of the volume and some of the contributors have shied away from detailed discussion of this aspect.

This volume will be of practical value to the clinician with a gastrointestinal interest who has limited access to a drug information service. It will serve as a reference book but will rapidly become dated. I shall continue to consult with my colleagues in the Pharmacy Department. BC

The kidney and hypertension in diabetes mellitus. Edited by Carl Erik Mogensen. (pp 423. £80.95). Boston: Nijhoff, 1988. (Topics in renal medicine, 6).

The development of nephropathy as a complication of type I diabetes is of major importance. The 40 per cent of patients who develop persistent proteinuria have a 40-fold increased mortality rate when compared with age- and sex-matched control subjects. Only one in four of diabetic patients diagnosed as having persistent proteinuria survive for 10 years. Many believe that nephropathy is part of a process of malignant angiopathy. Recent studies have shown that early monitoring of urinary albumin excretion can predict later development of established nephropathy and that the progress of the disease can be slowed by therapeutic intervention.

The above text is a multiauthor review which covers, comprehensively, the pathology, pathophysiology and treatment of both renal disease and hypertension in diabetes. Among the topics covered are early renal hyperfunction, prevalence and incidence of microalbuminuria, diabetic nephropathy and pregnancy and the effect of antihypertensive therapy on the course of renal function. The authors are well-established international authorities and have produced an up-to-date and valuable reference work, which should, as the editor hopes for in his introduction, stimulate further advancement in this important clinical area. As a book I recommend it to all serious students of diabetes. ABA

Color atlas of AIDS. By Alvin E Friedman-Kien. (pp 300. £25.00). Philadelphia: Saunders, 1988.

This beautifully produced colour atlas is written by the physician who first described the association between Kaposi's sarcoma in young homosexual men. In fact, an alternative title for this work could have been 'Kaposi's sarcoma and other manifestations of HIV disease', as 80 pages of the book are dedicated to the mainfestations of Kaposi's sarcoma, its diagnosis clinically and microscopically, its ultrastructure, and differential diagnoses. There are 333 colour plates in all (22 dedicated to Kaposi's sarcoma). The remainder of the book gives a concise account of the development of the epidemic, the identification of the virus, and other clinical manifestations, especially dermatological ones. The text is not detailed enough to make this a work for specialists in the field of HIV although it is well referenced. It would be a very useful book for general medical departments and general practitioners who increasingly will be expected to recognise cutaneous and other manifestations of HIV disease. The text provides a guide to relevant microbiology investigations. At £25.00 it would seem too expensive for medical students, but it is a book which could usefully be carried by medical libraries for reference by students. RDM

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