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THE ULSTER MEDICAL JOURNAL

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Contents continued —

The Ulster Medical Journal, Volume 55, No. 1, April 1986.

The Ulster Medical Journal

THE ULSTER MEDICAL JOURNAL

NOTICE TO CONTRIBUTORS

1. Authors are reminded that concise and clearly expressed papers are those most welcomed by readers and the Editorial Board.

2. Manuscripts should be typewritten in double spacing, with wide margins. They should be fully corrected and alterations in proof may be disallowed or charged to the author. A sample typescript showing layout is available on request from the editorial office.

3. The text should indicate the purpose of the paper, and should include an introduction, sections on materials and methods, results, and a discussion relevant to the findings. A brief factual summary should be provided at the beginning of the paper.

4. Scientific measurements should be in SI units (*Units, symbols and abbreviations; a guide for biological and medical editors and authors,* 3rd ed. London: Royal Society of Medicine, 1977). Blood pressure may be expressed in mmHg and haemoglobin concentration as g/dl.

5. Tables must be kept simple and vertical lines should be avoided. Tables and illustrations must be kept to a minimum and data should not be given in both text and tables. Line drawings should be used where possible and symbols must be large enough to be legible when reduced to text size. Where possible, size of illustrations and tables should be planned so that one or more can easily fit the page size of 20 × 12.5 cm. Photographs and other illustrations should be unmounted, and authors may be charged for these at cost price. Authors' names and the top of the figure should be marked in soft pencil on the back.

6. References should be restricted to those really necessary and useful. This journal uses the 'Vancouver' style (see British Medical Journal 1982; **1**: 1766-70 and Lancet 1979; **1**: 429-30). Text references are numerical. Each reference should include:
   i) a list of all authors when six or less (when seven or more only the first three should be listed followed by *et al*).
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   eg
   McCoy GF, Dilworth GR, Yeates HA. The treatment of trochanteric fractures of the femur by the Ender method. *Ulster Med J* 1983; **52**: 136-41.

   Book references should give the author, title, edition, town of publication, name of publisher, year of publication, and, where appropriate, volume and page numbers.

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sacrificed a lot of his leisure time to the preparation of this book and it deserves to become the logical successor to *Physics for the anaesthetist* by Sir Robert McIntosh. I can highly recommend it to anaesthetists of all grades, theatre and intensive care technicians and nurses. DLC

Orthopaedics and trauma. Edited by S P F Hughes. (pp 118. Illustrated. £19.95). London: Baillière Tindall, 1985. (Current operative surgery).

It is a pleasure to receive this book for review and I cannot imagine that any orthopaedic specialist would think otherwise.

Professor Hughes and the publishers have amply fulfilled their remit in selecting, from established experts in their fields, a wide spectrum in current orthopaedic operative technology. That super-specialisation is inevitable is well demonstrated and it would be virtually impossible for any reader to evaluate each and every chapter critically from personal experience. Some chapters may even incur frank hostility!

That each team of orthopaedic surgeons (and we do work in teams) will require a copy is certain. It will be simply devoured by those in training with an appetite for further knowledge and innovative technology. The layout and standard of production is excellent, with a slight lapse in proof-reading at the word 'neoplasm'. The diagrams relating to operative technique are exceptionally clear and pleasing, the radiographs only occasionally indistinct, and clinical photographs are restricted, as (though restful to readers) they are seldom instructive. This excellent and varied collection of specialist articles is well worth the price and will find its way on to many individual bookshelves and into every departmental orthopaedic reading room — my own copy is already on its way there. PO

Blood transfusion and blood banking. Guest editor, William L Bayer. (pp 306. £13.75). London: Saunders, 1984. (Clinics in haematology, vol 13, no 1).

During the past few years there has been a very marked change in the pattern of blood transfusion therapy. Thus, whereas requirements for red cells have remained fairly constant, the usage of various blood components has increased enormously. The many developments in blood component therapy are well reflected in this volume of *Clinics in haematology.* The book comprises a series of review articles, mostly of North American authorship but also including a substantial input from the U.K. Most of the articles are directed towards haematologists and transfusion specialists, so that transfusion support in haematological malignancies, congenital coagulation disorders and haemolytic anaemias are each very fully covered by well established experts. Major omissions include the management of haemorrhage, acquired coagulation disorders and transfusion support of intensive care patients. Some parts will be of interest outside haematology and in this respect I particularly enjoyed the chapter on the clinical use of immunoglobulins. This growing field is very well reviewed by workers from Edinburgh who are actively involved in the development as well as clinical assessment of new immunoglobulins.

Another rapidly expanding area of transfusion medicine involves the use of apheresis techniques for therapeutic purposes and also for collection of blood components from donors. Both aspects of apheresis are discussed very fully in separate chapters, each extensively referenced. Description of the side effects of transfusion therapy is limited to one chapter on transfusion-related infections. The latter is disappointing because, while transfusion transmitted cytomegalovirus infection is discussed at great length, reflecting the interests of the authors, the more important areas of AIDS and particularly non-A non-B hepatitis are dealt with rather sketchily. In the case of AIDS, much of the information is unavoidably obsolete, having been written before the discovery of HTLV III as the causative agent.

WMMcC

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The Ulster Medical Journal, Volume 55, No. 1, April 1986.

THE ULSTER MEDICAL SOCIETY

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May we, therefore, appeal to you to join the Ulster Medical Society, and so enable us to widen its influence and sphere of usefulness still further? A proposal form is appended; your proposer and seconder must be Fellows of the Society. If you do not know any Fellows please contact the Honorary Secretary. All persons registered as medical practitioners under the Medical Act shall be eligible for election as members of the Society (Constitution, Section VI). Temporary membership may be allowed at the discretion of the Council.

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. . . . . 19 . . . .

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This covers one volume (two numbers) of the Journal.

The Ulster Medical Journal, Volume 55, No. 1, pp. 1 – 14, April 1986.

Dialysis and transplantation: problems for the future

Presidential Address given to the Ulster Medical Society, 31 October 1985.

Mary G McGeown

HAEMODIALYSIS

The effective treatment for renal failure dates from the invention during World War II of the first workable artificial kidney by Dr Wilhelm Kolff in Klampen in occupied Holland. He demonstrated that it is possible to remove waste products of metabolism from the bloodstream, thereby cleansing the body when the kidneys have failed. Moreover, he showed that clinical improvement followed rapidly. By repeated use of this treatment, some patients could be kept alive long enough for their own kidneys to regain normal function. As far as I am aware, he provided the first evidence that acute renal failure need not be irreversible. While the idea of removing toxic substances from the bloodstream was not new, Kolff was the first to devise practical equipment for this purpose (Fig 1). The patient's blood was passed from a cannula in the radial artery into a long tube of cellophane sausage-skin which was wound around a supporting drum-shaped framework. The frame rotated on a spindle so that the blood-filled tubing was bathed in a tank containing dialysis fluid. The cleansed blood was returned by another cannula inserted into a convenient large vein and the process repeated until the biochemical abnormalities were considerably ameliorated. The blood was prevented from clotting by repeated injection of heparin into the blood circuit.

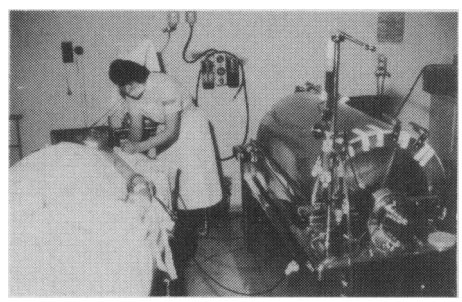

Fig 1. One of Kolff's rotating drum artificial kidneys, first developed about 1948.

Kolff went on to devise an improved artificial kidney in which the cellophane tubing is contained within a compact coil, the necessary large surface area provided by two tubes, the bloodstream being divided into two to supply each tube. This design was capable of being manufactured and sterilized ready for use — the first disposable kidney. In all coil dialysers the dialysis fluid is pumped around the blood-filled dialysis membrane, rather than the tubing itself being rotated through a stationary bath of dialysis fluid. The coil is supported in a container and the dialysis fluid is pumped through the coil and splashes back into the bath to be recirculated again. Fig 2 shows the twin-coil kidney with which we began our service for renal failure in Belfast in 1959.

Mary G McGeown, CBE, DSc, MD, PhD, FRCP, FRCPEd, FRCPI, Consultant Nephrologist, Physician in Administrative Charge, Renal Unit, Belfast City Hospital, Belfast BT9 7AB.

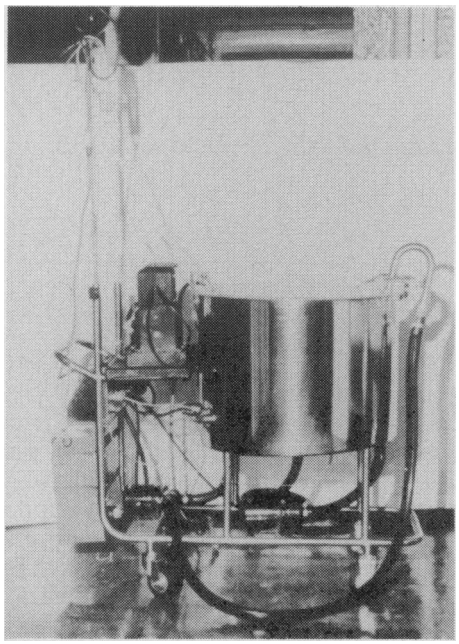

Fig 2. Kolff's twin coil kidney, Belfast, 1959.

Several other forms of artificial kidney have been devised. These include flat plate dialysers in which two sheets of dialysis membrane are sandwiched between plates of polypropylene which are engraved with grooves, along which the dialysis fluid is pumped and passed to waste. The prototype of this was designed by Kiil in 1960 and uses two such sandwiches. The artificial kidney now most widely used is the capillary kidney in which the blood is passed through enormous numbers of capillary tubes made of dialysis membrane, around which the dialysis fluid is pumped.

After the War, Kolff gave artificial kidneys to Hammersmith Hospital in London, Mount Sinai Hospital in New York, and the Royal Victoria Hospital in Montreal, all of whom reported successful treatments. Sadly the kidney which he gave to Amsterdam was never used. The artificial kidney stimulated interest in the kidney, its functions and its diseases. At Hammersmith Hospital there was very active interest in the kidney when Dr Graham Bull came as a Research Fellow from Cape Town in 1948. He was involved, with Milne, Borst and others, in the early use of the artificial kidney. By his accounts the original Kolff with its wooden tank and framework was difficult to set up and patients developed convulsions and rigors during treatment. He became more interested in water and electrolyte balance and the dietary management of renal failure, and published seminal papers with Borst and Milne.[1,2] They developed the subsequently famous Bull-Borst diet, a protein-free regimen which provided energy as pure carbohydrate and oil. The aim was to provide enough energy to prevent wasting of body tissues, thus sparing nitrogen production, without giving sodium and potassium. The diet was difficult to take, the patients became nauseated by the mixture of glucose or lactose and oil, and developed sore mouths. Equally important was the concept of fluid restriction, because patients with uraemia become thirsty, and, if allowed to drink freely, develop oedema and then heart failure. Graham Bull was appointed the first full-time Professor of Medicine in Queen's University in 1952, bringing to Belfast the new expertise in management of kidney diseases. I had the good fortune to join his Department as an MRC Research Fellow in 1953.

DEVELOPMENT OF HAEMODIALYSIS IN BELFAST

Up to this time Professor Bull had maintained that most patients with acute renal failure would recover with conservative treatment and that the population of Northern Ireland was not big enough to justify an artificial kidney. In 1958, the son of one of our consultant obstetricians developed acute renal failure following a road traffic accident. Conservative treatment did not seem to be enough and he

was flown to Hammersmith, where it was found he was already producing urine again and he did not need dialysis after all. However, questions were asked in Stormont about the need for an artificial kidney for Northern Ireland, with the result that it was decided to set up a Renal Unit in the Belfast City Hospital. The choice of the City Hospital for the siting of the new service was due to the late Mr John Megaw (Fig 3). I was given the task of setting up the service, with the grade of Junior Hospital Medical Officer, in 1958, to be elevated to Senior Hospital Medical Officer in 1960.

Fig 3. John McIlroy Megaw, FRCSEd, consultant surgeon at the Belfast City Hospital, 1948-71.

The artificial kidney arrived one day in early June 1959 but the ward was not ready for occupation for another nine months. It happened that a GP refresher course was being held at the City Hospital two days later and Mr Megaw, always an enthusiast, insisted that I should set it up and demonstrate it to the GPs. By this time a technician, Mr Maurice Bingham, had been recruited from the Biochemistry Laboratory to assist me. I had seen a twin coil once; he had never seen one at all. The pair of us read the booklet, which came with the apparatus, and proceeded to set it up using red ink to mimic the blood circuit, much to the satisfaction of Mr Megaw and his class of GPs.

Meantime, Dr Eliahou from Israel, who was finishing a year's attachment with Professor Bull, learned of the arrival of our equipment and wanted to see it in action before departing for home. Professor Welbourn tied the ureters of a dog and we waited 48 hours for it to become uraemic. Our first haemodialysis was carried out in the animal theatre in the Department of Surgery. We managed to give the dog a short dialysis, enough to demonstrate a satisfactory fall in blood urea. Dr Eliahou returned to Israel where he later set up the Renal Unit in Tel Aviv, and he is now a world expert on acute renal failure.

At this point, I felt that I was ready to visit the Renal Unit at Halton to see how dialysis should really be carried out. However, the following week an elderly man was admitted in uraemia due to prostatism. He was semicomatose with a blood urea over 600 mg/100 ml. Mr Megaw demanded that I should treat him by dialysis. 'You dialysed a dog last week, you cannot let my patient die without trying'. Mr Maurice Bingham and I set up the twin coil kidney in Ava 2 theatre (which no longer exists) and Miss Eileen Martin, the Medicine Department technician, weighed out three sets of chemicals to make up three changes of the dialysis fluid. Mr Megaw inserted the cannulae in the radial artery and cephalic vein, and in fear and trepidation we treated our first patient. My stepson, John Freeland, sat in my car at the front of the Ava to act as messenger. When I wanted to send a blood sample to the laboratory, or when I needed to have an extra set of chemicals weighed out after I had stupidly spoilt one bath of dialysis fluid, I just stuck my head out of the window and hollered for him to come up. It was a very

successful treatment in that the blood urea fell from around 600 mg/100 ml to 200 mg/100 ml after about six hours, and the patient wakened up. Unfortunately he died within a week from a cerebrovascular accident.

From then on we were in business. We were still without the promised accommodation in Ward 9, and our headquarters consisted of a small room in Ward 15 formerly used as a store. The hospital van took us and the kidney to the patient, most often to the Royal Victoria Hospital where we dialysed many times in the classroom between Wards 1 and 2, and 3 and 4, in the old ward theatres and later in Ward 22 where Dr Gray was beginning to provide ventilation for patients with respiratory failure. The second patient recovered from 36 days of virtual anuria and was well when I last heard of her a few years ago, more than 20 years later. Another early recovery from acute renal failure after an incompatible blood transfusion is working in the Wakehurst Unit. Not all the patients recovered but, looking back at those early days, the recovery rate seems to have been higher than it is now. This is probably because patients with grave injuries or illness are now resuscitated and live long enough to develop renal failure but may not survive other complications, sometimes dying after renal function has returned. And so I never got to Halton to see how dialysis should be done, and was entirely taught by the book.

There were many problems for our travelling dialysis service. Plugs never fitted and there always seemed to be a problem about the water supply. The team consisted of Maurice, replaced in the spring of 1960 by Mr Jack Lyness, and myself. About two hours were needed to prepare the apparatus; the treatment lasted six hours, with another hour or more to clear up. It never seemed possible to complete the procedure in less than 10 hours.

Things became easier when we moved into the new unit in Ward 9. Thereafter the patients were brought to us, except for the very seriously ill on respirators. I was still the only doctor; there was no question of a rota. Eventually, in 1963, I was able to persuade Matron that we needed our own nurse and Staff Nurse Kay Maguire was appointed. She has contributed enormously to the development of the service and as Senior Nursing Officer leads the now large nursing team. From this point the work increased greatly and patients with undiagnosed renal failure began to be admitted. Many of these had chronic renal failure. At that time the artificial kidney was not used for treatment of chronic renal failure because all available superficial blood vessels were soon used up. This situation was changed in 1960 by the invention by Scribner[3] of an external shunt between artery and vein, which could be opened for treatment, then reunited as a shunt. This could be repeated many times allowing treatment to be continued almost indefinitely.

Our first arteriovenous shunt was inserted by Mr Will Hanna, then a senior registrar, in 1964. The first patient died from septicaemia after a short time. This occurred because we attempted to make the treatment less expensive, re-using the twin coil by washing it free from blood with saline and storing it in the refrigerator between treatments. His brother was to become our first transplant later on. Both brothers had renal failure from polycystic disease.

The second patient commenced treatment in January 1965, and soon became well enough to live at home, returning twice a week for treatment; thus we were able to keep her well but were prevented from accepting any new patient. She had a transplant in St Mary's Hospital, London, in 1965, which functioned well for seven years, eventually being lost due to a deep venous thrombosis which

followed an aeroplane journey. This transplant led to a partnership with Professor Peart's unit and a regular small trickle of patients was sent to London for the transplant operation, returning to Belfast for long-term supervision of the immunosuppressive treatment necessary to prevent rejection of the graft. I will return to the transplant story later.

By this time the flat plate kidney had appeared. This had several advantages for treatment of patients requiring regular haemodialysis, the most important of which was that the volume of the complete blood circuit was much less than that of the twin coil kidney. This meant that the kidney could be primed with saline instead of blood which was needed for the larger volume coils. Moreover the cost of the cupraphane membrane sheets was very little compared with coils costing £22.00 in 1965 money. An engineer in London called Heppel made a copy of the original Swedish model for treatment of his wife in the Royal Free Hospital. He later set up a small business to manufacture Kiil kidneys. He was kind enough to let me have two sets on the understanding that I would pay for them some time. We set to and taught ourselves how to build the brutes, by sweat and tears. Professor Love may remember finding me one Saturday literally in tears because the wretched kidney developed a leak every time it was tested. There were many new tricks to that trade which had to be learnt.

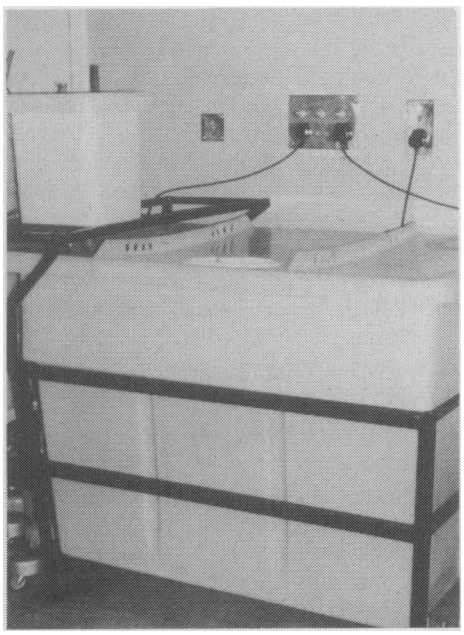

Fig 4. Four hundred litre tank for dialysis fluid, installed 1965.

The Kiil kidney is flushed by fresh dialysis fluid at the rate of half-a-litre per minute, and, as the treatments then lasted 14 hours, a large volume of dialysis fluid was required (Fig 4). The necessary salts had to be weighed out and the fluid mix prepared in a huge plastic fish tank, containing 400 litres. We ordered our tank but it was not to be ready for several months so we set about treating two patients in a make-shift way using the 100-litre tank of the original coil kidney. The patients were treated at night so that the coil kidney could be used during the day for the acute service. Mr Megaw kindly lent us one of his two cystoscopy theatres for this purpose. The tank of fluid had to be replenished four times during the night. The service was possible only because Mr Jack Lyness volunteered to sleep on a couch in the surgeons' room four nights a week, to be awakened by the night nurse when the fluid level went down to a critical point. Mr Lyness did this in addition to his daytime duties for about six months until the new tanks arrived. The cleansing of the Kiil kidneys presented a problem. It is horrifying now to think that, oblivious to the perils of hepatitis B, the bloodstained Kiils were carried the length of Ward 9 to the bathroom at the far end where they were scrubbed in the patients' bath. The dialysis tanks had to be scrubbed and sterilised with hypochlorite and, on occasions, our washings leaked through to Casualty situated just beneath us. At

one stage, patients frequently developed rigors during dialysis, but the cause eluded us for some time. Eventually we discovered pigeon droppings in the water tank which supplied the Unit. The rigors disappeared when we installed a direct supply from the water main.

In 1964 it was envisaged that there would be a haemodialysis unit in the new Tower Block, and in that year I drew a sketch plan, with the enthusiastic help of Mr Paddy Semple, of what I thought was required. In 1965 it became clear that some provision for the growing needs of the Renal Unit would be necessary long before the most optimistic estimate for the opening of the Tower Block. The only way this could be provided was by a new building behind Ava, as there was no space available in the main hospital. The successful treatment of our second patient by regular haemodialysis, followed by transplantation, determined us to develop a service for the treatment of chronic renal failure, which would include transplantation. There was still no other doctor assigned to the Unit though some of Professor Bull's British Council Research Fellows worked with us from time to time to gain practical experience in the management of renal failure. From 1960 onwards a succession of British Council Fellows came to Belfast for the specific purpose of working in the Renal Unit, a number of whom stayed long enough to acquire a PhD. They were Doctors P Metaxis, A Billis, N Papadoyannakis, D G Oreopoulos (all from Greece), R Gupta, J Jindal (India), H Goetz (Hamburg), I Taraba (Hungary), and M A O Soyannwo (Nigeria).

The first phase of the building, now known as Renal I, was ready in July 1968. It contains six single-patient rooms, a theatre and a two-bed dialysis room, later to become the transplant theatre, plus the usual offices. The patient rooms are situated between 'clean' and 'dirty' corridors, communicating with each. The staff enter the Unit through changing rooms, as to a theatre suite. All medical and nursing care is provided from the 'clean' corridor. Disposal of used items is via pass-through cupboards to the 'dirty' corridor. Each area has its own air supply and differential pressure ventilation ensures that air flows from 'clean' towards 'dirty' areas. The system provides good quality reverse barrier nursing, without need of airlocks, provided that the discipline of usage is carried out meticulously. The design of the Unit makes it possible to nurse patients with low white cell counts due to immunosuppression, who are very susceptible to infection, in rooms adjacent to patients with acute renal failure who are often infected.

The expansion of the service has depended on a series of technical advances. When several patients are treated simultaneously a very large volume of dialysis fluid is required, and it is no longer feasible to make up batches of fluid in large tanks. This problem was solved by the introduction of concentrated dialysis fluid made commercially at 35 times the working concentration, which is diluted automatically by a proportioning system of pumps controlled by conductivity meters. The dialysis fluid is no longer recirculated, as it was in coil kidneys, but is a single pass to waste, so that the blood is always dialysed against fresh fluid. Enormous quantities of water are required to dilute the commercial concentrate to working strength. The water must be purified by filtration to remove suspended 'gunk', then passed through a de-ionizer to remove calcium and aluminium, both of which are injurious if continuously absorbed during the many hours of treatment. Ideally the water should have a final purification by reverse osmosis but we have been able to dispense with this due to the good quality of Belfast water.

During the late 1970s, several systems were developed permitting dialysis by single needle. The bloodstream is immediately divided by a Y-junction into two

streams, one entering and one leaving the artificial kidney. The best of these systems uses two blood pumps, the return one being set to return the blood very rapidly to prevent mixing with the blood about to pass into the dialyser, as mixing would obviously reduce efficiency. This extra equipment is expensive but has obvious advantages to the patient. Thanks to the Northern Ireland Kidney Research Fund we were able to change to single needle haemodialysis as early as 1978.

HEPATITIS

The account of haemodialysis would be incomplete without mention of hepatitis. Hepatitis infection increased rapidly in European renal units in the late sixties. It affected staff as well as patients and some staff members died. In 1967-68 a transplant surgeon died in Edinburgh, and Manchester lost a doctor and a nurse. At that time it was customary to transfuse patients on dialysis as a matter of routine when their haemoglobin became low. It was thought that hepatitis was spread by blood transfusion and by 1968 many units began to avoid transfusion as much as possible. In 1969 laboratory tests for hepatitis B (formerly known as Australia antigen) became available and a pilot study carried out by the Public Health Laboratory Service showed that most hepatitis in renal units was hepatitis B. Attempts were made to control the outbreaks by testing sera from staff and patients regularly, dialysing infected patients in isolation and improving cross-infection precautions. Some centres trained patients with hepatitis for home dialysis but the disease sometimes spread to family members. Hepatitis B has a long incubation period, six weeks to six months, and these patients, unlike most other groups of patients receiving blood transfusion, return repeatedly to hospital, eventually becoming infective when viraemia develops. Staff and other patients become contaminated by spillage of blood or staff pricking themselves accidentally with contaminated needles.

In Belfast we were fortunate to have the help of Dr John Connolly who set up the tests very quickly, and routine testing was instituted. Renal II was opened in May 1972 and from the beginning followed the practices recommended by the Rosenheim Committee. Despite this, we experienced hepatitis for the first time in July 1972. At the beginning of June, a patient with renal failure was admitted from a peripheral hospital, and as usual on admission had the hepatitis B antigen test carried out which was reported as negative. He soon required regular haemodialysis, and was prepared for the programme. He had his first haemodialysis in Renal II on 11 July, blood being taken for a second hepatitis B test on that day as part of the routine surveillance of the Unit. On account of the 'Twelfth' holidays, it was not until 15 July that it was discovered that the test was positive. It then became known that he had received transfusion the day before he came to us, and so must have been incubating the disease. In the meantime the patient had received a second haemodialysis in the normal way. He was then isolated in a side-ward and a list of volunteers was drawn up to treat him. As the Unit was not yet fully operational there had not been any sharing of equipment with any other patient and we were optimistic that all would be well. However, three other patients developed hepatitis, the last one in October 1972. On the advice of Dr Sheila Polakoff from Colindale, we set up isolation dialysis in two mini-caravans at the back of the Unit. The Unit was closed for the admission of new patients and transplantation ceased for six months. Some patients died untreated as a result of this action. However, no further cases developed. Three patients eventually died but one survived, eventually to be declared non-infective and to have a transplant

in 1973, being well with excellent graft function over 12 years later. You can appreciate this was a very anxious time for us all, anxiety becoming acute when a patient developed a positive test shortly after one of the nurses had pricked herself while putting him in dialysis. Mercifully she did not develop jaundice but later was found to have the antibody indicating sub-clinical infection.

Hepatitis now occurs only as rare sporadic cases in the UK, but it seems to be on the increase in Europe as a whole. However, immune globulin is now available for protection of staff and patients if there is a serious risk of infection.

PERITONEAL DIALYSIS

Over the years during which these improvements were taking place in haemodialysis, other developments were occurring. It had been known from the end of the nineteenth century that water and some other substances could pass from the peritoneal cavity into the bloodstream and vice versa, the peritoneal membrane acting as semipermeable membrane. From 1920 onwards numerous experiments in animals and a few attempts in humans were made to use the peritoneum for dialysis.

Peritoneal dialysis can be carried out without special equipment other than the plastic cannula and modified Y-piece infusion sets. In theory, at any rate, it can be carried out in any hospital ward. Although the method is simple, great care is needed to prevent infection of the peritoneal cavity. Like everything else, it works better for the experienced. Since 1977 a new concept for the use of peritoneal dialysis has evolved from the work of Popovich and Nolph[4] in the USA. They showed that continuous peritoneal dialysis for six or seven days per week, changing the fluid only four times in the 24 hours, provides very effective control of uraemia. The patients experience remarkable improvement in wellbeing. The disadvantage of this method is a high incidence of peritoneal infection. Oreopoulos[5] in Toronto has introduced several improvements into this system. The most important is the use of plastic containers for the dialysis fluid which, after the fluid has run in, can be rolled up and carried in a pouch attached to a belt around the waist. The same bag is then used to collect the spent dialysis fluid. A special flexible permanent silastic cannula is used.

The CAPD method can be taught to patients of even moderate intelligence in about two weeks, compared with the two or more months' training needed for self-haemodialysis. The only equipment needed is a stand to support the bag while the fluid is flowing into the peritoneum by gravity, and a small table on which to set out a sterile towel, wipes, cleansing fluids and clamps. Storage space is required for the bags of fluid, sterile dressing packs and the necessary cleansing fluids. Oreopoulos has done a great deal to make CAPD an acceptable method of treatment and continues to investigate the long-term effects. I am proud of the fact that he received his nephrology training in Belfast and holds a PhD of Queen's University.

RENAL TRANSPLANTATION

As long ago as 1902, Ullman in Vienna demonstrated that a kidney could be transplanted from its normal site into the neck, and urine would be produced. A kidney from another animal, even another species, would produce urine. Carrell and others repeated this experiment and found that a dog whose own kidneys had been removed would remain well for a few days, after which the transplant ceased to function. Attempts to transplant kidneys in the human by

Voronoy in the Ukraine in 1933 were unsuccessful, as were several attempts in Boston in the early 1950s. About the same time, animal experiments in Boston showed that skin could be transplanted between litter mates, the animals not 'recognising' the transplant as foreign tissue. This led to the first successful kidney transplant operation in 1954 by Murray, also in Boston, between identical twins. Over the next five years, transplantation between identical twins was carried out in a number of centres in Europe and America. Some failed for technical reasons but in none was there evidence of rejection. An identical twin transplant carried out in Belfast in 1962 was a technical failure.

It is obvious that few patients reaching end-stage kidney failure are fortunate enough to have a twin able and willing to provide a kidney. The question was, could rejection be prevented when the graft was taken from a less closely related individual? Whole body irradiation was used to prevent rejection but the patients died from uncontrollable sepsis because of bone marrow suppression. Attempts were made to use immunosuppressive drugs and Roy Calne and others reported in 1960 that mercaptopurine prolonged survival of kidney grafts in dogs but was very toxic. Burroughs Wellcome produced a derivative of mercaptopurine, azathioprine, which proved to be a good immunosuppressive drug and much less toxic that the original. In 1962 azathioprine was used successfully when a kidney taken from a patient dying during an open heart operation was transplanted into an unrelated individual. Azathioprine thereafter became generally accepted as the main immunosuppressive drug for transplantation for almost two decades. In 1962 Goodwin reported successful treatment of several rejection episodes with steroid, in a mother-to-child transplant, although the child finally died from sepsis. After this report the combination of azathioprine and steroid was accepted as standard therapy for kidney transplantation. In 1976 cyclosporin A, a fungal metabolite, was found to be a potent immunosuppressant and is now widely used, though it too has serious drawbacks. The age of drug immunosuppression had arrived, and opened up the possibility of treating end-stage kidney failure by transplantation.

From the time of Murray and Calne's demonstration in 1962 that cadaveric transplantation was possible with the aid of azathioprine, it seemed to me that transplantation would prove to be the definitive treatment of end-stage renal failure. Good quality dialysis therapy would be needed to make the patients fit and maintain them until kidneys could be provided. If the graft fails, the patient should survive to return to dialysis therapy and have a second, even a third or fourth graft.

My first attempts to put this philosophy into practice were possible through the help of the few centres in the UK doing experimental work in transplantation. Professor Woodruff transplanted one of my patients from her father in 1962. She did not survive, but over the years 1965-68 Professor Peart at St Mary's Hospital, and later Professor Roy Calne, accepted my patients. Figure 5 shows Professor Calne, by this time Professor

Fig 5. Professor R Y Calne, FRS, FRCS, with Mr J Neill, who received a renal transplant in Cambridge in April 1966.

of Surgery in Cambridge, with his longest surviving cadaver transplant, one of my patients, who was transplanted in April 1966. The graft continues to function excellently.

Our transplantation programme began in 1968 soon after the opening of Renal I. A second consultant nephrologist was appointed and surgeons were designated to take part in the transplantation programme. (Table I). Some are no longer working with us — Dr John Hewitt, Dr Joseph McEvoy and Mr Stewart Clarke have left Northern Ireland, Dr Sam Nelson has left tissue typing. Nurse Kay Maguire who was the first renal nurse in 1963 and is now our senior nursing officer, and Mr Jack Lyness, technician since 1960, have both contributed greatly to the development of the service. I must stress that the good results we have achieved are due to the efforts of the whole team. I must also pay tribute to the great help we have received from the staff of intensive care units who have provided kidney donors. We owe a particular debt of gratitude to the Respiratory Intensive Care Unit in the Royal Victoria Hospital who have provided by far the largest number of donors, and to the others who have found kidney donors. The procurement of a donor entails a considerable amount of extra work for the doctors and nurses concerned. Our tissue typing service was set up under the leadership of Dr Sam Nelson. Following his resignation, Dr Derek Middleton has become head of the service, and indeed has contributed greatly to the development of the work over the last 13 years.

TABLE I

Renal transplantation in Belfast

Anaesthetists:	J P Alexander, C J Hewitt, J Gamble, C Gardiner.
Nephrologists:	M G McGeown, J McEvoy, J F Douglas, C C Doherty.
Surgeons:	S Clarke, J A Kennedy, W G Loughridge, R A Donaldson, R Johnston.
Tissue Typing:	S D Nelson, D Middleton.
Nursing Officer:	K Maguire (1963).
Chief Technician:	J Lyness (1960).

Between 1968 and 1985, 347 patients have received 392 grafts, there being 38 second grafts, six third grafts and one fourth graft. Most of the grafts came from cadavers, only 36 coming from live donors. Two hundred and forty-five patients are alive with functioning grafts, and 11 others are alive, having returned to dialysis after failure of their graft. We had the good fortune to achieve outstandingly good results of patient and graft survival from the beginning of our transplantation programme, and over the years since statistics have been collected centrally, have been at the top of the 'league table'.[6]

Now after almost 17 years it is possible to identify at least some of the factors which contribute to our high success rate which has continued over all this time. The careful organisation of the team has resulted in good clinical care. The most important point in the preparation of the recipient is now known to be the giving

of blood transfusion, which we have done from the beginning of our programme, despite the fact that transfusion was then beginning to be considered a risk factor rather than helpful. The drugs we have given to prevent rejection of the kidney were those in general use but we decided to use much lower doses, particularly of prednisolone, in the hope of reducing toxic side effects. We hoped to improve the results of transplantation by avoiding infection, particularly fatal infection. This we were able to do, by isolation of the patients through the use of the specially designed building, and by using very low doses of steroid compared with other centres.

THE PROBLEMS OF RENAL REPLACEMENT THERAPY

I have described to you a truly marvellous story — the discovery of how the function of a vital organ may be replaced after failure, permitting life to continue for years. Indeed the kidney is the only organ where complete failure of function can be treated. Hard on the heels of this discovery came the development of renal transplantation. Successful transplantation restores the patient to almost complete normality. Machine treatment, dietary and fluid restriction become unnecessary and the patient merely has to take a few pills daily to prevent rejection. Normal energy is regained, indeed patients with transplants become fit, even for competitive sport. One patient starred in the recent victory of Clandeboye in the Irish Senior Cup Golf Competition. Parenthood is possible even for women. One patient has had three successful pregnancies since transplantation. Many others have had one child and several have had two.

As soon as a new service develops, the demand for it grows rapidly. By the end of 1964 we were treating one patient. It was possible to squeeze in treatment for the first patient without detriment to the acute programme. The first patient died after a short time, and other patients followed one by one during 1965. Table II shows the number of patients receiving treatment from 1964 when we began regular haemodialysis until we moved to Renal I in 1968. The number of patients transplanted each year is also shown. Thanks to Professor Vallance-Owen, and the devoted work of Sister Wallace and the nursing staff of Wards 3 and 4 RVH, two patients were regularly given twice weekly peritoneal dialysis from 1968 until 1972 when Renal II was opened. After 1968 the numbers of patients treated increased slowly year by year (Table III), but this in no way satisfied the demand.

TABLE II

Renal replacement therapy in Belfast 1964-1968

Year	**Number of patients on treatment*	*Number of patients transplanted*
1964	1	2
1965	1	2
1966	4	3
1967	4	2
1968	8**	7

*Patients on treatment at any one time.
**Includes 2 patients in Ward 3, RVH.

TABLE III

Renal replacement therapy in Belfast 1969-1985

Years	*Number of patients on treatment	Number of patients transplanted
1969-72	8	16
1972-73	20	31
1974-78	30	106
1979-80	37-39	61
1981	40	25
1982	46	46
1983	50	38
1984	55	37
1985	65	34

*Patients on treatment at any one time including PD.

Since 1978 the increased efficiency of the dialysis equipment, and technical advances in general, have allowed the duration of dialysis to be progressively reduced from 28 to 16 hours weekly for an average-sized adult. We give the weekly treatment divided into two sessions. Most other centres give it in three sessions. This is more costly, which means that fewer patients can be treated, and that the patients have to attend on three occasions. My own observations lead me to consider that two longer sessions provide adequate treatment for most patients, provided they follow the regular discipline of fluid intake and salt restriction. The fact that some of our patients have remained reasonably well for as long as seven years on twice weekly haemodialysis shows that thrice weekly dialysis is in the nature of a luxury.

The shortfall in facilities has always meant that many patients who could benefit do not receive treatment. The very success of the treatment compounds the problem, as relatively few patients die. I have used the figure of 40 patients/million/year as the annual incidence of new patients with end-stage renal disease. This figure is based on a survey carried out during 1969-71 in which many Northern Ireland doctors took part by filling in letter cards to notify their patients with renal failure. It refers to patients aged 5-55 years, medically suitable for treatment, and excludes diabetics, and some other patients who would now be considered treatable. The problem of the shortfall of facilities has been made worse because we have progressively expanded the possibilities by showing that older patients do remarkably well on dialysis therapy and can also be transplanted. We have transplanted with success up to the age of 65. Moreover, it has been shown that some diabetics, and even patients with coronary bypass or ileal conduits, can be transplanted successfully.

How do the facilities available measure up to the needs of our population? You must bear in mind that the figure of 40 new patients per annum does not permit us to treat anyone older than 55 years, and does not allow for diabetic or other disadvantaged patients. It is, however, the target set by the DHSS for all units to achieve by 1987. A few patients die during the first year but about 83% are alive

at the end of the year to be joined by 40 new patients appearing in the second year. In the third year 86% of the first set of patients and 93% of the second set of patients remain to be joined by the new ones. Even at the end of the third year, 80% of the first set of patients remain alive. The number of patients goes on increasing until eventually equilibrium is reached when the new patients are equalled by patients dying. The statisticians tell us that equilibrium will be reached only after 25 years, but 90% of steady state will be reached after 10 years.

The cost of regular dialysis therapy, of course, escalates year by year as the number of patients increases. If the programme is expanded to include older people, diabetics and other disadvantaged patients, more will die each year, but in the meantime they will have had more medical complications and cost more to treat. It is difficult to arrive at a true cost but several years ago the DHSS costed hospital haemodialysis at £11 – 13,000 per annum, and home dialysis at £6 – 8,250 per annum. Home haemodialysis costs less but, if the patient receives a transplant, the cost of converting the home is lost and it takes two years of home treatment to break even. Satellite dialysis is nearly as cheap as home treatment and, as it avoids the cost of converting individual homes, it is the best buy. However, it is cheaper only because of the economy of staffing that becomes possible when only fit stable patients are treated. The saving in cost in the satellite is, to some extent, offset by increased costs in the mother unit which copes with the early problems, and accepts back patients who get into difficulties. Moreover, the older and otherwise disadvantaged patients are not suitable for treatment in a satellite centre.

Transplantation is cheaper but the immunosuppressive drugs cost about £1,200 per annum or more if cyclosporin is used. The saving is illusory as the transplanted patient is replaced by a new patient. The transplant often does not cover the life of the patient, who may need dialysis treatment again. While one of our patients has had a well-functioning graft for more than 20 years, and 47 for 12-15 years, the average is less than 10 years. Six patients have had three grafts and one a fourth graft. We must, however, remember that transplantation provides much better quality of life than any form of dialysis. While it is not possible for all patients the contraindications are relatively few. The limitation to transplantation is the number of donors which can be obtained.

The United Kingdom lags far behind other European countries in its provision for dialysis treatment, particularly for patients over the age of 45, because of lack of resources. Dialysis and transplantation are paid for here by the National Health Service. There is virtually no private dialysis or transplantation, except in London, where it is used almost entirely for patients from overseas. In other European countries, the financial cost of treatment is provided by insurance, and each treatment is paid for directly. If we lag behind Europe, all Europe lags behind the United States. In 1972 laws were enacted there which effectively provided treatment for all American citizens with end-stage renal failure, without charge to themselves. This led to a phenomenal growth of haemodialysis in America, where all patients are accepted regardless of age and of other diseases present. The Medicare budget for end-stage renal failure now approaches three billion dollars. In America it was considered morally intolerable to ration treatment for financial reasons, but this rocketing cost has caused a complete re-think. The medical profession there have been very critical of British nephrologists, blaming them for accepting rationing of treatment because of limited finance. They are now having to face the same moral dilemma.

I put it to you that the very success of our treatment for end-stage renal failure has produced a moral and ethical, as well as financial, crisis of the first magnitude. All patients who could benefit cannot be treated. Who then should be treated and how do we select them? Other advancing medical fields such as heart and liver transplantation must face these issues also. When resources are limited, how to resolve the competing claims of 'high-tech' medicine and the care of the mentally handicapped or the elderly? There is no real solution to this moral dilemma. In Northern Ireland we are on the brink of further development of renal services. The opening of the Tower Block will increase facilities for renal transplantation — but only if a sufficient supply of donors becomes available. However, we can be cautiously optimistic as fewer kidneys will have to be exported because of lack of suitable recipients and of beds. The beds vacated by transplantation moving to the Tower will permit considerable expansion in CAPD, to about 30 new patients per year. The provision of a satellite dialysis centre in the northwest of the Province would provide a further small increase in patients. It should eventually become possible to accept 60 new patients/million/annum, that is 90 for the Province. Even then there will be hard decisions to be made.

Research into the causes and treatment of kidney diseases holds the prospect that some types of end-stage renal disease may be prevented, or at least delayed in onset. Let me conclude by acknowledging the enormous help which we have been given by the Northern Ireland Kidney Research Fund.

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The last of the fifty — a time of change

Annual Oration at the opening of the 1985-1986 teaching session, Royal Victoria Hospital, delivered on 3 October 1985.

John W Dundee

It has been said that some things never change. As an example one can take the opening words of the 1956 annual report of the Royal Victoria Hospital:

> 'Although the financial stringency continued, the organisation of the Hospital has been adapted to meet the difficulties'

This was an eventful year. The death occurred through a motor accident, on 4 April, of the pioneer neurosurgeon, Cecil A Calvert. The Hospital was awaiting the opening of wards 25 and 26 for metabolic and skin diseases and of wards 23 and 24 for gynaecology (thus vacating 17-18, which would become a fracture unit). Continuity of junior staffing was simplified by changing the date of appointment of senior house officers from 1 October to 1 August, although housemen now lost the opportunity of a well-earned rest, a lucrative GP locum-tenancy, or a short trip as a ship's doctor. The Hospital had 595 beds and 4 cots, a 'Consulting Staff' of 50, and 30 other consultants with clinical responsibilities. One third of the latter were anaesthetists and a further third from various surgical specialties (EENT, gynaecology, thoracic and neurosurgery). The Staff, as distinct from the total body of consultants, was limited to 50, was self-elected and, although it only consisted of about 60% of consultants working in the Hospital, was the recognised channel of communication from the doctors to the Hospital Management Committee and the University.

Vacancies on this prestigious body occurred on the retirement or demise of an active member, and the writer was elected in 1961, on the death of Ivan McCaw. This was the last election to the Staff; the author was the last of the fifty and this was a time of change.

On reading the history of Belfast medicine, one becomes aware of the very major part played by eye, ear, nose and throat surgeons in many important developments. It was only in the mid-thirties that these specialties began to separate. An early pioneer, Samuel Brown, RN, founded Mill Street Dispensary, pioneered the establishment of the Belfast Ophthalmic Hospital and was elected Mayor of Belfast in 1870. His son, John Walton Brown (later Sir John), appointed to the staff of the Belfast General Hospital in 1876, had specialist training in both ophthalmology and otology and combined them with duties as a general surgeon. He, as Chairman of the Medical Staff when the new hospital was officially opened on 27 July 1903, made a speech of welcome to King Edward VII and Queen Alexandra. Brown, who was Deputy Lieutenant of the City of Belfast in 1913, was knighted for his services to the community in 1920, and has been affectionately referred to as the 'last of the Dandies'.[1]

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Two EENT surgeons presided over the changes which were to take place in the Staff. James R Wheeler was elected Chairman of the Staff in 1961. A rugby player of no mean ability with seven Irish caps to his credit, he had been appointed assistant surgeon to Belfast Ophthalmic (now Shaftesbury Square) Hospital in 1928, becoming senior surgeon in 1936. Assisted by Kennedy Hunter, he was the mainstay of ENT services in Belfast during the last war and it is on record that he saw 90 outpatients on one morning session (without the help of an SHO or Registrar). He was followed by J Allison Corkey, son of a wellknown Presbyterian cleric who was the first person in Belfast to come on the staff of a teaching hospital purely as an ophthalmologist (in 1934). With William Anderson (son of the founder of Anderson & McAuley Ltd) and William Dickey, he kept the ophthalmic service going in the Benn Ulster, Ophthalmic and Royal Victoria Hospitals during the war years. These three hospitals amalgamated in 1948-49 resulting in the opening of our new Eye and Ear Clinic in 1964-65.

STAFF CHANGES

The 1961 annual report lists 119 people who provided specialist medical or dental services on the Grosvenor Road site (Table I). Only about 40% of these formed the staff committee (the *Staff*) i.e about half of the doctors. The largest group comprised those actively involved in the treatment of disease, followed by those who encouraged patients to heal themselves, with a smaller group who aided in the diagnosis of disease. At that time anaesthetists formed the largest group of specialists not on the Staff.

TABLE I

Purveyors of specialist medical care

The Staff		49
Surgical	30	
Medical	13	
Diagnostic	6	
Other consultants		42
Anaesthetists	10	
Surgical	8	
Others	24	
Senior Hospital Medical Officers		13
Dentists		15
	Total	119

The minutes of the Staff held on 12 February 1962, with Mr J R Wheeler in the Chair and 34 members present, contains the following statement:

> 'The Royal Victoria Hospital Medical Staff Committee has, for some time past, realised that its present constitution did not give all members of the staff a representation. It was also felt that if the Staff did not put their own house in order it might be done for them at a higher level.'

As the feeling of the Staff seemed to be in favour of some reorganisation, the Chairman gave notice that he would raise the matter in more detail at the March

1962 meeting of the Committee. At a meeting held on 12 March 1962 he made this appropriate proposal:

> 'For some time past the Consultant Medical Staff of the Royal Victoria Hospital have realised that the Medical Staff Committee, as at present constituted, was not fully representative of all the interests concerned. Prior to the Health Services Act, the Staff Committee numbered somewhere about 30 persons. With a rapidly increasing number of staff this number was considerably increased, and the Staff at that time agreed to bring the numbers of the Staff Committee up to fifty persons.
>
> 'Now the number of consultants appointed by the Hospitals Authority to the Royal Victoria Hospital is 86 and the question is how to give a proper representation to everyone concerned. With this object in view it is proposed to hold a general meeting of all consultants who have a contract with the Northern Ireland Hospitals Authority to discuss the whole matter.'

This was a contentious issue but most of the talking and argument appears to have taken place before the actual meeting. Dr John Weaver proposed and Mr Ernest Morrison seconded that the Staff Committee should immediately be opened to all specialists who worked on this site. Dr D A D Montgomery proposed and Dr J F Pantridge seconded a more conservative motion that membership be opened to all consultant doctors with a major commitment on this site. The final decision to open up the Staff to all who provided specialist services on this site was taken as the result of a postal ballot, with 25 for and 10 against. The result was announced on 9 April and Mr Allison Corkey had taken over as Chairman before the first full meeting was held on 8 October.

To accommodate the expected large crowd the venue was moved to the Institute of Clinical Science and 52 attended what was the first open meeting of the Medical Staff Committee of the Royal Victoria Hospital in more than 60 years of its existence. As might have been imagined, the attendance rapidly dropped off almost back to its old levels and certainly the percentage attendance which had been 70-80% soon became 35-40%.

At the beginning of 1961 the Staff was mainly composed of the surgical specialties (Table II). These increased almost overnight to 49 out of 106 doctors, but the percentage was virtually unchanged. There was, however, a great difference in the disciplines within the surgical group which had changed little

TABLE II

"Surgical" specialties on the Medical Staff Committee

	1956	*1961*	*1962*
General	9	10	11
EENT	5	4	7
Gynaecology	3	2	4
Neurological	1	3	3
Orthopaedic	2	2	3
Thoracic	2	3	3
Plastic	1	2	2
Anaesthetists	3	4	16
Total	26	30	49

during the five-year period 1956-61. During the same interval representation of anaesthetists had increased by 300 per cent.

From 1962 on, we have had a democratic form of representation of medical staff on the appropriate committees which determine hospital policy. The extent to which advantage was taken of this differed greatly: some played a very full part in hospital committees, others felt that this was the province of the administrators and some were just not interested. In an expanding health service there was some administrative work for each of us and it was up to us to find where our particular knowledge was most needed.

FAMILY TIES

Was the old Staff a family clique? It would be easy to think this, but at the time of enlarging the Committee there were only two sons of previous members on the Staff, J W S Irwin and H M Stevenson, with one son-in-law, C H G Macafee. There were the orthopaedic brothers-in-law, R J W Withers and R J Wilson, and one very complicated intermarriage — Mr J Allison Corkey's cousin, Denise, married to T T Fulton, whose sister is married to J W S Irwin whose cousin is married to J S Logan. The author found that he had some remote family ties with at least three other members of the Staff. Taken together, one quarter of the Staff had some family ties with previous or current members, but this reflects family traditions in medicine rather than family cliques.

Since 1962, seven sons of former consultants have become members of the Medical Staff Committee, so family tradition continues. Ties are now growing up across the Donegall Road with the Logan, Lowry, Bridges and Mackay families.

TIME FOR CHANGE

The writer of Ecclesiastes reminds us (Chapter 3)

> 'For everything there is a season, a time for every matter under heaven
> A time to be born and a time to die
> A time to plant and a time to pick up that which is planted
> A time to kill and a time to heal
> A time to break down and a time to build up . . . '

Could it be that for our Hospital this was not only a time *of* change but a time *for* change? Many of the structural changes to the Hospital, with their staffing and financial implications, were even more radical than modification of the structure of the Staff Committee. These were only part of a number of changes occurring in Belfast medicine at that time; those of us involved were probably not aware of their significance and they only became obvious in retrospect.

ULSTER GRADUATES

In 1946, all clinical and paraclinical professors were Ulstermen — Crymble, Thomson, Wilson, Myers, Biggart, Allen and Lowry. By the early fifties the first four of these had been replaced by Englishmen (as were the first six eventually), who brought new ideas to the Medical School. The Royal Victoria Hospital had even appointed an English surgeon and a Scottish neurosurgeon.

This trend was reversed in 1956 when John R Gibson was appointed to the newly founded Chair in Mental Health. He had trained in London and at Massachusetts General Hospital in Boston. The author was the next Ulsterman to return, in

1958, to head the new Department of Anaesthetics, having trained in Liverpool and at the University of Pennsylvania. The appointment of local graduates who had gained their postgraduate experience outside the province, and usually outside the UK, soon became established practice with both the University and the Northern Ireland Hospitals Authority. Other Ulster-born academics appointed in the early sixties include J H M Pinkerton and I C Roddie.

The writer of the book of Proverbs had advised this practice, many centuries ago (Ch. 5, v. 15) when he told the people to

> 'Drink waters out of their own cistern, running waters out of their own well'.

The powers that be must have liked the water they provided as the University staff list for 1985-86 shows that 20 out of 26 medical professors are local products and that all but two of the clinical departments are headed by Ulstermen.

NEW SUB-SPECIALTIES

The development occurred, often assiduously, of completely new specialties (or sub-specialties), nephrology, cardiology, medical statistics, epidemiology and metabolic diseases were among the earliest of these. Each had its own requirements, including special fundings and it is reasonable to think that each would want representation in the 'corridors of power'.

ANAESTHETICS AND INTENSIVE CARE

There was unexpected expansion in some existing specialties, of which anaesthetics is a good example. There has already been one depressing quotation on the subject of hospital finance from the 1956 hospital report. An equally depressing one dealt with anaesthetic services.

> 'The demand for anaesthetic services continues to rise, and this, combined with the shortage of junior staff entering the specialty in Northern Ireland, has made the task of providing anaesthetic cover extremely difficult during the year.'

As today, services were provided on a group basis (Royal Victoria Hospital, Royal Maternity Hospital, Royal Belfast Hospital for Sick Children), and junior cover for the site was then provided by three registrars and 6 SHOs. All new trainees spent three months with consultants before being permitted to do operating sessions.

It was in the years 1960-62 that the main changes occurred, both in the training, the work and the status of the anaesthetist, although many of these were initiated years previously. Anaesthetics is now among the best organised specialties on a province-wide basis and there are three reasons for this rise from the doldrums:

(i) *Teaching.* From 1958 postgraduate courses and rotations were organised on a city-wide basis and from 1960 on a province-wide basis. Anaesthetics was the first specialty to do the latter, a practice which is now widely accepted. Only when one organises comprehensive postgraduate teaching courses do hidden talents among colleagues emerge. Good teaching encourages good trainees, who feel someone is interested in them. Equally important, a trainee who has been encouraged will, in turn, pass this enthusiasm on to others when appointed as a consultant.

(ii) *Developing specialised services.* The 1956 hospital report mentions the introduction of hypothermia for the treatment of 'closed' head injuries. It states that 'up-to-date refrigeration equipment has greatly facilitated this

work'. Credit must go to J F Bereen for initiating this service. It was introduced very soon after the initial Liverpool publication on the subject.[2]

Hypothermia is not now used in these circumstances but it was one of the early instances of an anaesthetist perfecting a complicated technique which helped his surgical colleagues. Controlled hypotension for plastic surgery and middle ear operations is another example of this combined approach to a surgical problem, but the best known is open-heart surgery.

Tribute must be given to Dr W M Brown who, with Mr T B Smiley, initiated animal experiments which led to the first use of cardiopulmonary bypass in man in the province. It is on record that in December 1961 the Royal Victoria Hospital Committee agreed to finance a research fellowship for R S J Clarke, in the grade of Senior Registrar for a four-month period, to perfect the running of the extracorporeal circulation (heart-lung machine), a task for which he had gained specialised experience in St Bartholomew's Hospital, London. The anaesthetist was now becoming more than the 'gas man' and it was only as his vision and skill increased that the work of the surgeon could advance.

(iii) *Respiratory intensive care.* Seemingly most important in present-day practice, but yet depending on the points already mentioned, was the development of a completely new type of medical care-management of respiratory failure from which has evolved the Intensive Care Unit.

The use of muscle relaxants and the production of apnoea is routine practice in many anaesthetic techniques and manual or mechanical artificial respiration is part of the everyday practice of anaesthesia. When a severe outbreak of paralytic poliomyelitis occurred in Denmark in the mid-1950s, the method of ventilating these patients was with an 'iron lung'; which was bulky and not nearly as efficient as the method used in anaesthesia. Lassen, Ibsen and their colleagues, all familiar with ventilating patients during thoracic operations, organised a relay of medical students and manually ventilated over 300 patients with poliomyelitis.[3] At the beginning of the epidemic the Blegdam hospital in Copenhagen, the chief centre for communicable diseases, had one tank and six cuirass respirators.

> 'During several weeks we had 40-70 patients in our hospital requiring continuous or intermittent bag ventilation. To do this we have employed about 200 medical students daily. Their pay was about 30s (£1.50) for eight hours.'

The outcome was dramatic: the mortality in a disease which was almost universally fatal was reduced to under 50% and positive pressure breathing had established its place in medicine. The same workers later[4] reported the survival of three out of four patients with severe tetanus, who were similarly ventilated after inducing muscle paralysis with curare.

The 'iron lung' forced air out of the lungs by pressure on the chest wall from without (like the Schaefer method of artificial ventilation), whereas the anaesthetist blew air (or oxygen) directly into the lungs as in mouth to mouth respiration. Because of their familiarity with artificial ventilation in the operating theatre, anaesthetists became the recognised authority on its therapeutic use and they were frequently consulted by their medical and surgical colleagues.

Three groups of patients often required respiratory assistance in the late 1950s. The first were chronic bronchitics who had a flare-up of the disease during the cold moist Ulster winter. The second were patients with severe poliomyelitis — in

1962 there were 31 paralytic cases of poliomyelitis in the province, of which two were severe. Tetanus was also common at that time and the spasms of a severe attack were sufficient to kill the patient through hypoxia. These spasms could be reduced by drugs but best results were obtained by completely paralysing the patients and ventilating them artificially. Dr William Bingham had already successfully managed the manual ventilation of two patients with tetanus in Lurgan Hospital around 1955-56, while Dr Maurice Brown had similarly ventilated a myasthenic patient in the Royal Victoria Hospital.

Owen Wade, then Professor of Therapeutics and Pharmacology, sought help with two chronic bronchitis patients in the winter of 1959, and early in 1960 curarisation and ventilation was organised for two patients with tetanus. A successful outcome of these and ventilation of a patient with poliomyelitis prompted further requests, but this was very demanding work and difficult to combine with normal clinical practice. In October 1960 Dr Robert C Gray was appointed full-time research fellow with the objective of investigating the need for and feasibility of providing a clinical service for patients with respiratory insufficiency. At the same time the author was allocated two clinical sessions per week for this work, and the involvement of the anaesthetist in direct patient care, as distinct from the mere provision of a 'service-specialty', had started. They were providing *intensive care* — although focussed on respiration. Patients' vital functions were monitored continuously and the foundations were laid for current practice.

Ventilation on several sites was impractical and steps were taken to organise a proper unit. The neurologists had provided facilities with two beds in ward 22 and in September 1961 the Medical Staff Committee supported the suggestion of the Management Committee that

> 'in view of the greater risk of cross-infection among cases under treatment in this unit than among VD cases, the two twin-bedded wards at present being used for VD cases should be exchanged for a four-bedded ward, thus leaving the two twin-bedded wards for respiratory failure cases.'

The balcony on Ward 22 was later closed off to provide a bedroom for the resident anaesthetist and a functional respiratory failure unit (RFU) became operative by the end of 1961. Once the nursing authorities realised that these patients required specially trained nurses, their co-operation was excellent, as was that of the physiotherapists, who had now found a useful new application for their skills.

The continuing success of this Unit was shown when Dr Gray was asked to give the 1962 Calvert Lecture on his work in the development of the Respiratory Failure Unit. This service was getting growing support from the whole hospital staff and cases were being referred from all over the province. To complete the service a consultant anaesthetist with a major interest in this subject was needed and an appointment was made in the following year.

In more ways than one, this was a time of change. It saw not only the 'end of the 50' but the beginning of a revolution in anaesthesia. Ventilators have changed beyond recognition since then. They started with a simple electrically-driven bag-squeezing machine which embodied a three-speed Sturmey-Archer gear change (as was then popular in bicycles). This was followed by the more sophisticated and sturdy Smith-Clarke and Cape ventilators designed by Captain Smith-Clarke, who had helped design Daimler and Alvis cars, and whose daughter had poliomyelitis. These 'heavies' were later augmented by simple oxygen-driven

machines (Bennett; Bird); more versatile in performance, but not as reliable. Now there are very sophisticated electronic machines which only the experts can understand.

In the space of a decade — 1956-1966 — medical practice was revolutionised yet it remained patient-centred. A computer-programmed ventilator is typical modern equipment: with the advent of the microprocessor we are in the midst of the next decade of change. Will this remove us far from direct personal contact with our patients? Can we still remain as humane and caring a profession at the end of this era as we did in the period reviewed here?

Thanks are due to the Medical Staff Committee for permission to quote from old minutes and to Mrs Olive Russell for patiently tracing the relevant paragraphs.

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Post-transfusion hepatitis: a problem in Northern Ireland?

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SUMMARY

A retrospective analysis of post-transfusion hepatitis reported to us from 1980 through 1984 revealed 16 patients. We believe that this apparently low incidence is due to lack of notification and make a case for direct notification to us of any suspected cases. Disqualification of implicated blood donors is of prime importance in prevention of transfusion-associated hepatitis.

INTRODUCTION

Reports from different parts of the world suggest a range of incidence of post-transfusion hepatitis from approximately 7% in the USA,[1] 3.4% in one Dutch study[2] to 2% in Sydney, Australia.[3] The only available information for the UK shows an incidence of 2.4% in patients undergoing cardiac surgery in Newcastle-upon-Tyne.[4] Hepatitis in these studies included clinical and sub-clinical episodes (indicated by abnormal liver function tests after blood transfusion).

The aetiology of jaundice in a patient who has previously received blood and/or blood products includes hepatitis B, Epstein-Barr virus and cytomegalovirus. Non-A, non-B hepatitis continues to be associated with blood transfusion, but there is little published evidence of the true incidence of this condition, its clinical importance or long-term consequences.[5, 6] We report on a retrospective analysis of post-transfusion hepatitis notified to us from 1980 through 1984. Prior to this period, notification was anecdotal and follow-up tests on blood donors were minimal.

METHODS

All blood used in the province is collected by the Northern Ireland Blood Transfusion Service from volunteers. Blood donors are routinely tested for hepatitis B surface antigen (HBsAg). Since 1982, the method used is a radio-immunoassay (RIA: Blood Products Laboratory): a sample of each unit of blood is tested within 24 hours of the donation and prior to issue to hospital blood banks. An aliquot of serum from each donation is stored as a reference sample at −20°C for 18-24 months. When an incident of post-transfusion hepatitis is notified, all the units of blood and blood products are traced to individual donors by the date and unique donation number. It is possible to perform additional tests on implicated donors using the relevant stored samples if the notification is within 18-24 months of transfusion.

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When post-transfusion hepatitis is recognised in a patient, the first step is to initiate laboratory tests for hepatitis A and B, cytomegalovirus and Epstein-Barr virus. These tests are performed by the Virus Reference Laboratory, Royal Victoria Hospital. No test is available at present for the detection of non-A, non-B infection which remains a diagnosis of exclusion.[7]

The next step is notification of the patient to the NI Blood Transfusion Service with date(s) of transfusion and donation numbers of all blood and blood products. The investigations and follow-up include:

a) identification of all volunteer blood donors implicated by the unique donation numbers;

b) the relevant stored (−20°C) donor serum is tested for hepatitis B surface antigen (HBsAg), antibody to hepatitis B surface and core antigens (anti HBs, anti HBc), and for liver function tests;

c) recall of donor(s) for a fresh sample of blood to repeat the tests and ensure that implicated donors are excluded from blood donations until all investigations are completed.

RESULTS

Sixteen patients with post-transfusion hepatitis were reported from 1980 to 1984 (see Table). There was a sudden increase in the number of patients reported in 1984 which we attribute to a letter written by one of us (CB) in December 1983, to all haematologists in the province, stressing the importance of notification. Eleven of the 16 patients had no evidence of infection with hepatitis B. Four patients were HBsAg positive and one patient had anti HBc of IgM type indicating response to recent infection with hepatitis B virus. Results of subsequent tests for HBsAg on this patient were not available to us.

The Table shows the correlation between hepatitis B infection in donors and patients. Hepatitis B markers sought included HBsAg, anti-HBc and anti-HBs. There was no evidence of infection in any of 22 donors for three patients positive for HBsAg, indicating that hepatitis B infection in these three patients was not attributable to blood transfusion (1980.1, 1984.5, 1984.7). Two donors out of 20 were implicated in the transmission of hepatitis B to two corresponding patients. One (patient 1980.2) showed a very weak positive reaction when the test for HBsAg was repeated but this had been missed during the initial screening procedure. The other donor (patient 1983.2) was repeatedly HBsAg negative and therefore would not have been identified as infective on routine screening. Anti-HBc was the only marker in this donor and was only detected on further investigation following the report of post-transfusion hepatitis. It is of interest that one further donor out of 14 was anti-HBc positive but no evidence of hepatitis B infection was found in the corresponding patient (1983.3).

DISCUSSION

Sensitive methods are now available for the detection of 'healthy carriers' of hepatitis B virus. The radioimmunoassay currently used in the NI Blood Transfusion Service for screening donors is very sensitive and calibrated to detect very low concentrations of hepatitis B virus (HBsAg) particles. The national minimum requirement is two British Standard Units and we are currently able to detect the lower level of one British Standard Unit. The risk of hepatitis B transmission through blood transfusion has, therefore, been minimised in the

TABLE

Patients with post-transfusion hepatitis notified to the NI Blood Transfusion Service, 1980-1984

Year	*Patients*	*Hepatitis B markers in the patient*	*Number of donors implicated*	*Hepatitis B markers in donors*
1980	1	HBsAg	7	Nil
	2	anti-HBc	6	HBsAg in one donor detected by RIA only*
1981	1	Nil	2	Nil
	2	Nil	15	One donor anti-HBs
1982	1	Nil	3	Nil
1983	1	Nil	2	Nil
	2	HBsAg	14	Anti-HBc in one, but negative six months later.
	3	Nil	14	One donor repeatedly anti-HBc.
1984	**1	Nil	6	Nil
	2	Nil	13	Nil
	3	Nil	6	Nil
	4	Nil	2	Nil
	5	HBsAg	8	Nil
	6	Nil	5	Nil
	7	HBsAg	7	Nil
	8	Nil	26	No follow-up: all donation numbers not available.

HBsAg: hepatitis B surface antigen
Anti-HBc: antibody to hepatitis B core antigen
Anti-HBs: antibody to hepatitis B surface antigen
RIA: radioimmunoassay.
*Original testing by less sensitive method. **Follow-up: drug-induced.

past 5 years, but a few cases of post-transfusion hepatitis B will continue to occur due to very low levels of antigen not detectable by radioimmunoassay.[8] Other transmissible agents such as the non-A, non-B agent, cytomegalovirus and Epstein-Barr virus continue to be important in the aetiology of transfusion-associated hepatitis. There are no simple, reliable and cost-effective screening tests available for the detection of blood donors who are capable of transmitting these infections.

In this report, 11 of 16 patients developed post-transfusion hepatitis which was due to causes other than hepatitis B. If a diagnosis of non-A, non-B hepatitis is suspected it is important to identify the donors implicated, in order to prevent further transmission to other patients through future blood donations. In the absence of a screening test,[7] our present policy is empirical exclusion of any donor implicated in two instances of post-transfusion hepatitis. It is to be noted that, in one patient, the hepatitis was subsequently shown to be drug-induced. Non-transfusion-related causes must be considered early in the differential diagnosis.

One donor who was HBsAg negative but anti-HBc positive transmitted hepatitis B to one patient. This is a rare occurrence but is a known possibility.[8, 9, 10] The value and cost-effectiveness of screening every donation for anti-HBc has been debated.[11, 12, 13] In Northern Ireland, the incidence of chronic asymptomatic carriers of hepatitis B is very low: one in 1500 healthy new donors is HBsAg positive. The risk of hepatitis B transmission from donors who are only anti-HBc positive in our region is estimated to be negligible and does not justify the high cost that such screening would entail.

No hepatitis B markers were detected in one patient although a donor involved was repeatedly positive for anti-HBc. The post-transfusion hepatitis in this patient was attributable to non-A, non-B infection. We have been unable to establish a satisfactory duration of follow-up with regard to hepatitis B markers in this case, although there are other reports of non-A, non-B transmission from donors who are positive only for anti-HBc.[14] Blood products including platelet concentrates, fresh frozen plasma, cryoprecipitate and coagulation factor concentrates are known to transmit hepatitis but none of our present series of patients were exposed to these.

The reported incidence of post-transfusion hepatitis shows great variation, the lowest being 2%.[3] There is little doubt that the apparently negligible incidence in Northern Ireland during the years 1980-1983 must be due to lack of notification. It is still uncertain how many cases are missed, and even among a small population like that of Northern Ireland it is difficult to hazard a guess at the true incidence of the disease. Of the non-B infections, non-A, non-B hepatitis is a significant problem particularly in terms of chronic liver damage, despite the mildness of the initial illness. In the absence of tests capable of detecting an infective donor, we must rely on notification of transfusion-acquired infection and retrospective investigation of blood donors for eventual disqualification of implicated donors.

Lack of notification may be attributed to several factors. A significant proportion of non-A, non-B infection is sub-clinical and serum transaminase levels fluctuate independently of clinical illness.[15] Vague symptoms after surgery or anaesthetic may be ignored by the patient and doctor. The prolonged incubation time of hepatitis B and moderate incubation time of non-A, non-B infection sometimes make the correlation between transfusion and clinical illness difficult. Patients are discharged from hospital and the present system of liaison between general practitioners, hospital doctor and the NI Blood Transfusion Service is unsatisfactory. There is a good case for direct notification to the NI Blood Transfusion Service of any cases of suspected transfusion associated-infection.

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Outcome of patients admitted to an acute geriatric medical unit

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SUMMARY

To find out what happens to patients admitted to an acute geriatric medical unit, all admissions during 1982 were reviewed. Demographic features were compared with those of the community served, and rehabilitation, inpatient mortality and mortality in the year following discharge were assessed. Inpatients accounted for 4% of the community aged over 65, and most patients were discharged back to the community. Inpatient mortality was 25% and mortality in the year following discharge was 23%, giving a two year mortality of 42%, which was similar in all age groups. The achievement of high rehabilitation rates was tempered by the considerable mortality rates following discharge.

INTRODUCTION

Population statistics for the past twenty years show a considerable increase in persons aged over 65 years. It is predicted that there will be a further increase by the end of the century, especially in those aged 85 and over.[1] Since there will be an increased demand for acute geriatric beds, it is of value to assess (1) which members of the present community aged over 65 are more likely to be admitted to a geriatric medical unit, (2) the outcome of this hospitalisation and (3) mortality following discharge of such patients. We have therefore reviewed these criteria for all patients discharged from a geriatric medical unit over a one-year period with particular reference to patient age.

METHODS

The 72-bed geriatric medical unit at Altnagelvin Hospital is the only one in the Londonderry, Limavady and Strabane district and accepts patients aged over 65 years. Patients are referred to the consultants from general practitioners or from other hospital wards, (9 am – 5 pm, Monday to Friday). Acute geriatric admissions outside these hours go initially to acute medical beds and, if rehabilitation is considered necessary, they are transferred to the geriatric medical unit. There are 187 long-term care beds elsewhere in the district, but these are not considered in this report.

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In 1982 the turnover was 531 patients who had 613 admissions, 65 having multiple admissions. Data on each patient was obtained from the Hospital Activity Analysis, records of weekly ward meetings and from inpatient records. All patients who died in the first year following discharge were identified by notification from the Registrar of Deaths. Population statistics were derived from the 1981 Population Census and from the report of the Registrar of Deaths. Data were stored and sorted on a simple microcomputer database ('Masterfile, BBC B Microcomputer'). Statistical significance was tested using the chi square test.

RESULTS

The demographic features of the patients and their one-year survival rates following discharge were compared with those of the community population and its mortality data. Placement of patients on discharge from the Unit was assessed as a measure of rehabilitation.

(1) *Demographic features*

The district population was approximately 157,000 with 14,500 aged over 65 years. Table I illustrates the proportion of various age groups in the community admitted to the Unit. These proportions were similar for males and females. The marital status of patients was compared with that of the district population (Table II). Since the proportions were different for males and females, these are shown separately. Marital status was not recorded for 44 patients.

TABLE I

Number of persons in 5 year age groups in the Geriatric Medical Unit and in the district

Age	*District population*	*Geriatric Medical Unit (%)*
65 – 69	5309	86 (1.6)
70 – 74	4261	121 (2.8)
75 – 79	2575	119 (5.0)
80 – 84	1491	135 (9.0)
85 – 89	687	50 (7.3)
90 – 94	178	16 (9.0)
95 – 99	71	4 (5.6)

TABLE II

Marital status of district and Geriatric Medical Unit populations

	MALE		*FEMALE*	
Marital status	*District*	*Geriatric Medical Unit (%)*	*District*	*Geriatric Medical Unit (%)*
Married	3456	82 (2.4)	2473	53 (2.1)
Single	1206	59 (4.9)	1944	79 (4.1)
Widowed	1018	56 (5.5)	3492	150 (4.3)

(2) *Rehabilitation*

Placement of patients after discharge was used as a measure of rehabilitation, and was classified as (1) in the community, (2) in residential accommodation and (3) in long-term care wards. Table III shows placement on discharge in relation to placement prior to admission for all 613 admissions. Using these criteria, 66 patients (14% of discharges) did not return to their original level of placement but went instead to a setting of higher dependency. Patients aged over 75 years (50 patients) and females (46 patients) were less likely to be rehabilitated ($p < 0.05$).

TABLE III

Placement on discharge in relation to residence prior to admission

Residence prior to admission			*Placement on discharge*		
Home	535	(87%)	Home	353	(66%)
			Residential	12	(2%)
			Long-term care	46	(9%)
			Deceased	120	(22%)
Residential	73	(12%)	Residential	45	(62%)
			Long-term care	8	(11%)
			Deceased	20	(27%)
Long-term care	4	(1%)	Long-term care	1	(25%)
			Deceased	3	(75%)

(3) *Mortality following discharge*

Date of death following discharge, and hence survival at one year, was determined for all patients. In the event of a patient with multiple admissions, only the first admission in the year was used for this purpose. Of the 531 patients, 132 (25%) died in hospital and 399 were discharged. A further 92 died within one year giving a post-discharge one-year mortality of 23%. The cumulative mortality from admission to one year after discharge was 42%. In Table IV the one-year mortality for various age groups is compared with the community mortality for the same age groups. The similar mortality rates for the patients in various age groups contrast with the lower community mortality in the 65 – 75-year-olds as shown in the last column of the Table. One-year mortality following discharge was unrelated to sex, marital status or duration of stay in the Unit.

TABLE IV

Age-specific mortality for patients discharged from the Geriatric Medical Unit compared with community mortality

Age	*Patients discharged*	*Deceased at 1 year*	*Patient mortality rate/100*	*Community mortality rate/100*	*Ratio patient/community mortality rates*
65 – 74	158	38	24.0	4.1	5.8
75 – 84	184	41	22.3	9.7	2.3
85 +	57	13	23.2	20.4	1.1
TOTAL	399	92	23.0	6.8	3.4

DISCUSSION

Critical assessment of the outcome of patients admitted to a geriatric medical unit should include identification of people likely to be admitted, appraisal of rehabilitation success and of associated mortality.

Comparison of the geriatric medical unit population with that of the community revealed that 4% of the district geriatric population were inpatients during the year. Patients aged 65–75 years were under-represented, as noted by others.[2] This may be explained by the number of these patients admitted to the general medical wards,[3] and by increasing morbidity with advancing age. Married persons were also less likely to be admitted than widowed or single persons and were less likely to require residential or long-term care. This 'protective effect' of marriage has been described previously[4] and must reflect the importance of social support in coping with illness at home without resort to hospital care, rather than a true difference in health.

The role of the Unit in rehabilitation was confirmed by the high proportion of patients returning to their original placement following discharge. Whereas a previous report suggested a poor outlook for patients admitted from residential homes compared with those admitted from the community,[5] in this study rehabilitation outcome and mortality rates were similar.

The inpatient mortality rate of 25% was similar to that published for other units, 19%–33%,[2, 6, 7] although direct comparison is difficult. Mortality was unrelated to age or sex in contrast to a previous report.[8] Mortality following discharge was higher than that of a similar age group in the community, being increased six-fold for persons aged under 75 years and double for those aged over 75 years. Indeed, for those aged over 85 years the mortality rate was similar to that in the community. This difference in mortality rates is due to ex-patients having similar mortality rates in all age groups in contrast to the increasing mortality with age found in the community. This pattern is probably due to a combination of the management at home of very elderly persons with a terminal illness and referral of patients under 75 years to other acute services.

The addition of this information on deaths in the year following discharge to inpatient mortality reveals that almost half of all patients admitted to the geriatric medical unit die within this period regardless of age. Patients discharged from this Unit represent an 'at risk' group who may merit special surveillance by their general practitioners.

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General practitioner workload with 2,000 patients

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SUMMARY

This study was designed to investigate the relationship between general practice workload, the number of partners in the practice, and the use of health centre premises. Thirty general practitioners in twelve randomly selected practices (each with a list size of 2,000 patients per doctor) agreed to record a week's work on pre-printed forms. Information was gathered on content of care in the surgery, number of non-surgery and indirect contacts and time spent on work activities. Content of care was influenced by whether or not the doctors were based in a health centre, rather on how many partners they had. Conversely the numbers of non-surgery and indirect contacts and the time spent on all work activities were more affected by the number of partners. Two factors — consultation rate and the rate at which doctors initiate consultations — were found to be independent of either of the two variables considered.

INTRODUCTION

In his study of list sizes in general practice, Butler[1] cites a number of official bodies who have regarded list size per doctor in the range 2,000 to 2,500 as optimum.[2, 3, 4, 5, 6] He also states that doctors themselves regard an average list of about 2,000 to 2,100 as ideal. But what does a list of 2,000 patients actually entail in terms of workload for the general practitioner? And to what extent are the various components of workload affected by practice characteristics apart from list size?

In Northern Ireland, particularly in the Belfast conurbation, two features of general practice have recently become established — the building of health centres, and the increasing number of new general practitioner principals which has resulted in a fall in the average list size. This fall in the average number of patients per doctor has provoked discussion in many quarters. Some see a threat to income; others see potential for preventive and anticipatory care. To date, the target average list locally remains at 2,000 per principal.

We therefore proposed to carry out what should be considered a pilot study on existing workload among a representative sample of general practitioners from

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the Greater Belfast area. We wished to describe how doctors with varying numbers of partners, practising both within and outside health centres — but all with the 'ideal' list size of about 2,000 patients per doctor — manage their patients. Our hypothesis was that, despite the constant list size per doctor, there would still be considerable variation in workload between the practices. We were interested to see to what extent such variation was influenced by the number of partners in the practice, or affiliation of the practice to a health centre.

METHODS

All the general practitioners in one-, two-, three- or four-man practices within a 15-mile radius of central Belfast, and with a list size of approximately 2,000 patients (range 1,882-2,153), were identified with the help of the Central Services Agency. Thirty suitable practices were identified. From these 30 practices, 12 were selected to give three four-man, three three-man, three two-man and three single-handed study practices. The practices were based either in a health centre or in private premises.

The doctors were allocated randomly to one of two observation periods (1 October to 16 December, or 9 January to 30 March), and asked to fill in a set of forms for one complete working week during this period. The study weeks were selected randomly but where a doctor felt that his or her workload would be atypical during that week (due, for example, to their own or their colleagues' holidays) a replacement week was selected. Three data collection forms were used (copies available from the authors). The first obtained information on each patient seen in the surgery, including sex and date of birth and whether the consultation had been doctor- or patient-initiated. The doctor then indicated whether the patients had received a local, system or general examination, whether they had been sent for an X-ray or laboratory test, given a prescription or advice and information and whether they had been referred to other internal (treatment room, health visitor, etc) or external (hospital outpatient department) agencies. The doctor indicated what he considered to be the primary reason for the patient coming to the surgery, but it was not necessary to give a diagnosis. The contents of this category were then coded using the RCGP and Office of Population Censuses and Surveys classification of morbidity.[7] The second form enabled the non-surgery aspects of a general practitioner's workload — within working hours — to be quantified. The doctor also recorded the number of new and return home visits made, the number of indirect consultations dealt with and the number of repeat prescriptions issued during working hours. The third form measured the amount of time spent on various work activities by each doctor. For each day in the study week the doctor was asked to record how long was spent in surgery, on home visits (both travelling and with the patient), clinics and sessions outside the practice, indirect consultations and administration and on any other work-related activity (such as reading journals).

RESULTS

Practice characteristics

Table I shows the characteristics of the 12 study practices, together with equivalent figures for the Greater Belfast area from which the practices were drawn. The selected practices showed a similar distribution of the various facilities studied.

TABLE I

Practice characteristics

	Practices in Greater Belfast area		*Study practices*	
	number	*%*	*number*	*%*
Total practices	124	100	12	100
Health centre	53	43	4	33
Attached treatment room nurse	81	65	9	75
Attached district nurse	74	60	8	67
Attached health visitor	95	76	9	75
Open access to laboratory	124	100	12	100
Open access to X-ray facilities				
— Chest clinic only	99	80	6	50
— Chest and skeletal	13	10	4	33
— Chest, skeletal and contrast media	12	10	2	17

Surgery consultations

Individual doctors' results were grouped and analysed in relation to number of partners and whether or not they were based in a health centre. The morbidity distribution of patients consulting in the surgery showed no significant difference either between the four partnership sizes (i.e. one- to four-man) or between health centre and non-health centre doctors. This enabled a valid comparison of their treatment by various groups of doctors. All figures refer to a period of five working days. Consultation rate was independent of the number of partners ($p = 0.446$, Kruskal-Wallis one way analysis of variance[8]) and of the practice premises, although the figures show a higher rate in the three- and four-man practices. The overall mean of 46 patients seen/1,000 registered/week was lower than that found by two earlier Northern Ireland studies,[9,10] but is in keeping with rates observed throughout the UK in the past 20 years.

We did not measure the exact amount of time spent with each patient seen in the surgery but obtained an approximation for comparative purposes by dividing the total time spent in the surgery during the study week by the total number of patients seen. The mean consultation rate (per 1,000 patients per week) ranged from 33 for the one-man practices to 57 for the three-man practices, with no difference between health centre and non-health centre practices. The mean time spent with patients varied widely from six to 27 minutes, and between 5% and 29% of the consultations were initiated by the doctor, with no significant differences for the various types of practice.

There was no difference between the various partnership groups in the rate of general examination (Table II) but there were very significant differences in the use of local and system examinations. The situation was reversed in the case of the health centre and non-health centre doctors, with only the proportion of patients receiving a general examination showing significant variation. The single-handed doctors' lower mean usage of the laboratory was influenced by one out of the four doctors requesting no tests during the study week. The mean value for

TABLE II

Patient care: percentage of patients who receive a service

Service	Range for all doctors	One-man	Two-man	Three-man	Four-man	p*	Health centre doctors	Non-health centre doctors	p*
Local examination	15 – 73	36	49	28	35	0.000	34	36	NS
System examination	7 – 61	19	25	44	34	0.000	37	32	NS
General examination	0 – 32	9	9	10	12	NS	13	9	0.001
Lab test	0 – 25	4	8	11	8	0.012	11	7	0.003
Prescription	36 – 91	63	70	68	64	NS	59	69	0.000
Advice or information	9 – 87	29	36	34	33	0.015	41	30	0.000
External referral	3 – 26	10	10	9	11	NS	9	11	NS
Item of service**	2 – 26	7	7	9	9	NS	10	7	0.010

*A p value of <0.01 from the Chi squared test is taken to show a significant result.
NS: not significant.

**Refers to activities such as immunisations, antenatal care, cervical smears, for which the doctor earns a fee.

the remaining single-handed doctors and the other doctors was similar. Comparison of the use of X-rays and internal referral (i.e. referral to workers attached to or working in association with the practice) proved impossible due to the varied nature of the circumstances pertaining to different practices. The rate of prescribing, although similar for all partnership sizes, was significantly higher among non-health centre doctors. The giving of advice or information, on the other hand, was used to a significantly greater extent by the health centre doctors. The rate of external referral (e.g. to hospital out-patient departments) was constant, at around 10% for all the doctor groups studied.

The mean number of new home visits during working hours varied very little (15 to 18) between the different types of practice (Table III), although the range for all doctors was between three and 39 visits per week. The mean number of doctor-initiated re-visits varied from four to six per week, except in the single-handed practices where a mean of only one was found. The range for all doctors

TABLE III

Mean values for non-surgery and indirect contacts during working hours

	Range for all doctors	One-man	Two-man	Three-man	Four-man	Health centre	Non-health centre
New home visits	3 – 39	16	18	18	16	15	18
Re-visits*	0 – 22	1	6	4	4	4	4
Indirect consultations**	3 – 99	26	29	44	22	33	27
Repeat prescriptions	0 – 258	49	119	98	106	63	123

*Visits which the doctor decides to make of his own accord rather than at the request of the patient.
**Consultations taking place via phone, letter or a third party.

was 0 to 22 re-visits per week. Indirect consultations were very variable (range three to 99 per week) but the mean values showed no difference. The lowest mean number of repeat prescriptions — 49 per week — was in the single-handed practices, doctors in the four-man practices issuing 106 per week. The non-health centre doctors issued 123 and the health centre doctors 63 per week.

The mean time spent by doctors on various work-related activities during the study week is shown in Table IV. No report is made on out-of-hours work because of the relatively short study period and the fact that some doctors, due to their rota, were not on call during their study week. The longest mean time spent on home visits, both in travel and with the patients, was for the two-man practices, and for the non-health centre practices. The two-man practices spent the least amount of time at clinics or other sessional work, and the most time in medical reading and with pharmaceutical company representatives. Both one- and two-man practices, and the non-health centre doctors, spent longer on administrative work than the others. The doctors in two-man practices overall spent the longest time in the surgery and had the longest overall workload (40 hours). There was no difference between the health centre and non-health centre practices for these measurements.

TABLE IV

Mean times spent on work activities (excluding 'on call', out of hours) during study week (hours)

	Range for all doctors	*One-man*	*Two-man*	*Three-man*	*Four-man*	*Health centre*	*Non-health centre*
Home visits — travel	0.6 – 10.0	2.6	4.4	3.2	2.2	2.6	3.2
Home visits with patient	0.3 – 13.0	4.3	6.5	4.4	3.5	3.4	5.0
Clinic or sessions	0.0 – 11.0	3.0	1.1	2.3	2.7	2.1	2.4
Indirect consultations	0.2 – 4.6	2.1	1.5	1.5	1.3	1.6	1.4
Administration	0.2 – 7.6	3.3	3.2	1.9	2.5	1.9	3.1
Others (medical reading and pharmaceutical representatives, etc)	0.0 – 13.9	1.3	3.6	1.3	2.2	1.9	2.3
Hours in surgery	6.0 – 20.0	13	20	17	15	17	16
Total (hours)		29.6	40.3	31.6	29.4	30.5	33.4

DISCUSSION

Several questions arise in this type of general practice study. How comparable are the practices? How representative is the sample of general practitioners being studied? How typical is the period (in our case one week) chosen for recording? How reliable are the observations made by the general practitioners?

Neither the age/sex distribution of the practices selected nor the morbidity recording during the study weeks differed significantly. Social class distribution in the practices, all drawn from the same geographical area, was unlikely to have differed significantly. Using the Belfast conurbation as the study area, a random sample of general practitioners, each looking after 2,000 patients, was chosen, representing practices varying in size from one- to four-man. The study was limited to these partnership and list sizes because, as recently as July 1983, 85% of the principals in Northern Ireland were in one- to four-man practices and the

average list size was 1,951. The percentage of study doctors who qualified prior to the 1965 General Practice Charter (which brought about a marked change in general practice conditions and thus, possibly, behaviour) is almost identical to the figure for Northern Ireland. The study practices were also found to be broadly representative of the local area as regards facilities.

Despite the small size of the study, the overall patient age/sex and morbidity distribution was the same as found for the United Kingdom by the very large National Morbidity Study[7] (these data are available from the authors). Equally, there was no significant difference in the distribution of the same characteristics among patients seen by the various sizes of partnership or by doctors in health centres or other types of practice premises. Doctors were asked to pick a week during the study period which they thought would be typical, avoiding public and colleagues' holidays, etc. Some features of workload (e.g. surgery attendances) vary much less than house call rates. Obviously the general practitioners knew they would be 'observed' but they had no reason for distorting their figures and their anonymity was guaranteed.

Doctor-initiated consultations are substantially under the doctor's own control, thus allowing him to exert influence over his own workload. The practices studied here, though not each individual general practitioner, showed little variation in the extent to which they are influencing their workload by this means. The single-handed doctors chose not to examine 36% of their surgery patients — almost double the figure for the other partnership sizes. The single-handed general practitioners reported giving fewer of their patients advice or information during a consultation. Yet the single-handed general practitioners spent longer with each patient seen than did their three- and four-man colleagues. They also showed no significant difference in their rate of prescribing or external referral. The situation is far from clear-cut, and it is important to remember when considering aspects of workload such as investigations, prescribing and referral that at least some of such doctor activity may be a means of coping (e.g. ending a consultation by prescribing rather than continuing with more appropriate discussion), rather than a reflection of real workload. The health centre and non-health centre doctors showed significant variation in the use of general examinations. The formers' higher usage of general examination (and the fact that health centre doctors chose not to examine only 16% of their surgery patients compared with 23% for the non-health centre doctors) may well be influenced by available facilities and the full-time presence of nursing staff, rather than by workload. The higher rate of laboratory test usage by health centre general practitioners may well also have been similarly influenced.

The health centre doctors showed a higher rate of 'item of service' work (cervical smears, vaccinations, etc) than their non-health centre colleagues. But the overall mean for all general practitioners for 'item of service' work was only 8%, which seems a low figure when both the potential for earning extra income and the opportunity for preventive medicine are constantly emphasised. It would appear that while certain aspects of surgery workload — consultation rate, external referral, doctor-initiated consultations — were unaffected by either the number of partners in the practice or whether the practice was based in a health centre, other aspects were affected by one or both of these practice characteristics. The mean time spent with each patient, and the rate of local and system examination only varied with the number of partners, while the rate of general examination, prescribing and 'item of service' work was influenced solely by whether or not the

general practitioners were in a health centre. The giving of advice or information was found to be influenced by both practice premises and partner number.

We looked at the ratio of new visits to re-visits for each study group on the premise that a significantly higher ratio could indicate that the general practitioners concerned are reducing their workload due to the lower number of re-visits they make. The ratio for the single-handed doctors (16:1) is much higher than the other practices' figures (3:1, 4.5:1 and 4:1), but, due to the very low numbers of re-visits overall, we are wary of attaching too much importance to this finding. Considering the very large range for indirect consultations (3 – 99), all the study groups show remarkably similar usage. The single-handed general practitioners' use of repeat prescriptions was actually lower than that of the other three partnership sizes. The large difference in the number of repeat prescriptions issued by health centre and non-health centre doctors can only be partially explained by the higher proportion of elderly patients in the latters' practices which might be expected to account for a higher rate of repeats. The finding that the two-man practices' mean for time spent travelling to patients is much higher than the other partnership sizes' means can probably be attributed to the fact that two of the three two-man practices (both non-health centre) were in less densely populated areas. But distance cannot explain why the two-man general practitioners spent on average two hours per week longer with their patients at home. They were not seeing more patients at home but spending longer with each patient seen. The non-health centre doctors each devoted about 20 minutes per day more to their patients at home, while the health centre doctors spent around eight minutes per day more with their surgery patients. The non-health centre general practitioners would still seem to be following the more 'old-fashioned' way of operating in general practice, i.e. spending longer with patients at home than in the surgery. Possibly in consequence, they averaged 190 minutes a week (approximately half-an-hour per day) more time 'at work' than the health centre doctors.

The doctors studied spent a very varied amount of time on clinics and sessions. The amount of time spent on such work was found to decrease with the decreasing number of partners (the single-handed practices' figure is biased by one doctor who was involved with the Schools' Medical Service). The longer average time spent on administration by general practitioners in smaller practices and in non-health centre practices might well be expected. Doctors in a larger practice have more colleagues available to share the workload which may well more than balance the extra administrative burden, and in a health centre extra clerical help may be available to assist with administrative tasks. Overall, both the numbers of 'non-surgery' activities and the time spent on various surgery and non-surgery activities (with the exception of time spent travelling to patients on home visits) are influenced to a far greater extent by the number of partners than by whether or not the practice is in a health centre.

In this study several aspects of doctor workload involved with 2,000 patients were examined. Only time spent within conventional working hours was noted (range: 29.6 – 40.3 hours weekly). However, for most full-time doctors, when the weekly average out-of-hours 'on call' time was added, total working time per week was increased by 50%. But workload is not just 'items of service' per unit time. As can be seen, even when aspects such as patient number are held constant, patterns of provision of care can vary while facilities seem to exert a significant, though selective, influence. These areas of primary care merit further, more extensive scrutiny.

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Andrew Malcolm and C D Purdon
Pioneers of occupational medicine in Belfast

James A Smiley

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Fifty-one years ago half-a-dozen doctors employed in industry met in London and formed an association — now the Society of Occupational Medicine. Ten years ago the Royal College of Physicians in Ireland brought into existence the first faculty of occupational medicine in Europe followed two years later by the London College. The first textbook on occupational diseases was published in 1700 in Italy by Ramazzini[1] but it was not until 1832 that Thackrah[2] in Leeds distilled his vast experience and knowledge in a series of fascinating chapters on a variety of conditions attributable to occupations and for which he recommended preventive measures. These men and others gained their experience from observation in the ordinary practice of medicine. It was only after 1802 that doctors had any formal or legal responsibilities in the sphere of industry. After the passing in that year of the Health and Morals of Apprentices Act (which we now regard as the first Factory Act), the need for the advice of doctors became apparent, for one of its provisions was that no child under the age of ten years might be employed in the mills. At that time, because of increasing mechanisation which allowed operations to fall within the physical capacity of children, there was much adult unemployment. Consequently many parents driven by poverty overstated the age of their little ones in order that the family might escape the consequences of the harsh Poor Law of the time. Baptismal certificates were not always available and often did not refer to the youngster presented for employment. Registration of births and deaths did not come into operation until 1837 (later in Ireland) so some method of ascertaining the age and enforcing the Act had to be found. The 1802 Act had established the principle of factory inspection — resisted by the employers holding the *laissez-faire* philosophy of the times, and initially the inspectors were appointed on a local basis by the district magistrates.

These inspectors (of whom one had to be a magistrate and another the vicar or rector of the parish) invited local medical men to certify the apparent age of the children. For reasons which do us no credit the system fell into disrepute.[3] In 1819 another Act was passed which provided for the appointment of four paid inspectors with responsibility for Great Britain and Ireland, but it reduced the childhood age to nine. These inspectors were empowered to appoint medical practitioners to certify the apparent age of the child. Two problems thus arose — whom to appoint, for the status of qualified and registered practitioners did not become precise until after the Medical Act 1858, and how objectively to determine the age of children. On both these topics a correspondence of great virulence appeared in the medical press. Apothecaries, surgeons and physicians

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all claimed the appointments, and a multiplicity of opinions on the determination of age by weight, height, appearance and other evidence enlivened the journals of the day.

The inspectors however, were men of learning and probity — the man whose bailiwick included the north of Ireland (which at that time was north of a line from Dublin to Galway) was Leonard Horner. He was of a distinguished Scottish family and a polymath with interests in chemistry, geology and other natural sciences, subsequently becoming a Fellow of the Royal Society and one of the founders of London University. Another inspector was Robert Saunders, who, although non-medical, entered the controversy by publishing a monograph on the usefulness of the dentition in determining age.[4] Horner concurred with his conclusions — 'I am becoming rather knowing in that I have looked into 500 little mouths lately. I suppose it has got wind, for when the doctors and I go round the mills and call any to us who look too young . . . they sometimes come running with their mouths open and turn up their little heads without being told'.[5] Thus was formed the certifying factory surgeon system by which medical men assumed a formal degree of responsibility for some of the human problems arising in industry.

It was for one of these appointments that in 1849 Dr Andrew G Malcolm (1818-1856) unsuccessfully applied to James Stuart, who in a reorganisation of districts had succeeded Horner. That the position of Certifying Factory Surgeon had by this time achieved some esteem in the minds of the profession may be gauged by the fact that in 1854 when the post again became vacant 'six of the eight members of the honorary medical staff of the General Hospital applied, Malcolm himself being the senior attending physician'.[6] Calwell suggests that on this occasion Malcolm was successful and there appears to be confirmation of this in his lecture to the British Association in 1855 when in reference to his researches into the influence of factory life on the health of the operatives he spoke of 'experience in my public appointment . . . I personally inspected the workers at the factories . . . '[7]

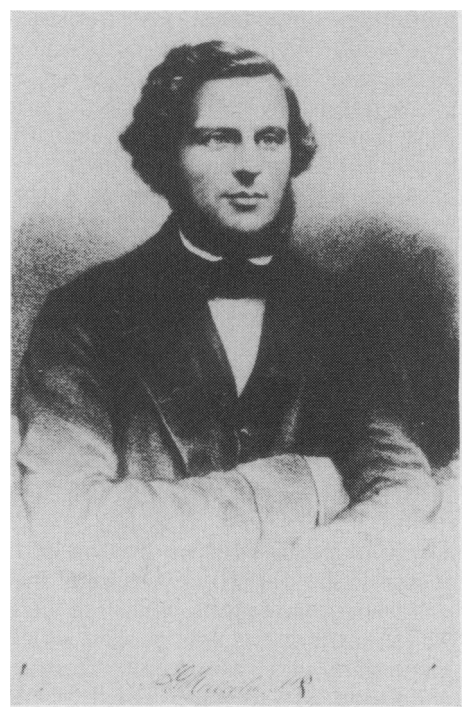

Fig 1. Andrew G Malcolm (1818-1856) (from *Andrew Malcolm of Belfast,* by H G Calwell, reproduced with the author's permission).

By the eighteenth century Europe had well and truly cast off the bonds of mediaevalism and by the beginning of the nineteenth century the great period of differential diagnosis and clinical description of syndromes was flourishing. The application of statistical methods to medical data as distinguished from vital statistics had been made by Pierre-Charles Alexandre Louis[8] in his works on tuberculosis (1825) and typhoid (1829), and in 1835 his statistical proof that

blood letting was of little value in the treatment of pneumonia, heralded the dawn of medical ststistics. Although Belfast at the time was the scene of growing sectarian tensions and great industrial and social upheavals, there was also throughout the country a great humanitarian movement and Calwell has graphically described Malcolm's leading part in it. In the midst of all his frenetic activity, Malcolm led the profession not only by the introduction of new teaching methods but by the adoption of precise methods of enquiry and rational analysis of the material obtained by assiduous effort rather than by anecdotal evidence. He was part of the intellectual activity of the time and a leader amongst his peers.

Living at 29 York Street, between the hospital and Mulholland's Mill, it was almost inevitable that Malcolm's interest in the effects of flax dust on 'operatives' would be stimulated, and his modern approach to medical enquiry is shown in the reading of his paper — not to a medical group but to a meeting of the statistical section of the British Association in Glasgow in 1855.[9] With his friend Professor Hodges he examined microscopically the filaments of flax, noted in detail their structure and estimated the content of the ash. He described in detail the various processes through which flax was passed, the relative amounts of dust generated in each department, the temperature and humidity of the workrooms and the working position of each of the operatives. He thus demonstrated the *sine qua non* of a good occupational health physician — he knew the processes and the environment in which they were carried out. He had travelled to Leeds — then the centre of the linen trade in England — visited the great Marshall's Mills (the ventilation system of which he was critical) and, because of Thackrah's association with the trade there, it is probable that Malcolm met him and had read his book.

The evidence for Malcolm's paper came from three different sources — the returns of attendances and the causes thereof from six dispensary districts in Belfast for the years 1852-55 as well as the occupations of the patients, similar returns from the General Hospital between 1848 and 1854, and the visitation of '2,078 workers to whom queries were submitted and indiscriminately visited at their homes'.[10] One could not do justice to the assiduity with which these researches were pursued nor to the meticulous analysis of the data without reading the actual paper. Not only did he identify the pernicious effects of flax dust and demonstrate that the amount of disability was related to the amount of dust in the workroom but he made recommendations designed to improve the working environment. In these he anticipated at least two of Legge's axioms[11, 12] when he advocated the extraction of dust at its site of origin and wrote that 'the freest change of atmosphere should not be subject to the control or whim of the operator'.

While Malcolm was engaged in his multifarious activities, Dr Thomas Henry Purdon (1806-1886) was medical attendant at the Institution set up by the Belfast Charitable Society. (He was the second of a succession of the Purdon family connected with the Society from about 1800 until the death of Dr E B Purdon in 1947). In 1846-47 this institution agreed to accept non-infectious cases from the General Hospital during an outbreak of typhus on the emigrant ship Swatara[13] and it was arranged that those transferred patients would be attended by the hospital physicians, of whom Malcolm was one. As a result Dr Charles Delacherois Purdon (1818-1882) who had succeeded his brother as medical attendant at the Institution, for a period at least, would have worked with Malcolm. At that time linen manufacture was a domestic industry and the Board of the Society had made provision for spinning to be carried out on their premises.

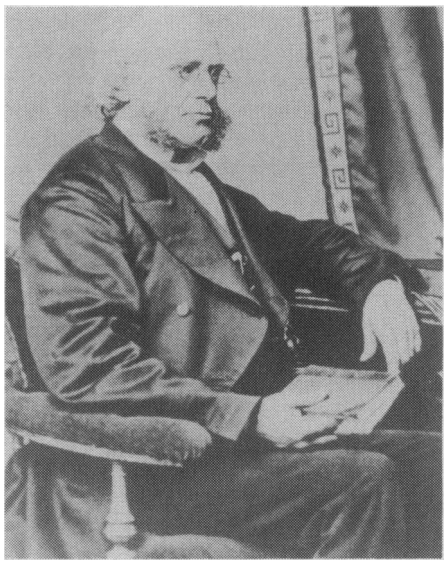

Fig 2. Charles D Purdon (1818-1882) (from the portrait as President of the Ulster Medical Society 1874).

Strain[14] writes 'as early as May 1775 the spinning of flax by inmates must have reached considerable proportions'. It may be that in his association with Malcolm and his experience in the Institution, Purdon's interest in the textile trade was excited. He later became a certifying factory surgeon, and in 1873 read a paper to the annual meeting of the Certifying Factory Surgeons in Leeds — 'The mortality of flax mill and factory workers'.[15] He set out a series of tables 'classified according to age, employment, whether flax mill and factory classes, artizan and labouring classes, gentry and mercantile classes and nature of diseases'. He showed the excess of mortality from respiratory disease among the operatives, especially those in the preparing rooms. He observed 'the reason that the machine boys appear to suffer so little is that when they become "poucey" (i.e. asthmatic) caused by flax dust, numbers of them leave the mills and go to other trades' ('poucey' is a degradation of the French word for dust). His description of the paroxysms of dyspnoea and coughing show how distressing they must have been.

But it was not only with respiratory disease that Purdon was concerned — he attributed many accidents to vertigo and fainting resulting in falls into machinery. He noted a number of common conditions — anaemia, oedema of the ankles, varicose veins and others. His description of mill fever is precise: 'This comes on when they are a few days engaged at work. The symptoms are rigors, nausea and vomiting, speedily followed by pain in the head, thirst, heat of the skin etc. This state continues from two to eight days, when the disease subsides of itself'. Although Purdon's observations are concerned mainly with the respiratory system, they were not confined to it. He draws attention to 'a peculiar eruption which attacks the uncovered parts of the body, this I call "lichen". I have never seen an adult affected with it'. (The Purdon family were all interested in diseases of the skin and C D Purdon in 1865 founded a skin hospital which is now an integral part of the Royal Victoria Hospital).[16] Not content with drawing attention to the results of his enquiries, Purdon proceeded to propose a series of measures which in his view would 'mitigate the mortality'. These included modifications of the half-time system, — selection of the more robust and older children for the dustier jobs, thorough ventilation, the compulsory use of respirators and the 'quarterly inspection', not only of the children but also of the lodging houses.

Two years later in Edinburgh, Purdon was discussing 'The longevity of flax mill and factory operatives'.[17] In this paper he thanks the proprietors for their co-operation in his researches and demonstrated that in the country mills the longevity of workers is greater and their ability to work 'longer' than in town mills. In 1877 he published *The sanitary state of the Belfast factory district 1864-73.*[18] This communication uses much of the previous material and describes in much

more graphic detail the appalling conditions not only in the mills but in the homes of the 'working classes'. He mentions that recruiting sergeants were forbidden by army surgeons to enlist men who had been employed as dressers because of their affected lungs. What is perhaps of most interest to us as clinicians is his observation about spinners: 'On each Monday morning after being in for a short time, many of them become so faint and giddy that they are obliged to go out into the lobbies in order to recover themselves'. This appears to be the earliest report of the Monday morning syndrome of byssinosis.

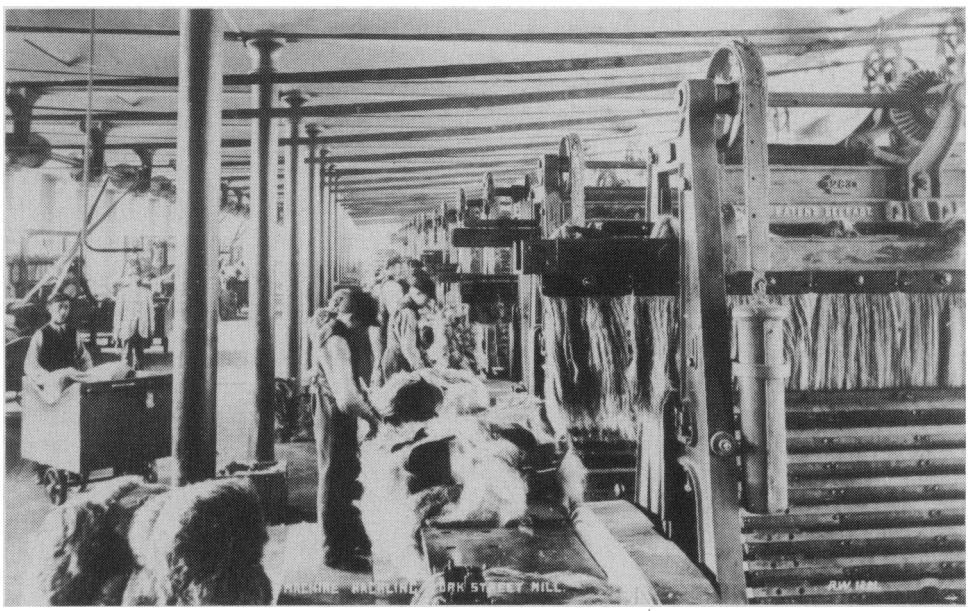

Fig 3. The hackling room, York Street Mill, early 20th century.
(*reproduced by permission of the Ulster Museum*).

Not only is this paper informative and descriptive of working conditions and the results of working in them, it has attached a number of letters and reports from colleagues and others which indicate the assiduity with which he conducted his enquiries. There is a letter from an employer which concludes: 'We consider that visits of a medical man to all manufacturers like this, is most decidedly to the advantage of both workers and employers'. There is a report from Professor Hodges who recommended a different dressing for the yarn which would dry out at a lower temperature. A section on the early decay of permanent teeth, improper and insufficient nourishment (bread and tea), the use of opium, chewing and smoking tobacco, drunkenness and the causes thereof, follow. A reference is made about the mode of life and the effects of a change of diet as well as the use and abuse of athletic pursuits.

In an appendix Dr D Hamilton, a certifying factory surgeon in Cookstown, gives graphic and horrifying descriptions of the mutilating and often fatal accidents caused by the horizontal rollers used for breaking the flax straw prior to scutching. He refers to intemperance — 'the farmers often bringing whiskey with them to treat the workers and scutchers — the latter being proverbial for hard drinking, as the dust and close atmosphere induce thirst'. Appended too is a condemnation of

the housing of the poor with detailed and precise recommendations by Dr Robert H Newett, the certifying factory surgeon in Ligoniel. These predecessors of ours were dedicated, hard-working, observant and caring men to whom not enough credit is given for their work in ameliorating the effects of the factory system.

C D Purdon died in 1882 aged 64 after two days' illness. His obituary notice in the British Medical Journal occupies only 14 lines. His motivation, like Malcolm's, was that not only of the scientific researcher but also of the compassionate physician. He was succeeded as Certifying Factory Surgeon by his son H S Purdon whose classic account of the linen trade and its processes is recorded in Sir Thomas Oliver's massive work *Dangerous trades.*[19] His interest in respiratory disease was manifest in his appointment as one of the original physicians to the first specialist chest hospital in Belfast — the Forster Green Hospital. As if this were not sufficient outlet for his energies, in 1875 he published a treatise on cutaneous medicine and diseases of the skin and for some years was editor of the *Journal of Cutaneous Medicine.*

Malthus had enunciated his theory of population in 1798 which was held to justify the *laissez-faire* policies of later Victorian Britain. Darwin propounded his theories in 1859 which were interpreted by many as 'every man for himself and the devil take the hindmost'. In spite of the prevailing philosophies of the times, we can look back with pride to many of our forbears. As a result of the interest, the compassion and the indefatigable energy of that mid-Victorian generation of 'medical gentlemen' in all parts of the kingdom, the traditions to which each member of our profession is heir, were established. In that profession, occupational medicine is a vocation which beckons to it technically good doctors, generous in their sympathies, liberal in their sentiments and courageous when, as sometimes happens, they are misunderstood by those whom they serve.

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Malone Place Hospital (1860 – 1981)

(THE BELFAST MIDNIGHT MISSION)

A note by Dr H G Calwell †

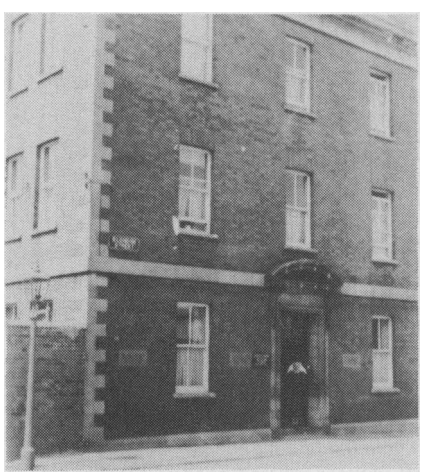
The archive of local medical history continues to grow, which is especially important at a time when so many hospitals are being closed or are losing their identity by being used for the treatment of other diseases than their founders intended. A recent example is Malone Place Hospital, Belfast. A brief history (1981) has been published. The author is Renée E Pelan.

The story begins in 1860 when some Belfast ladies, being concerned about women and girls who frequented the public houses in Belfast, purchased four houses in Malone Place. The ladies went out at midnight and invited the women to the 'Belfast Midnight Mission' for the night. The homeless and strangers were helped to find homes and jobs. After 40 years, the plight of unmarried mothers was met by setting aside a room in the Home for confinements, and by 1903 there were 10 births, and in 1944 there were 116. Homes were found for the babies, some by adoption and some by fostering. A trained nurse and midwife had been appointed when the maternity work began.

In 1922 it was decided to erect new premises, and an appeal for funds (estimated £8,000) was made by the President, the Duchess of Abercorn. The site and five adjoining houses were purchased, and the new hospital was opened in 1926. It was known as 'The Belfast Midnight Mission Rescue and Maternity Home'.

The Northern Ireland Hospitals Authority purchased the complete hospital in 1949. Before this time the home had been under the supervision of the Belfast City Council and it received a grant from the Public Health Committee towards its work. It was non-denominational and open to all ministers and Christian workers. One of the Mission's tasks was to provide legal assistance and advice to help unmarried mothers to compel putative fathers to contribute towards the maintenance of children and the expense of maternity care.

During the early years Dr Bell was the medical officer; she was succeeded in 1926 by Dr Elizabeth Robb. Ante-natal care was undertaken, and three rooms were set aside for married mothers



Malone Place Hospital (about 1950). The figure at the door is Miss D Hamilton, who was matron 1942 – 65.

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Based on *Malone Place Hospital — a short history,* by Renée E Pelan, SRN, SCM, RMN, published privately, 1981.

(£5.25 for 10–12 days). During the war in 1939–45, Malone Place opened its doors to many other distressed persons such as mothers and children following fathers and husbands in the Forces, girls rejected for enlistment, and stranded, blitzed refugees. Some 700 such were given shelter for one or more nights.

Dr Elizabeth Robb had begun the teaching and training of student midwives, and by 1936 the home was recognised by the Joint Nursing and Midwives Council and approved as a training school for both parts of the professional examination. The cost for such students was £40 for one year's training. This continued until 1960. Obstetricians and paediatricians attended Malone Place Hospital, but in 1960 there was yet another radical change when, under the Northern Ireland Hospitals Authority, Malone Place became a general practitioner maternity hospital with 32 beds. It then ceased to be a training school and was staffed by qualified midwives. In 1973 it was amalgamated with the Jubilee Maternity Hospital. Between 1966 and 1972, owing to a shortage of midwives, a few state-enrolled nurses were introduced to assist — especially in the post-natal wards. In 1966 there were almost 1,500 deliveries, and often extra beds had to be used.

On the closure of the hospital in 1981 it was intended to base the South Belfast District Schools Dental Department in the premises so that (as the historian concluded) 'though the wards are silent, though they may never again re-echo to the cries of babies, the voices of children may be heard'.

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Investigation of non-cardiac chest pain — which oesophageal test?

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SUMMARY

Five different tests were used to evaluate oesophageal function in 22 patients who presented to a cardiac unit with acute chest pain but whose cardiological investigations were negative. Eight patients had an abnormality on oesophagoscopy, 10 had an abnormal pH monitoring study, six had a positive acid infusion test, 10 had an abnormal manometric study and six had an abnormal oseophageal transit scintiscan. Concordance for the three tests of gastro-oesophageal reflux disease was low at 28%, and for the two tests of oesophageal motility only 55%. Only two patients had normal results in all five tests.

INTRODUCTION

Patients with typical angina pectoris but without demonstrable ischaemic heart disease are a small but important problem in a specialist cardiology unit, and may be more frequent in general medical practice. Between 10% and 30% of patients investigated because of chest pain appear to be free of ischaemic heart disease.[1-3] Suggested cardiac reasons for their pain include coronary artery spasm, occult cardiomyopathy, myocardial bridging, small vessel disease of the myocardium, oxyhaemoglobin dissociation defects, or misinterpreted investigations.[4-5] Follow-up studies of these patients indicate a uniformly good cardiac prognosis,[6-8] and it is attractive to consider, therefore, that the cause of their pain lies outside the heart. Since the heart and oesophagus have a common sensory innervation, it is not surprising that pain from either organ is similar in nature and location.[9, 10]

Several reports have shown a high prevalence of gastro-oesophageal reflux,[2, 9, 11] or oesophageal dysmotility[11-15] in patients with 'non-cardiac' chest pain. We have

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used five of the many techniques currently available to seek oesophageal abnormalities in a group of patients with no demonstrable cardiac disease who presented with typical anginal pain to the cardiac unit. We have compared the yield of oesophageal abnormalities detected by the different tests in an attempt to determine a practical approach to the investigation of these patients.

PATIENTS AND METHODS

Over a 30-month period, 22 patients were identified as having non-cardiac chest pain. There were nine men, 13 women, age range 39 to 67 years (mean 51.3 years). All had presented acutely to the cardiac unit with pain typical of cardiac ischaemia and were provisionally diagnosed as having unstable angina or myocardial infarction. No patient complained of typical oesophageal symptoms such as heartburn, regurgitation or dysphagia.

In all cases myocardial infarction was excluded by serial electrocardiography and cardiac enzyme assay. No patient had a previously documented myocardial infarct. In all cases the electrocardiograph on admission was normal or showed non-specific ST segment or T wave changes only. All patients underwent a maximal symptom-limited exercise test with combined thallium scintigraphy soon after admission. In every case the exercise electrocardiograph was negative ($\geqslant$85% maximal predicted heart rate without $\geqslant$1 mm ST segment depression) or non-diagnostic ($<$ 85% maximal predicted heart rate with $<$ 1 mm ST segment depression). The thallium scans were assessed by two observers without knowledge of the clinical findings and all were considered normal. In six patients coronary arteriography was also performed and in all six this was normal.

Five tests were used to evaluate oesophageal function. Three tests — oesophagoscopy, prolonged ambulatory pH monitoring and an intra-oesophageal acid infusion test — were used to assess gastro-oesophageal reflux disease. Two tests — intraluminal oesophageal manometry and the oesophageal transit scintiscan — were used to assess oesophageal motility.

Oesophagoscopy: After administration of 5-10 mg diazepam intravenously the endoscopist intubated the fasting patient with an Olympus Q10 or D3 forward-viewing flexible endoscope. The mucosa of the lower oesophagus was assessed for erythema, linear streaking, erosions or gastric epithelialisation. The oesophago-gastric junction was noted and evidence of hiatus hernia sought by noting diaphragmatic 'pinching' of the gastric lumen during sniffing. Routine examination of the stomach and duodenum was also performed.

Prolonged ambulatory pH monitoring: A small pH sensitive radio-transmitter, similar in size to an antibiotic capsule, was suspended in the lower oesophagus, 5 cm above the oesophago-gastric junction. A portable radio receiver and recording device recorded oesophageal pH continuously while patients were fully mobile. All studies were conducted during an overnight stay in hospital. Food and drink of pH $<$ 5 were excluded from the diet. The pH sensitive capsule was swallowed at approximately 3.30 pm and removed at 9.00 am the following morning so that 17-18 hours of recording were obtained. Details of the apparatus and the technique of pH monitoring used in this study have been published.[16, 17] Reflux data from the patients both by day, when upright, and by night, when recumbent, were compared with normal values obtained from 27 control subjects (age 18-64 years) who were studied with the same apparatus under identical

conditions.[16] A reflux episode was defined arbitrarily as a fall in oesophageal pH to < 4 units.

Acid infusion test:[18] A nasogastric tube was placed with its distal end in the lower third of the oesophagus and connected by a T-piece to bottles of isotonic saline and 0.1 M hydrochloric acid. The control solution of saline was administered for up to 15 minutes, and then changed, without the patient's knowledge, to acid infusion. Both solutions were administered at a rate of 6 – 7½ ml/min. Repetitive reproduction of the patient's usual chest pain, typical in site and intensity, with rapid relief on changing to saline infusion, which occurred on three consecutive occasions was considered a positive test. A test was negative when acid infusion for 30 minutes provoked no pain. A test was considered 'positive-unrelated' when acid provoked a new and unfamiliar pain.

Intraluminal manometry: A four-lumen catheter was used, in which three lumina had side holes distally at different levels to measure intra-gastric, oesophago-gastric sphincter and intra-oesophageal pressures, and the larger fourth lumen was available for the instillation or aspiration of material. Each of the manometry lumina was connected separately to pressure transducers by a Y connection, the other limb of each connection leading to an infusion pump adapted to take three 50 ml syringes. Deionised water was infused into each lumen at a rate of 0.42 ml/min. The pressure transducers were connected to an amplifier and recorder, and their output was displayed on heat sensitive paper. The lower oesophageal sphincter length and pressure were measured using a 'station pull through' technique. Here the end of the catheter was placed in the stomach and then pulled back, step by step, through the sphincter so that first the proximal and then the middle and distal side holes passed through. This allowed a record of mean sphincter pressure and length. In response to a wet swallow, both sphincter relaxation and oesophageal peristalsis were assessed. Spontaneous motor activity in the oesophagus was also recorded.

Normal values for lower oesophageal sphincter pressure were adopted from Calvert's work in healthy controls using the same apparatus.[19] Normal values for mean sphincter length and degree of relaxation after a wet swallow were derived from the work of Benz et al.[20] Spontaneous motor activity was abnormal if there were > 3 non propagating spikes per 5 cm of recording at 2.5 mm/sec (RAJ Spence, unpublished data).

Oesophageal transit scintiscan:[21, 22] The patient swallowed 5 ml water containing 18 MBq technetium 99m tin colloid. Nine consecutive images of the oesophagus at three-second intervals were collected by a gamma camera. The test was performed twice for each patient. In normal subjects the water progresses smoothly from mouth to stomach in less than 12 seconds. In subjects with disturbed oesophageal peristalsis, there is a delay in transit time, or the bolus of water is broken up in the lower oesophagus with failure of propulsion into the stomach. The test was reported as abnormal if one or both demonstrated any of these abnormal features.

RESULTS

Oesophagoscopy: Twenty-one patients underwent this investigation. Three (14%) had a hiatus hernia, three (14%) had oesophageal erosions and two (9%) had both a hiatus hernia and oesophageal erosions. In the remaining 13 (63%) patients no endoscopic abnormality was detected (Table).

TABLE

Oesophageal function tests in 22 patients with non-cardiac chest pain

H = hiatus hernia, E = erosion of oesophageal mucosa, N = normal, A = abnormal

	Reflux Tests			*Motility Tests*	
Patient	*Endoscopy*	*pH monitoring*	*Acid infusion*	*Manometry*	*Scintiscan*
1	H+E	N	A	A	N
2	N	N	N	N	N
3	N	A	N	N	A
4	H	N	N	N	A
5	H+E	N	N	A	N
6	H	–	N	A	A
7	H	A	N	A	N
8	N	A	N	A	N
9	N	A	N	N	N
10	E	A	A	N	N
11	E	A	N	N	N
12	N	N	N	N	N
13	N	N	N	A	N
14	N	A	A	N	N
15	N	A	A	N	N
16	N	A	N	A	A
17	N	N	N	A	N
18	N	A	N	N	N
19	E	–	A	–	N
20	N	–	N	N	A
21	–	–	N	A	A
22	N	N	A	A	–

Prolonged ambulatory pH monitoring: Two patients refused this test and in two others the apparatus failed. In the other 18 patients a technically satisfactory recording was obtained. Abnormal gastro-oesophageal reflux, mostly of a minor degree, was noted in 10 patients (56%). This was either an increase in the frequency (seven patients) or an increase both in frequency and duration (three patients) of reflux episodes (Figure). In nine of these patients, abnormal reflux only occurred whilst in the upright position, and in one patient it occurred in both the upright and the recumbent positions. In two patients typical chest pain occurred during the pH monitoring. One of these patients developed pain of such severity that admission to the coronary care unit was arranged. Oesophageal pH was noted to be normal while the patient had the pain before the probe was removed at the patient's request. The other patient had two episodes of typical chest pain during monitoring, of which the first was associated with acid reflux and the second was not.

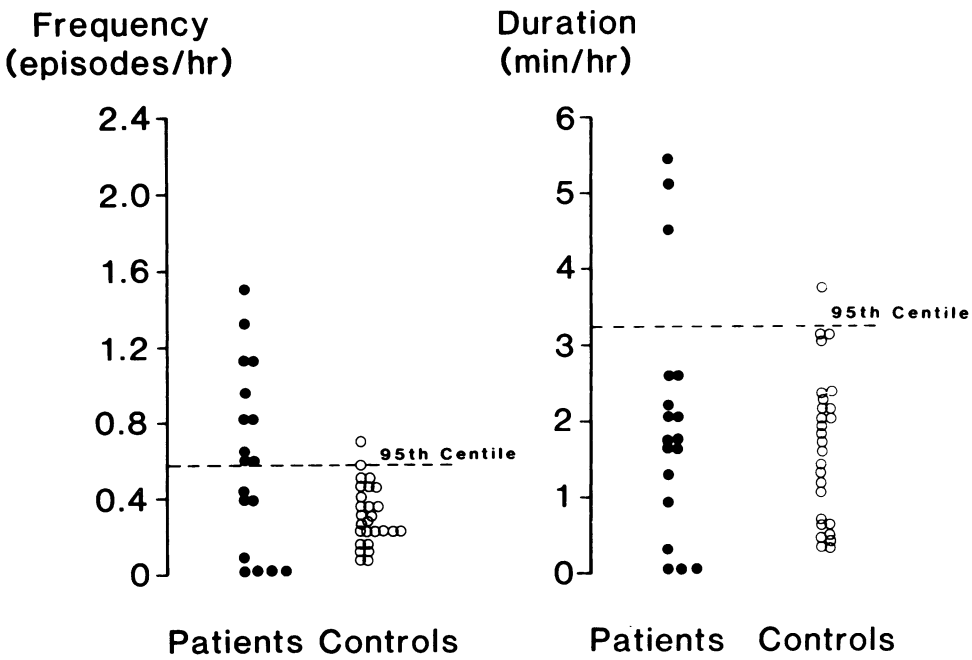

Figure. Reflux pattern (pH < 4) in chest pain patients and healthy controls

Acid infusion test: All patients underwent this investigation and in six (27%) a positive result was obtained. Two other patients had a 'positive unrelated' result and these were regarded as negative for oesophageal disease. No patient experienced acid-induced pain unrelieved by change to saline infusion.

Intraluminal manometry: This study was performed in 21 patients. In 10 (48%) an abnormality was noted. In two patients there was inadequate relaxation of the lower oesophageal sphincter and in one of these there was also inco-ordinated peristalsis in the oesophageal body. The other eight patients had increased spontaneous activity and inco-ordinated peristalsis of the oesophagus. All patients had a normal sphincter pressure and length. No patient experienced chest pain during the manometric evaluation.

Oesophageal transit scintiscan: This test was performed in 21 patients and in six (29%) patients one or both swallows were abnormal. No chest pain occurred during the scintiscan studies.

Concordance for all three reflux tests, either all normal or all abnormal, was very low at 28%. Between pairs of reflux tests concordance was also low. For endoscopy and pH monitoring concordance was 44%, for endoscopy and the acid infusion test 62% and for pH monitoring and acid infusion 50%. Concordance between the two tests of oesophageal motility was 55%.

DISCUSSION

Our results confirm the previously reported high prevalence of abnormalities which can be detected during oesophageal function testing in patients with non-cardiac chest pain.[2, 9, 11-15] In only two of our 22 patients were all tests normal.

The pattern of abnormalities found in the other 20 patients is disturbing in view of the low concordance between tests which purport to detect either gastro-oesophageal reflux disease or oesophageal motility disturbances. Differences in the sensitivity of these tests must account for some of the discrepancies in the results observed. It is also likely that disturbed oesophageal function is an intermittent phenomenon. If this is so, then caution is required in comparing tests used to detect such disturbances when they are not performed simultaneously. It is also necessary to consider the relevance of any detected abnormality to the chest pain which led to the patient's initial cardiological assessment.

Apart from the acid infusion test, which aims to provoke chest pain, only two patients experienced spontaneous chest pain during any of the other tests. For most of our patients, therefore, we cannot prove a causal link between the oesophageal abnormalities we detected and the chest pain they experienced. The two patients who developed spontaneous chest pain did so during prolonged pH monitoring. We suspect that more of our patients would have experienced pain if they had worn the apparatus at home or at work and had engaged in activities liable to provoke symptoms.[23]

We found that oesophageal manometry was a sensitive test of disordered motility, but the interpretation of minor abnormalities in particular is difficult since their clinical relevance is not well established.[24] The oesophageal scintiscan is a relatively unproven test of oesophageal dysmotility, but it is attractive as a screening procedure in view of its simplicity. Our finding of low concordance between these two tests carried out sequentially is disappointing. Studies using simultaneous manometry with the transit scintiscan are needed to determine the correlation between dysmotility and disordered bolus transit through the oesophagus. Further information may be derived from provocative studies using ergometrine[25, 26] or edrophonium,[26] particularly if chest pain is provoked and coincides with disordered motility.

The intra-oesophageal acid infusion test, introduced by Bernstein,[18] was useful in six patients in whom their usual chest pain was reproduced. In three of these patients there was no associated abnormality detected by endoscopy.

In conclusion, can we recommend oesophageal investigation routinely in patients with non-cardiac chest pain; and which of the tests should be performed? Fibre-optic endoscopy is widely available and allows examination not only of the lower oesophagus but also of the stomach and duodenum. We regard the high yield of erosive oesophagitis (five patients) in our study as justification for the use of endoscopy as the first line investigation in patients with non-cardiac chest pain. We do not share the view of some workers[27] that oesophageal biopsy is a proven adjunct to endoscopy, particularly when other tests of reflux are available. In patients with a normal endoscopic appearance the acid infusion test or, if available, prolonged ambulatory pH monitoring are the logical next steps. The tests of oesophageal motor function — transit scintiscan and manometry — are of limited value unless they can be performed during a manoeuvre which reproduces pain.

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Malaria in Northern Ireland

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SUMMARY

The clinical features, parasitology and prophylactic history of 67 patients who imported malaria to Northern Ireland between 1974 and 1983 are reported. P. falciparum *infections were encountered more frequently than anticipated from current United Kingdom experience. The clinical implications of these findings are discussed.*

INTRODUCTION

Malaria remains a major health problem for more than one thousand million of the world's population, and areas where infected anopheles mosquitos may be encountered are growing. Once endemic in the British Isles, malaria declined in importance during the 18th and 19th centuries. The indigenous anopheles mosquito prefers animal to human blood, and changes in animal husbandry and marsh reclamation meant that, by the early 20th century, indigenous malaria had almost disappeared.[1] Thereafter in the British Isles malaria has been a disease of importation, a trend strikingly illustrated by peaks in malaria incidence that followed the return of those who served in malarial areas during two world wars and the Korean conflict.

The situation changed radically during the decade surveyed with the explosive growth in international travel. The incidence of malaria in the United Kingdom increased from 540 cases (1973) to 2,053 cases (1980). These figures constitute more than half of all malaria importation into Europe.[2] Two groups contribute to this excess of malaria importation — new immigrants and former immigrants returning from a visit to their country of origin. Since the majority of these patients acquire infection in Asia, the predominant parasite involved is *P. vivax*. In the United Kingdom infection by *P. vivax* and other relapsing malarias accounted for more than two-thirds of isolates during the survey period. This study was undertaken to investigate a clinical impression that the pattern of malaria importation to Northern Ireland differed from that of the United Kingdom.

METHODS

A retrospective survey of hospital records for the period 1974 – 1983 identified 63 malaria patients (59 in-patients, four out-patients). Laboratory records added

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four cases, giving a total of 67 patients with recognised malaria during this decade. All but five patients initially presented to their family doctor. Most were then referred to infectious disease consultants (40 cases), general physicians (14 cases), or consultant haematologists (three cases). For seven patients, the diagnosis was made clinically on the basis of fever following travel to an endemic area which responded to specific anti-malarial therapy. In the remaining cases, diagnosis was established by blood film examination in local haematological laboratories. Further confirmation of this parasitological diagnosis was obtained in 41 cases from the Public Health Laboratory Service, Malaria Reference Laboratory (London School of Hygiene and Tropical Medicine) or from the Liverpool School of Tropical Medicine.

RESULTS

There were 50 male and 17 female patients, mostly in the 18 – 40 age range. Europeans, of whom there were 43, formed the largest ethnic group. There were 18 patients of Indian extraction, four of African origin, one patient was of mixed African/European descent and in one case there was no record of racial origin. Reasons for travel are set out in Table I and compared with similar data for the whole of the United Kingdom during the same period. The most readily identifiable group among patients of local extraction were missionaries (14) and employees of voluntary organisations (11), returning on furlough from malaria endemic areas. Immigrants provided a much smaller number of malaria victims than would be anticipated from the comparable United Kingdom experience.

TABLE I

Reasons for travel

Reasons for travel in 67 cases of malaria diagnosed in Northern Ireland compared with the figures for the whole of the United Kingdom.

	Cases	*Percentage of Northern Ireland cases*	*Percentage of United Kingdom cases*[3]
New immigrants	6	8.8%	23.3%
Former immigrants revisiting country of origin	10	14.8%	28.6%
Air/sea crews/military	4	5.8%	1.8%
Foreign visitors	—	—	6.8%
Long-term residents overseas returning home on furlough	14	20.8%	3.7%
Children visiting parents living abroad	6	8.8%	1.3%
Voluntary service overseas business	11	16.4%	5.4%
No record	16	23.8%	29.1%
	67	100%	100%

Northern Ireland malaria patients were most likely to have been infected in sub-Saharan Africa (39 cases), almost equally divided between east, central and western regions of that continent. As expected, the predominant parasite demonstrated was *P. falciparum.* Twenty-two patients presented on return from the Indian sub-continent and, where a parasitological diagnosis was made, all showed *P. vivax* infection. (Table II). Of the 56 patients for whom clinical data is available, all presented to their doctor with a fever or rigor. Other common symptoms were headache (22), myalgia (13) and symptoms referrable to the gastrointestinal tract, including nausea (9), vomiting (12), abdominal pain (8) and diarrhoea (7). Few had positive clinical signs. Hepatomegaly was present in only 14, splenomegaly in nine; five were jaundiced and three anaemic.

TABLE II

Parasitological diagnosis and source of infection

	Africa	India	Other	Total
P. falciparum	27*	—	—	27
P. vivax	2*	17	3	22
P. ovale	2	—	—	2
P. malariae	1	—	—	1
Parasites not specified	4	4	1	9
'Clinical' diagnosis without confirmation	5	1	1	7

* One mixed infection.

Of the patients infected with *P. falciparum* in whom the interval between arrival in Northern Ireland and the onset of symptoms was recorded, three-quarters presented within one month and the remainder within three months. In patients with *P. vivax* infection, only a quarter developed symptoms within the first month of their arrival, the remainder presenting within 12 months. In one case of *P. ovale* infection, the latent period was longer than a year. The single case of *P. malariae* infection presented two months after return to this country.

Details of the malaria prophylaxis were recorded in only 45 of the cases. Seventeen patients took no prophylaxis whatever, six patients took some form of medication irregularly, six others had discontinued regular prophylaxis immediately on return or before completing four weeks in this country. Sixteen patients were recorded as taking full prophylaxis; proguanil in two cases, chloroquine in two cases, pyrimethamine (Daraprim) in five cases, or pyrimethamine and dapsone (Maloprim) in seven cases. All the patients were treated initially with chloroquine. Haemolysis occurred in one case and methaemoglobinaemia (following the use of dapsone) in another. Quinine was required for three cases of *P. falciparum* infection — all acquired in Africa — which proved resistant to chloroquine. There were no deaths and there is no record of any serious sequelae.

DISCUSSION

We located fewer malaria infections in Northern Ireland than overall United Kingdom statistics led us to expect. This probably reflects the small proportion of immigrants to our community. In the whole United Kingdom, more than half the patients with malaria were immigrants, most acquiring their infection on the

Indian sub-continent where *P. vivax* infection dominates. The resulting illness usually has a typical symptomatology, likely to be recognised as malaria, especially if the patient is Indian. Even where diagnosis is delayed, little harm ensues. In Northern Ireland most of those infected were of local origin and were infected in Africa where *P. falciparum* infection is common. This has considerable clinical significance. The resulting illness frequently follows a fulminant course and can progress rapidly to death, yet the symptoms and clinical findings are much less specific.

P. falciparum was responsible for almost all malaria deaths occurring in the United Kingdom, the annual case mortality varying from 1.4% to 10% during the survey period. In almost all instances, death from falciparum malaria was the consequence of late diagnosis. In Northern Ireland, there were no deaths during the survey period, but there is no room for complacency. Our current good record will be maintained only by extreme clinical vigilance. The presenting features of malaria, particularly the dangerous *P. falciparum* infections, mimic the many simple locally acquired fevers and gastrointestinal upsets. It is esential that both hospital doctors and family practitioners consider malaria as a possible diagnosis in any febrile patient recently returned from a malaria endemic region. Even completion of a full course of anti-malarial chemoprophylaxis does not exclude this possible diagnosis.

Many of those infected failed to seek or did not receive adequate advice about the need for malaria prophylaxis. The family doctor is usually approached to provide this and with the rapid spread of drug-resistance strains of *P. falciparum,* it is essential that such prophylactic advice is constantly updated. The Public Health Laboratory Service Malaria Reference Laboratory (London School of Hygiene and Tropical Medicine) periodically publish such details.[3] Together with the Liverpool School of Tropical Medicine, they provide a telephone answering service. The Medical Advisory Service for Travellers Abroad (MASTA) at the London School of Hygiene and Tropical Medicine also include advice on malarial prophylaxis within their comprehensive health brief. Whatever prophylactic therapy is chosen, patients must be advised to start medication a week prior to departure and to maintain drug cover throughout their stay in the malaria endemic area and subsequently for four to six weeks after their last possible mosquito contact. Prescriptions for drug prophylaxis must be supplemented by emphatic advice on methods of avoiding mosquito bites including the use of mosquito nets, repellent creams and sprays and suitable clothing for the period between dusk and dawn.[4] Such simple measures do much to reduce the risk of malaria.

The authors wish to thank the doctors and medical records officers who helped by making available patients' clinical notes.

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Surgeons' attitudes to some aspects of day case surgery

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SUMMARY

The level of day case surgery is much lower in Northern Ireland than in England. A questionnaire was sent to all 55 consultant general surgeons in Northern Ireland to assess attitudes to this form of care and 51 (93%) replied. They were asked about the suitability of five procedures for day surgery. The three minor procedures of vasectomy, cystoscopy and gastroscopy were regarded as suitable or very suitable by 50 (98% of those who replied), 48 (94%) and 48 (94%) respectively. For the two intermediate procedures, 25 (49%) regarded the repair of inguinal hernia as suitable for day case surgery and 22 (43%) ligation of varicose veins. When asked about eight factors limiting their use of day surgery for inguinal hernia repair, the two most frequently rated as important were 'home conditions' and 'level of provision of domiciliary care' (both by 44 (86%) of the surgeons). Of factors which might promote their use of day surgery for this operation the two most important were 'more efficient use of health service resources' (71%) and the 'ability to convalesce at home' (67%). The problem of under-reporting of day cases and the importance of accurate statistics are considered.

INTRODUCTION

In 1913 Fullerton, working in the Royal Belfast Hospital for Sick Children (then in Queen Street), was an early pioneer of day case surgery.[1] Since then, this form of care has not been greatly developed in Northern Ireland. The present decade has seen some renewed interest and developments starting with the opening of the day surgery unit in the Massereene Hospital in 1980.

Some 22% of all operations and procedures in England were treated as day cases in 1982.[2] There was no comparable data for Northern Ireland until the recent introduction of the Hospital Activity Analysis (HAA) 'Day case notification form'.

The well established Hospital Statistical Return (HSR) recorded day cases (in which day case operations are subsumed) as totalling 14,385 for all specialities in 1984 (DHSS Statistics & Research Branch). In that year 4,910 day case operations were reported through HAA, representing 2.2% of all operations performed in Northern Ireland (DHSS Statistics & Research Branch). While it is

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accepted that under-reporting of day cases occurs,[3] it seems unlikely that it should account for anything like the tenfold difference between England and Northern Ireland.

Whatever the true discrepancy, the Department of Health and Social Services (NI) maintains that there is substantial scope to increase the number of day case operations in Northern Ireland.[3]

It has been estimated that half of all patients operated on could be treated as day cases.[4,5] One operation of particular interest in relation to day care is the repair of inguinal hernia. It is the commonest operation undertaken in adult males in England,[6] and the condition contributes significantly to waiting lists as more serious conditions and emergencies take precedence. The feasibility of treating 'good risk' patients for this operation as day cases has been reported by many researchers. Most convincing are the results of several randomised controlled clinical trials which show no statistically significant difference in the complication rate compared with in-patient care.[7,8] Only 3.3% of inguinal hernia repairs were treated on a day case basis in England in 1982.[2] In Northern Ireland only 21 cases of inguinal hernia repair were reported through the HAA day case system in 1984.

This study was carried out to discover the attitudes of consultant general surgeons to day case surgery in general, and also what factors most influenced them for and against treating inguinal hernia repair as a day case procedure.

METHOD

All 55 consultant general surgeons in Northern Ireland were sent a short postal questionnaire. The questions had been formulated after a review of the literature and a pre-test which consisted of structured interviews with eight consultant surgeons. The first question asked about the suitability of five procedures in 'good risk' patients. Three of these were 'minor' and often treated on a day care basis. The other two, repair of inguinal hernia and ligation of varicose veins, were of an intermediate nature and responses here would be of special interest.

Two questions specifically related to repair of inguinal hernia. The surgeons were asked to assess the importance of eight different factors in limiting their use of day case treatment for this procedure. Conversely the third question asked about the importance of eight other factors which might promote the use of day care. The categories of response were of varying degrees of positive and negative with a space to include 'no opinion'. Question 4 asked whether they wished to see an increase, decrease or no change in the level of day case surgery.

RESULTS

Fifty-one of the 55 general surgeons returned completed questionnaires, a 93% response rate. Because of the small numbers involved, certain categories of response, eg. 'suitable' and 'very suitable', are amalgamated in the tables for clarity.

In response to question 1 the great majority regarded the three 'minor' procedures as being suitable (or very suitable) for day care. (Table I). Vasectomy was rated suitable by 50 (98%) of surgeons, and 48 (94%) rated both cystoscopy and gastroscopy as suitable for day care treatment. Responses as to the suitability of the two intermediate operations were different: 25 (49%) of the surgeons regarded inguinal hernia as suitable (or very suitable), while only 22 (43%) regarded ligation of varicose veins as suitable (or very suitable) for day care

treatment. The difference was more marked when only those who had expressed an opinion were considered, with a clear majority regarding inguinal hernia repair as suitable for day care.

TABLE I

Surgeons' opinions of the suitability of five operations for treatment as day cases. (Amalgamated categories)

Operation	*Very unsuitable or unsuitable*	*Suitable or very suitable*	*No opinion or missing information*
Cystoscopy	1 (1.9%)	48 (94.2%)	2 (3.9%)
Inguinal hernia repair	20 (39.1%)	25 (49.0%)	6 (11.9%)
Gastroscopy	1 (1.9%)	48 (94.2%)	2 (3,9%)
Vasectomy	1 (1.9%)	50 (98.1%)	0 (0.0%)
Ligation of varicose veins	24 (47.1%)	22 (43.1%)	5 (9.8%)

Table II refers to the results of question 2 on the importance of certain factors limiting day case treatment of inguinal hernia repair. Only 13 (25%) regarded the 'incidence of medium and long term complications' as important and 18 (35%) regarded both the 'incidence of immediate complications' and 'lack of theatre provision' as being important limiting factors. All the remaining factors were regarded as important by over half the surgeons. 'Patients' negative attitudes' were thought to be important by 33 (65%) while 34 and 35 surgeons (66.6% and 68.7%) regarded 'organisation and communications problems' and 'post-operative pain' respectively as important limiting factors. The factors most frequently cited as limiting were 'level of provision of domiciliary care' and 'home conditions', each of which was regarded as important by 44 (86%).

TABLE II

The surgeons' opinions of the importance of eight factors in limiting *their use of day case treatment for the repair of inguinal hernia. (Amalgamated categories)*

Factors	*Very unimportant or unimportant*	*Important or very important*	*No opinion or missing information*
Incidence of immediate complications	25 (50.0%)	18 (34.3%)	8 (15.7%)
Organisation and communication problems	10 (19.7%)	34 (66.6%)	7 (13.7%)
Post-operative pain	12 (23.5%)	35 (68.7%)	4 (7.8%)
Lack of theatre time	24 (47.1%)	18 (35.3%)	9 (17.6%)
Patients' negative attitudes	7 (13.7%)	33 (64.7%)	11 (21.6%)
Incidence of medium and long term complications	29 (56.9%)	13 (25.4%)	9 (17.7%)
Level of provision of domiciliary care	2 (3.9%)	44 (86.3%)	5 (9.8%)
Home conditions for convalescence	2 (3.9%)	44 (86.3%)	5 (9.8%)

Table III shows the answers to question 3 on the importance of some factors in promoting the use of day care for inguinal hernia repair. The factor which would have stimulated most respondents, 36 (71%), to use day care was 'more efficient use of health service resources'. Thirty-four (67%) regarded the 'ability to convalesce at home' as important. Other factors which would play a major role in promoting day surgery were 'more bed days available for the seriously ill' and 'pressure on beds'. Just over half, 26 (51%), rated both 'reduced waiting times' and 'promotion of integration between hospital and community issues' as important.

Only 24 (47%) cited 'less disruption for family' as important and only 10 (20%) regarded 'sooner return to work' as an important factor in promoting day care for inguinal hernia repair.

Answers to the fourth question showed that a small majority, 27 (53%), were in favour of an increase in day surgery, 21 (41%) wanted no change and no surgeon wished to see it decrease.

TABLE III

The surgeons' opinions of the importance of eight factors in promoting *their use of day case treatment for inguinal hernia repair. (Amalgamated categories)*

Factors	*Very unimportant or unimportant*	*Important or very important*	*No opinion or missing information*
Ability to convalesce at home	7 (13.7%)	34 (66.7%)	10 (19.6%)
Less disruption for families	12 (23.5%)	24 (47.1%)	15 (29.4%)
More efficient use of health service resources	5 (9.7%)	36 (70.7%)	10 (19.6%)
Sooner return to work	18 (34.4%)	10 (19.6%)	28 (45.0%)
Reduced waiting time	11 (21.5%)	26 (51.0%)	14 (27.5%)
Promotion of integration between hospital and community services	9 (17.6%)	26 (51.0%)	16 (31.4%)
Pressure for beds	10 (19.6%)	32 (62.8%)	9 (17.6%)
More bed days available for seriously ill patients	8 (15.6%)	33 (64.8%)	10 (19.6%)

DISCUSSION

The high response rate was a notable achievement in itself. We do not know of any surveys or reported response rates of general surgeons, and informed opinion was pessimistic about the response. The results demonstrate the common phenomenon of reported attitudes differing widely from actual practice. While nearly half of the surgeons judged the repair of inguinal hernia as suitable for day case treatment, very few such operations are so treated. The factors which most limited the use of day care for this condition were thought to be subjective patient factors (including pain) and wider community factors. With the partial exception of post-operative pain, all these factors are out of the surgeon's hands. It may well be that his concern is well founded and it is not good practice to

submit patients to inadequately relieved pain or poor care and conditions at home. Alternatively a surgeon can be seen as unwilling to submit patients to day surgery as it involves care outside his immediate control, the quality of which he cannot be certain. The organisation of domiciliary care is outside a surgeon's *direct* remit and the optimal co-ordination of care for day cases may not be seen as his responsibility. A third, more cynical view might be that some of these attitudes may reflect the discouragement of, or apathy towards a policy which involves much effort and which relatively generous bed provision has made unnecessary. By consciously or unconsciously blaming factors outside the surgeon's control the matter can be dismissed. The results may reflect a mixture of such attitudes, varying with each individual surgeon.

It is encouraging to find that most surgeons recognised day surgery as a means of improving the efficiency of the service and this would tend to influence them in favour of the practice. Why then does day surgery appear to be practised much less commonly in Northern Ireland? The relative provision of (acute) hospital services and the use of these is of some interest. Northern Ireland has proportionally about twice the number of general surgical beds, general surgeons and surgical admissions as England (Table IV).

TABLE IV

General surgical provision and use of facilities 1983

	Northern Ireland	*England*	*Scotland*	*Mersey RHA*
Available beds per 1,000 population	1.13	0.55	0.74	0.58
Throughput per bed	34.0	38.6	33.7	37.3
% bed occupancy	75.9	77.3	70.7	83.9
Admission rate per 1,000 head of population	38.4	21.1	24.8	21.8
Consultants per 1,000 population (WTE)	0.04	0.02	0.03	0.02
Waiting list numbers per 1,000 population	3.4	3.4	4.6	4.2

WTE = Whole Time Equivalent.

Source — Research and Statistical 1 Branch, Management Services, DHSS (NI).

It has been argued that a fairer comparison with England would be to amalgamate data for general, urological surgery with trauma and orthopaedic surgery. Even if this is done the number of available beds *per capita,* for example, in Northern Ireland is 39.3% higher than in England.

Four basic reasons to explain these figures can be put forward. Firstly, a higher level of morbidity may exist in Northern Ireland, justifying a higher level of service activity and provision. The civil disturbances and the higher levels of traffic accident injuries are two examples of this. Morbidity is notoriously difficult to measure and mortality is usually taken as a proxy measure. Standardised mortality is higher in Northern Ireland than in other parts of the UK. (Table V). However, this does not account fully for the very high admission rate. A region such as Mersey which is disadvantaged, still has a much lower level of provision of services.[9]

TABLE V

Standardised mortality ratio (SMR) by country and sex 1980

	Northern Ireland	*England*	*Scotland*	*Wales*	*UK*
Males	117	98	107	113	100
Females	112	99	103	109	100

Source — General Registrar's Office.

The number of consultants and their activity may be a second reason. Is there a fixed amount of disease which the available clinicians divide amongst them or does the appointment of more consultants result in more clinical activity? The results of research on this question are ambiguous.[10] Buttery compared the regions in England and suggested that the number of surgeons was inversely proportional to the number of operations performed by each, though this correlation was not statistically significant.[11] A study in Scotland showed that the number of cholecystectomies in each health board was positively correlated with the number of surgeons per head of population.[12]

Thirdly, a number of authors report a positive association between admission rates and the number of in-patient beds.[13-15] Linked to this is the threshold for admission. For general practitioners this appears to be altered by the perception of the bed availability and level of hospital activity. Several authors demonstrate that the number of admissions for emergencies and for terminal care respectively correspond with perceptions of a limitation of services.[16, 17] In relation to day surgery, a number of published examples show that it was the stimulus of limited beds and long waiting lists which led to the setting up of day care surgery programmes and units.[18-20]

Fourthly, it may be argued that surgeons (and other consultants) in Northern Ireland are able to provide a better service, and are improving the population's health and comfort because of the level of services here. A natural corollary to this is that any equalisation of provision vis-à-vis the rest of the UK should be a levelling upwards. Whatever the validity of that view, it would be naïve to maintain it seriously in the current economic and political climate. Rationalisation and re-distribution of resources within the health services in Northern Ireland with a reduction in acute beds, and more day case surgery seems inevitable.[3, 21]

In theory the last development should not be much at odds with the attitudes of general surgeons as reported in this survey. It may be prudent for surgeons and all clinicians involved in day care actively to translate theory into practice and to try to overcome the limitations to day care as they see it. In co-operation with other disciplines in the health and social services, real problems regarding, for example, communication, and co-ordination of domiciliary care can be addressed and overcome. By initiating change themselves, clinicians will be in a better position to respond to redeployment of resources and even to obtain extra resources that may be needed to set up efficient day surgery programmes in the first place. Accurate, complete, and up-to-date statistical information is vital for assessing situations and for rational planning and efficient running of the service. Clinicians will be best served if there is relevant data crediting them with their actual workload. Earlier in this paper some of the obvious deficiencies and

under-reporting in the realm of day case surgery were indicated. The importance of recording all day cases on the recently introduced HAA day case notification form should be realised and transmitted from clinicians to ward clerk and records staff.

We would like to thank all the consultant surgeons who participated in the survey, and Dr S N Donaldson, Senior Medical Officer, and Miss Maureen Boyd, Statistical and Research Branch, DHSS (NI).

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In view of the controversial nature of this paper, the editor has asked for a considered opinion from a surgical viewpoint.

SURGEONS' ATTITUDES TO DAY CASE SURGERY – Invited commentary.

The authors are fortunate in having obtained a 93 per cent response to a detailed questionnaire without personal contact with the surgeons circularised. However, the paper suffers from the fact that it is based on that questionnaire without fieldwork such as examination of at least some theatre operating book statistics. This form of research would have provided a better guide to the actual incidence of day case surgery than the HAA where 'it is accepted that under-reporting of day cases occurs'. Certainly no surgeon would quarrel with their views that 'accurate, complete and up-to-date statistical information is vital for assessing situations and for rational planning and efficient running of the service'. However, there are many weak links in the chain that leads from the clinician to the final draft of the HAA. Up to now, clinicians have felt that their time was best occupied in looking after clinical problems rather than statistical recording; obviously their attitudes will have to change.

In the survey, over 90 per cent of surgeons agreed to the eminent suitability of cystoscopy, gastroscopy and vasectomy for day case surgery. However, the authors dismiss these and focus on herniorrhaphies where less than half of the surgeons were in favour of day case procedures for selected patients with satisfactory back-up facilities. The discussion is based almost exclusively on day case herniorrhaphies, but the authors keep equating that with day case surgery. It must be pointed out that hernial repair represents only a small proportion of total surgery in any unit and an even smaller percentage of day case surgery. Indeed, in England and Wales, as they point out, only 3.3 per cent of inguinal hernias were done as day cases in 1982. If this figure of 3.3 per cent includes herniotomies in children, ideally suited to day case surgery, then the argument becomes even weaker. Thus during the year 1985 one surgical unit in the Royal Victoria Hospital admitted approximately 1,500 patients and operated on almost 1,000 in-patients. Of these in-patients only 50 had herniorrhaphy. Perhaps, at best, 20 of these patients might have been suitable for early post-operative discharge if we exclude elderly males with the risk of retention, various medical diseases in other old age group patients, those done under spinal anaesthesia, mothers of large families, etc. Should we have the back-up facilities for early discharge of these 20 patients they would still represent less than five per cent of the day case surgery in that surgical unit where there were approximately 500 day case operations in the same year (these included gastroscopies, vasectomies, lumps and bumps, etc). Only if one equates herniorrhaphies with day case surgery as the authors have done, could one conclude that 'day case surgery has not greatly developed in Northern Ireland since 1913'. The cynical view of 'surgeons' apathy towards a policy which involves much effort' is hardly borne out by the 93 per cent response to the questionnaire.

Comparison of the numbers of surgeons and surgical beds in Northern Ireland with those in England and Wales is made in a way to suggest that England and Wales have the right proportion. Some would suggest that comparison with rural Scotland would be fairer. In addition the United Kingdom has fewer doctors per unit population than any country in Europe except Turkey. Only 5.7 per cent of the gross national product of the United Kingdom is spent on health care compared with 8 per cent in France and Germany and 10 per cent in Sweden.

The Short report recognised the need for an increase in the number of consultants in the United Kingdom and recommended that this be carried out forthwith; unfortunately this has not been implemented in spite of pressure from the Royal College of Surgeons in England. Could it be that Northern Ireland is nearer the ideal than England and Wales? The fact that more cholecystectomies were carried out in Health Boards with greater numbers of surgeons per head of the population is no bad thing. Many patients were thereby saved from suffering obstructive jaundice, ascending cholangitis, pancreatitis and even cancer of the gall bladder — normal gall bladders are not usually removed surgically.

For the authors to suggest that it may be 'prudent for surgeons and all clinicians involved in day case activity to translate theory into practice' is hardly justified when they base all their arguments on herniorrhaphies which constitute less than 5 per cent of the problem. The 'accurate, complete and up-to-date statistical information' they require is available in the in-patient and out-patient theatre operating books in any hospital.

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Case report

Fatal *Listeria monocytogenes* meningitis in two previously healthy adults

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Two patients, previously healthy, presented with *Listeria monocytogenes* meningitis within nine months of each other to a district general hospital with a catchment population of 85,000. Early disturbances of consciousness, with haematuria, proteinuria and minimal signs of meningism were similar in each. Neither responded to recommended antibiotic therapy and both died. The serological types were dissimilar. Epidemiological studies in the second case failed to identify a source of infection.

CASE ONE

A 74-year-old builder was admitted on 24 October 1982, in a semiconscious state. Headache, myalgia and fever the previous day had been attributed to a viral illness, but on the morning of admission he was found unrousable in bed having been incontinent. His only past history was of surgery for duodenal ulceration in 1968. He had a pyrexia of 39.5°C and was moving all limbs purposefully to pain. There were no localising neurological signs. Pulse 80/minute, blood pressure 160/90 mmHg, fundoscopy revealed no abnormality. He had minimal neck stiffness but Kernig's sign was negative and he had no photophobia. There were no chest signs and the abdomen was soft. Urinalysis showed moderate haematuria and proteinuria but no pus cells and sterile on culture. Haemoglobin was 15.4 g/dl, white cells 14.6 (a differential count was not done), blood urea 7.7 mmol/l, chest X-ray moderate cardiomegaly with clear lung fields.

By the following morning he had deteriorated, with a fall in the level of consciousness and increased neck stiffness. Lumbar puncture revealed cloudy fluid, pressure 24 cm. Pyogenic meningitis was confirmed: CSF protein 3.75 g/l (normal value 0.15 – 0.45 g/l), glucose 0.85 mmol/l, (plasma glucose 10.5 mmol/l), 76 leucocytes/µl (70% polymorphs). No organism was isolated for two days but empirical therapy was started 24 hours after admission with intravenous benzylpenicillin, 12g/day, chloramphenicol, 3.6g/day, and sulphadimidine, 4g/day. Forty-eight hours after admission, due to the persisting pyrexia (39.5°C), therapy was changed to ampicillin, 8g/day, erythromycin, 2.4g/day, and, for 24 hours only (because of deteriorating renal function), netilmicin,

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450 mg/day. He initially showed some improvement: his pyrexia fell slowly over the succeeding 48 hours and he was able to answer questions. The following day, five days after the onset, he became apnoeic with fixed dilated pupils and unrecordable blood pressure and died despite resuscitative attempts. There was no post-mortem. Subsequent bacteriological investigations identified the organism as *Listeria monocytogenes* serotype IV.

CASE TWO

A 53-year-old farmer's wife was admitted on 15 July 1983, in a barely rousable state. She had been weak for two days with severe dizziness and nausea on standing and presented with a 24-hour history of increasing drowsiness, headache and restlessness. Her only past history was of joint pains since 1978, but rheumatoid serology was negative. She had been treated with phenylbutazone but never with corticosteroids. She kept a cow and drank unpasteurised milk. She had a pyrexia of 38°C. She was restless and responded only to pain. There were no localising neurological signs. Pulse rate was 80/minute, blood pressure 110/70; fundoscopy revealed no abnormality. There was ill-defined right hypochondrial pain but no organomegaly or peritonism. The chest was clear. Urinalysis showed marked haematuria and minimal proteinuria. Haemoglobin was 10.1 g/dl, white cells 12.7 (no differential), blood urea 6.2 mmol/l. AST was 251 U/l (normal range 2 – 35) but other liver function tests were normal. On admission there was no neck stiffness and Kernig's sign was negative, but these developed over the next four hours. Lumbar puncture then revealed cloudy fluid (pressure not recorded). Protein was recorded as 5.4 g/l, glucose 0 mmol/l, leucocytes 250/μl, mainly polymorphs.

Empirical therapy was started with benzylpenicillin, 12g/day, chloramphenicol, 3.6g/day, and sulphadimidine, 4g/day, and continued for 12 hours until an organism was grown (although not identified), with sensitivities to gentamicin, co-trimoxazole, ampicillin and erythromycin. Then, 20 hours after admission, therapy was changed to ampicillin, 10g/day and co-trimoxazole, 6.4g/day, both intravenously. Later that day *Listeria monocytogenes* was isolated both from CSF and blood culture. She began having generalised tonic-clonic convulsions and short periods of apnoea within 11 hours of admission. The pyrexia persisted. Conjunctival oedema, which developed on the day after admission, became progressively worse. She died after four days, never having regained consciousness. There was no post-mortem. The organism was identified as *Listeria monocytogenes,* serotype I.

Environmental veterinary studies were subsequently carried out on the beef cattle and milking cow on the farm. No sheep were kept and no silage was available for study. No illness was detected and *Listeria monocytogenes* could not be isolated from the animals, the environment or the milk.

DISCUSSION

Listeria monocytogenes is a gram-positive bacillus capable of producing a spectrum of disease in man of which meningitis is the most common.[1] Although rare, it has been reported with increasing frequency in recent years, probably because of greater awareness of its potential pathogenicity.[1,2] It has a wide range of hosts including mammals, birds and fish, but no constant link has been found between animals and man.[3] Unpasteurised milk has been implicated as a source of human infection[4] and asymptomatic carriers described.[5]

Listeria monocytogenes meningitis has been found to occur more often in neonates[2] and in immunosuppressed patients.[1, 6] The latter commonly include patients with chronic renal disease both on dialysis and post-transplant.[1] Underlying malignancy, connective tissue diseases and alcoholism are also predisposing factors.[1, 2, 3, 7, 8] Age (50 and over) may be the only apparent risk factor.[2, 6, 7]

Listeria monocytogenes meningitis is a rare condition with 49 reported cases in adults in the UK during the period 1982/83 (unpublished: PHLS Communicable Disease Surveillance Centre; Communicable Diseases (Scotland) Unit). These include three adults from Northern Ireland. The UK figures do not accurately separate previously healthy adults, but our two patients had no identifiable risk factor except age. It must be considered as a possible cause of purulent meningitis especially when acute disturbance of consciousness and mild meningeal signs are the presenting features, and also when the CSF suggests a purulent meningitis but no organism is immediately identified. In one study, no organism was seen on gram stain of the admission CSF in 19 out of 25 patients.[8] The CSF examination in Case 1 was delayed because of the paucity of meningeal signs. This is well recognised. In a study of 40 cases, signs of meningeal irritation on admission were questionable in seven and absent in three.[7]

Our mortality is higher than that reported: 46 per cent in the over-50 age group,[8] 13 per cent in adults without underlying disease[1] and 20 per cent in the over-50 age group without underlying disease.[2] Our experience with antibiotic therapy in these two cases is similarly at variance with the literature. It has been suggested that ampicillin alone is adequate.[6, 8] Chloramphenicol has been advocated[8] although others found it either ineffective,[2, 9] or associated with an increased mortality.[6] Other reports suggest that additional aminoglycosides improve survival,[8, 9] and synergy has been observed *in vitro* using ampicillin with an aminoglycoside.[10, 11]

The organism is ubiquitous; its ability to survive is well known (several months in moist soil), as is its lack of consistent route of entry or mode of transmission.[3, 5] Most cases are reported from urban areas in the United States, whilst in Europe rural cases are more prevalent. Our epidemiological study in Case 2 failed to show either illness or a carrier state in the stock. Neither was *Listeria monocytogenes* isolated from milk samples or the environment. The different serotypes described suggest that chance and not a reservoir of infection resulted in these two cases of a rare and virulent infection.

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Case report

False elevation of serum thyroxine in myxoedema due to thyroxine-binding autoantibodies. A diagnostic pitfall

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The presence of anti-thyroid antibodies can cause artefactual estimates of thyroid hormones by radioimmunoassay. We describe a case of myxoedema with spuriously high serum total and 'free' thyroxine (T_4) levels which were initially misleading. Further investigations revealed the presence of IgG autoantibodies to T_4.

CASE HISTORY

A 29-year-old male Caucasian with no relevant family history presented with a history of constipation, increasing tiredness and a feeling of intense coldness. His performance at sport, notably rugby and cricket, had fallen off badly largely because of weakness and muscle stiffness. His hair was falling out and his skin had become very dry. The clinical diagnosis was of myxoedema. He had a past history of nocturnal convulsions related to heavy alcohol consumption for which he was taking phenytoin sodium 100mg at night, but alcohol intake was now modest. Initial tests of thyroid function were surprisingly high — serum total T_4 124nmol/l (reference range 60 – 160), serum 'free' T_4 168pmol/l (reference range 9 – 28). He was referred to St Luke's Hospital, Guildford, for further investigation.

On examination he appeared myxoedematous. Heart rate was regular, 60 beats/min, and pulse was of small volume. Blood pressure was 120/80mmHg. The thyroid gland was not enlarged. Examination of the nervous system and in particular of the tendon reflexes produced normal results. Electromyography suggested a mild proximal myopathy. Motor nerve conduction velocities of the right median and lateral popliteal nerves were in the low normal range at 50 and 34 m/sec respectively; sensory action potential of the median nerve at the wrist on stimulation of the index finger had normal amplitude of 9μV but a prolonged peak latency of 4msec. ECG showed sinus rhythm with low voltage complexes and T-wave inversion.

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Detailed thyroid function tests are shown in the Table. Other screening investigations were normal apart from serum creatine kinase 3250U/l (normal <160) and aspartate transaminase 129U/l (normal <40). Serum phenytoin was <10μmol/l. Serum albumin was 47g/l and total plasma protein 83g/l with a normal electrophoretic pattern. Serum IgG, IgM and IgA were all within normal limits. Haemoglobin was 11.3g/dl with normal indices and differential white cell count and the ESR was 5mm/h. The basal metabolic rate determined by spirometry was low at 83.9kJ/h/m² (−45% of mean for age and sex). He was started on triiodothyronine replacement therapy and the dose titrated to his TSH response. He improved rapidly both symptomatically and physically. Within three months, ECG, EMG and nerve conduction tests reverted to normal and he reported that he had not felt so well for at least a year. BMR rose to normal (−4%, 153kJ/h/m²).

METHODS

Serum total T_4, total T_3 and TSH were measured by radioimmunoassay using polyethyleneglycol-accelerated double antibody methods and antisera developed in this laboratory. Serum total T_4 was also measured following extraction of serum by an acid-ethanol method.[1] Serum 'free' T_4 was measured by equilibrium radioimmunoassay using a commercial kit (IM 2050) obtained from Amersham International. Further free T_4 measurements were made by equilibrium dialysis[2] and by a two-step solid-phase antibody method.[3]

Endogenous thyroid hormone antibodies:

(i) Increasing (five-fold) dilutions of serum (50μl) were incubated with 200μl aliquots of either iodinated T_4 (800pg), T_3 (10pg) or the Amersham T_4 analogue (an unknown amount). After 4 hours incubation at room temperature, aliquots (250μl) of normal carrier serum were added to the mixture followed by an equal volume (500μl) of a 20% solution of PEG 6000. After thorough mixing, the immunoglobulin fraction was precipitated by centrifugation and the associated radioactivity counted. Binding of the tracers by the patient's serum was compared with that to normal serum specimens.

(ii) The patient's serum was used at a 1:25 final dilution to set up a thyroxine immunoassay standard curve using iodinated-T_4 as tracer and a variable amount of unlabelled T_4 within the range 0 − 480nmol/l. After overnight incubation at 4°C, bound and free labelled thyroxine were separated by the addition of carrier serum and PEG as described above. Data was computed by Scatchard analysis. Cross reactivity was estimated by a similar method and by comparing the percentage of bound tracer in the presence of a range of concentrations of unlabelled T_4 (0 − 480nmol/l) or thyroglobulin (0 − 230nmol/l).

(iii) An aliquot (100μl) of the patient's serum was incubated with 4ng of T_4 tracer for 2 hours at room temperature. A sample (10μl) of the incubate was subjected to separation by electrophoresis on cellulose acetate. Aliquots of normal serum treated similarly were run in parallel. A marker strip was stained with Ponceau S and the sample strips were sliced to correspond with the marker bands. The slices were counted for radioactivity content.

(iv) Serum was examined for T_4 autoantibodies by an ELISA system. The method uses T_4-coated micro-titred plates and enzyme-labelled antihuman IgG antibodies as the detector.[4]

Thyroid microsomal and thyroglobulin autoantibodies were estimated using commercial haemagglutination kits (Wellcome Reagents Ltd, London).

TABLE

Thyroid hormone profile at presentation

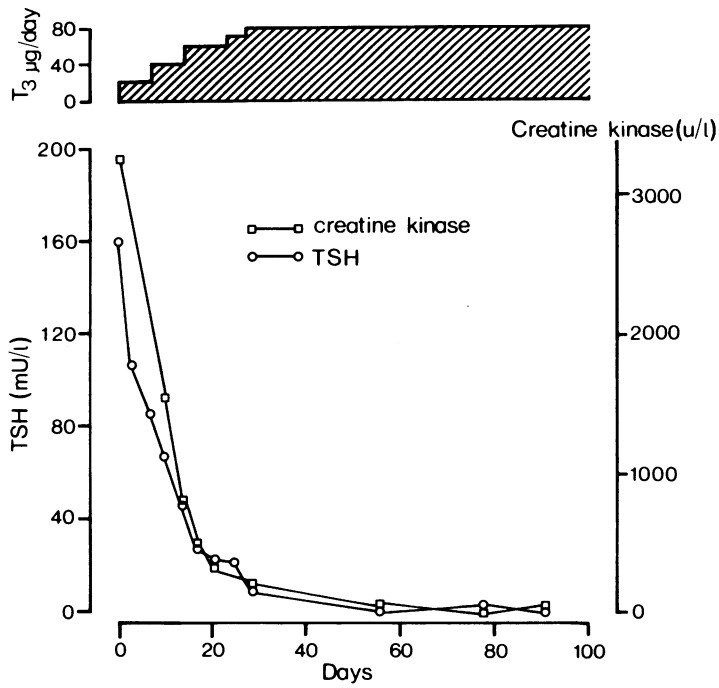
	Result	*Reference range*
Total T_4 (direct)	216	60 – 160 nmol/l
Total T_4 (extracted)	< 20	60 – 160 nmol/l
Total T_3	0.5	1.2 – 3.0 nmol/l
Free T_4 (Amerlex)	163	9 – 28 pmol/l
Free T_4 (2-step)	1	—
Free T_4 (dialysis)	5	9 – 17 pmol/l
TSH—basal	160	< 5 mU/l
—20 min post TRH (200 µg)	205	> 10 < 40 mU/l
—60 min post TRH	230	> 10 < 40 mU/l
TBG	9.7	7 – 17 mg/l
Anti-thyroglobulin Ab	1:320	
Anti-thyroid microsomal Ab	1:10000	

RESULTS

Thyroid function tests on presentation are shown in the Table. The serum total T_4 by the 'in house' radioimmunoassay was moderately elevated and the serum 'free' T_4 by the widely used Amerlex radioimmunoassay was very considerably elevated into the range for severe hyperthyroidism. Nevertheless the basal serum TSH was very high, consistent with primary hypothyroidism. The reason for the conflict in the results is suggested by the low serum total T_3, in the hypothyroid range. The true low values for total T_4 and free T_4 by methods which eliminated interfering factors ultimately confirmed that the patient had primary hypothyroidism. The effect of treatment with triiodothyronine on serum TSH and creatine kinase activity is shown in the Figure opposite.

The patient's serum bound 75.0% of the ^{125}I-T_4 tracer compared with only 4.1% binding by a control serum. It also bound 66.5% of the ^{125}I-T_4 analogue compared with 7.5% binding by control serum. Iodinated T_3 was bound 13.0% by the patient's serum and 11.2% by control serum. These tests were repeated many times but the results remained essentially unchanged throughout the first 100 days of treatment. After electrophoresis 72.2% of the T_4 tracer activity recovered was associated with the γ-globulin fraction of the patient's serum and 13.9% and 10.4% with the TBG and albumin/pre-albumin fractions respectively. The control serum produced values of 1.0% (γ-globulin), 81.2% (TBG) and 15.2% (albumin/pre-albumin). A positive reaction in the ELISA test confirmed the presence of T_4-binding IgG.

The effect of treatment with triiodothyronine on TSH & creatine kinase activity



The association constant of the autoantibodies for T_4 at 4°C calculated by Scatchard analysis was 1.2×10^8 litres/mole and the concentration of binding sites 0.3umol/l. Cross reactivity of the autoantibodies with thyroglobulin was between 200% and 900% of that with thyroxine on a molar basis, the exact figure depending on the incubation conditions. Thyroid microsomal and thyroglobulin autoantibodies were present in titres of 1:10,000 and 1:320 respectively on presentation and had fallen to 1:640 and 1:160 respectively after three months treatment.

DISCUSSION

Thyroid hormone-binding proteins in the γ-globulin region of the electrophoretogram were first described in a patient with papillary carcinoma of the thyroid following radioiodine treatment.[5] They have subsequently been described in euthyroid subjects, patients with autoimmune thyroiditis, Graves' disease, nontoxic nodular goitre and in hypothyroid patients treated with dessicated thyroid extract[6-10] and, rarely, in nonthyroidal illness. The subject has recently been reviewed.[11] The prevalence of thyroid hormone-binding in thyroid screening studies has been variously described as lying within the region 0.05%[12] to 1%.[13] The antibodies are directed against T_3 or T_4 though sometimes both occur in the same patient.[7, 9]

The anomalous thyroid function tests in the present case raised our suspicion of autoantibodies directed against one or both of the thyroid hormones. Laboratory analyses confirmed the presence of IgG antibodies capable of binding T_4 to a degree which was sufficient to explain the spurious results. The double antibody

separation method of RIA used in our laboratory led to a falsely high total T_4. Acid-ethanol extraction of the serum to eliminate interfering factors from the assay system revealed a true serum total T_4 concentration below the lowest standard (<20nmol/l). Similar interference was observed with the thyroxine analogue kit (Amerlex) which purports to measure 'free' T_4. Measurement of free T_4 by such analogue methods has been the subject of much ongoing controversy as to the soundness of the methodological precepts.[14, 15] Measurement of free T_4 by methods not susceptible to the presence of abnormal binding proteins revealed very low levels. The diagnosis of primary hypothyroidism was confirmed by the high TSH and low total T_3 levels, the response to TRH and the clinical improvement produced by treatment with triiodothyronine.

The calculated association constant of the T_4 autoantibody was low compared with other published estimates[7, 8, 9, 16] and its binding capacity was roughly equivalent to that of TBG. Consequently interference in the T_4 assay was relatively slight due to only partial sequestration of the iodinated T_4 used as tracer in the assay. This resulted in an apparently normal or only mildly elevated total T_4 level thereby allaying suspicion that the analytical result was very wrong and putting the clinical diagnosis in doubt. Antibodies of greater avidity and/or titre would have bound virtually all the assay label recording a result of such proportions that suspicion of its validity would have been raised immediately. In other cases[9, 17] the binding characteristics of the antithyroid hormone antibodies were such as to suggest a definitive role for them in the development of the hypothyroid state.

Investigation of the autoantibody in the present case confirmed thyroglobulin as a more specific antigen than T_4. There is a marked correlation between the presence of antithyroglobulin antibodies and antithyroid hormone antibodies[8] often with preferential binding of thyroglobulin to the antithyroid hormone antibodies[16, 18] suggesting a more extensive recognition site. The absence of (a significant titre of) antithyroglobulin antibody has, however, also been recorded[6, 18] but this may be a methodological artefact due to the presence of haemagglutination-inhibiting factors which can occur in autoimmune thyroid disease.[19] The fact that the autoantibodies showed minimal binding to T_3 suggested replacement therapy with triiodothyronine as the treatment of choice. The results of treatment on TSH and creatine kinase activity reveal their sensitivity to even suboptimal doses of T_3. This patient's lymphocytes have now been fused with a mouse myeloma cell line (Dr K Tan, Department of Biochemistry, University of Surrey, Guildford — personal communication) and the resulting hybridomas are producing thyroxine binding antibodies.

This case illustrates the need for further investigation in a patient whose symptoms and signs do not match laboratory results and emphasises TSH measurement as the investigation of choice for suspected primary hypotyhroidism.

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Case report

Acquired immune deficiency syndrome in Northern Ireland

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The first descriptions of the acquired immune deficiency syndrome (AIDS) were from the USA in 1981[1-3] although it is probable the first cases occurred in 1979.[2] In Great Britain the first case occurred in 1981.[4] In both countries there has been an exponential rise in the numbers of cases. The syndrome is defined by the Center for Diseases Control, Atlanta, Georgia, as characterised by opportunistic infections and malignant diseases in patients without a known cause for immunodeficiency.[5] There is now little doubt that the disease is caused by a human T cell lymphotrophic virus type III passed by sexual transmission or parenteral infusion of infected blood products.[6]

The major groups known to be at risk of acquiring the disease in the western world are homosexuals, intravenous drug abusers, haemophiliacs, Haitians, transfusion recipients of infected blood, female sexual partners of affected males and infants of affected mothers.[7] The full syndrome is often preceded by a prodromal illness described as the 'AIDS-related complex' consisting of fatigue, anorexia, weight loss, night sweats and diarrhoea. Examination reveals lymphadenopathy often associated with hepatomegaly or splenomegaly.[7] The commonest disorders seen in AIDS are Kaposi cell sarcoma and pneumocystis carinii pneumonia, although a plethora of opportunistic infections including cytomegalovirus,[2] herpes simplex,[3] candida,[8] mycobacterium[9] and cryptococcus[9] have been described.

CASE HISTORY

A 23-year-old male diabetic homosexual student first presented to another hospital on 10 January 1985 complaining of pyrexia, rigors, anorexia and vomiting for four days. On examination he had oral candidiasis which had resisted treatment for four months, and an abscess in the anterior upper chest wall, which was infected with *Staphylococcus aureus.* Despite treatment of the abscess, initially with flucloxacillin and netilmicin and later drainage and debridement, an ulcer formed which did not heal. During this admission, routine

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biochemical and haematological tests were essentially normal. He was discharged home but readmitted for further investigation on 17 February 1985. The chest wall ulcer had enlarged and he complained of alopecia, malaise, anorexia and weight loss of 3 kg. Following initial investigation, he was transferred to the Royal Victoria Hospital with a persistent chest wall ulcer 5 cm in diameter which penetrated to bony tissue. He was clinically anaemic and had enlarged lymph glands in the right axilla, right cervical area and both groins, but there was no hepatomegaly or splenomegaly. He had oral candidiasis and a tinea cruris infection. During this admission, he had episodes of confusion and his overall intellectual functions were not commensurate with his occupation raising the possibility of early dementia.

In February, his haemoglobin was 8.8 g/dl, white cell count 2,000/μl (65% neutrophils, 34% lymphocytes), platelets 100,000/μl. T-cell subset counts showed a reversal of the T-helper cells (OKT4): T-suppressed cell (OKT8) ratio of 0.33 (normal range 1 – 3.5). Delayed hypersensitivity skin testing with streptodornase, *Candida albicans* and tuberculin demonstrated total anergy. Serological tests to HTLV III were positive.

Staphylococcus aureus (coagulase + ve) was cultured from the chest wall abscess but blood cultures were negative. Serological tests for HB_SAg, cytomegalovirus antibodies and syphilis were negative. *Chlamydia trachoma* cultures of urethra and rectum were negative, as were cultures for *N. gonorrhoea* from throat, urethra and rectum. Chest X-ray was normal. CAT scan of the brain showed early cerebral involution (Fig 1).

The chest wall ulcer was treated with systemic flucloxacillin and sodium fusidate, the oral candidiasis with nystatin drops and the tinea cruris with topical miconazole. There was gradual improvement in symptoms and signs despite frequent insulin hypoglycaemic episodes and he was discharged home after three weeks. Over the next four weeks the skin ulcer improved but later worsened as did his oral candidiasis. There was marked deterioration in his mental state with episodes of paranoia and confusion. Psychiatric assessment confirmed the increasing dementia and electroencephalography was consistent with cerebral atrophy.

He was readmitted to hospital on 12 August for lumbar puncture and repeat CAT scan because of progressive dementia. The enlarged lymph nodes, oral

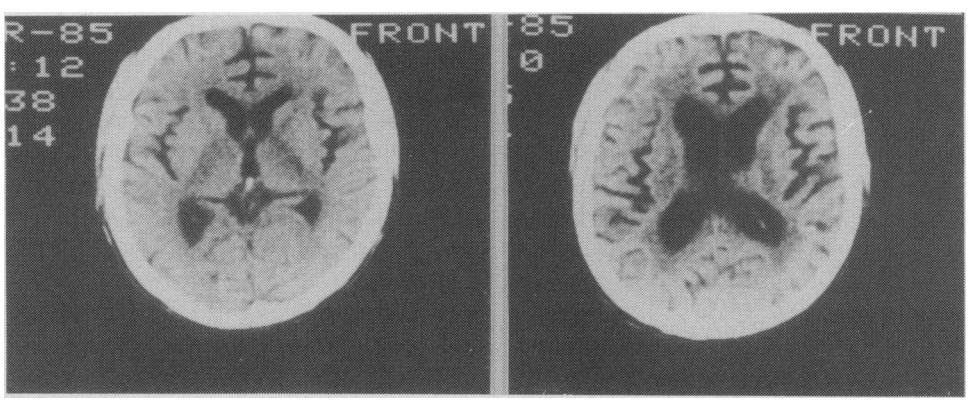

Fig 1. CAT scan showing progression of cerebral involution over a six month period.

candidiasis and chest wall ulcer persisted. CAT scan of the brain now showed dilatation of all the ventricles with marked prominence of the cortical subarachnoid spaces, basal cisterns and sylvian fissures, typical of severe generalised cerebral involution. The cerebrospinal fluid showed no increase of cells or protein, and antibody to HTLV III was not detected. Two days after admission to hospital he developed a temperature of 38.5°C with tachycardia (110/min) and tachypnoea (42/min) but with no other abnormal signs in the respiratory system. Chest X-ray revealed an atypical pneumonia consistent with that seen in pneumocystis carinii (Fig 2). He was treated with co-trimoxazole 2.4g twice daily. Bronchial biopsy was attempted using fibreoptic bronchoscopy, but a poor specimen was obtained which did not reveal pneumocystis carinii, and culture for cytomegalovirus was negative. His condition rapidly deteriorated and he died within 48 hours. No post-mortem was performed.

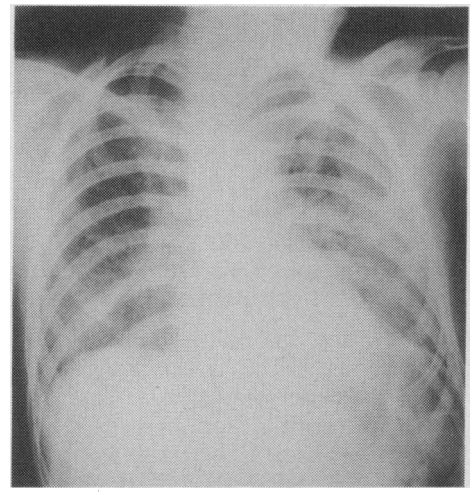

Fig 2. Chest X-ray showing areas of consolidation consistent with an atypical pneumonia 2 days before death.

DISCUSSION

Although we did not make a definitive bacteriological diagnosis of the terminal pulmonary infection, there is enough evidence with the immunocompromised state, the positive HTLV III serology, the persistent chest wall ulcer and the oral candidiasis in a homosexual male to make a diagnosis of AIDS. One of the most distressing features of this condition is its association with progressive dementia associated with cerebral involution which can be demonstrated by CAT scanning. This is probably due to destruction of cells in the cerebral cortex by infection with the virus, and the virus has been demonstrated in the cerebrospinal fluid of patients with this problem.[10] It has been suggested recently that the titre of antibody in sera of patients with AIDS declines with increasing severity of the condition[11] and it may be that the inability to demonstrate HTLV III in the cerebrospinal fluid in our patient was also a consequence of the terminal stage of the illness. While it appears that HTLV III can infect brain cells and replicate within them, it is not yet clear whether the neurological symptoms in AIDS are a direct consequence of the viral infection or whether other factors are important.

Although this is the first case of AIDS described in Northern Ireland, experience elsewhere in the world can only lead to the conclusion that others will follow in the near future and, as such a case may present itself to any medical practitioner, we must all be aware of the possibility of this diagnosis today.

We are grateful to our laboratory colleagues, and in particular to the immunology and virology departments for their assistance with the treatment of this patient. We are grateful also to Mrs Marie Loughran for typing the manuscript.

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Case report

Psychiatric disorder associated with fear of AIDS

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Accepted 13 February 1986.

The condition known as the acquired immune deficiency syndrome (AIDS) was initially described at the Center for Disease Control, Atlanta, USA,[1, 2] and the first case of AIDS in the United Kingdom was reported in 1981.[3] At the time of writing, only one established case has been reported in Northern Ireland.[4] In the absence of effective prevention and treatment, it seems certain that there will be an increased prevalence of the disease for the foreseeable future.

Despite a rational approach by the medical profession, fear of AIDS has spread through the affected societies with an 'AIDS panic' syndrome described by Schwartz in risk groups.[5] This is described as 'demanding AIDS testing at the first appearance of some cutaneous lesion or persistent cough' and is said to be more common in individuals with obsessional or paranoid personality traits. Case reports have been appearing in the literature in recent months of psychiatric symptoms associated with a fear of AIDS.[6, 7] Miller et al. describe two cases,[6] one with depressive features, the other with anxiety symptoms, and they suggest that such symptoms, arising from a fear of AIDS, might be referred to as a 'Pseudo-AIDS Syndrome'. Here I describe a recent case of psychiatric illness where the patient's concern about AIDS is a prominent feature of the disorder.

CASE HISTORY

A 26-year-old married man presented with a five-week history of persistently depressed mood accompanied by marked feelings of guilt and suicidal thoughts. There were also symptoms of anorexia, weight loss, fatigue, early morning wakening and loss of interest in his usual activities. His symptoms appeared to have been precipitated by an uncharacteristic episode of infidelity while he was in England at a sports event. He knew the girl shared a flat with a homosexual male and, beginning to fear he had contracted AIDS, made several visits to the venereology clinic, on each occasion seeking reassurance. By the time he presented at the psychiatric clinic, his belief that he had AIDS was of delusional intensity. His anorexia, weight loss and fatigue were confirmation to him that he had the disease. He was treated with psychotherapy and a tricyclic anti-depressant, and over a period of three to four months his symptoms, including the delusion, gradually resolved.

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DISCUSSION

The case reported is that of a typical depressive psychosis and fulfils the diagnostic criteria for major depressive illness according to the DSM-111,[8] (as does the case described by Miller et al[6]). Delusions of ill health, according to Fish's *Clinical psychopathology,*[9] 'are a characteristic feature of depressive illness' and 'depressives with hypochondriacal delusions believe they have some incurable disease such as cancer, TB, syphilis'. Since depressive illness characterised by over-valued or delusional ideas of syphilis or cancer are not referred to as 'pseudo-syphilis' or 'pseudo-cancer', it seems unnecessary to introduce the term 'pseudo-AIDS' into the nosology at this time. Furthermore, some of the symptoms of the AIDS-related complex[10] are similar to the somatic components of depression and anxiety, and the diagnosis of the physical disorder, where it actually exists, might well be prejudiced.

In conclusion, the term 'pseudo-AIDS' should not be used to describe a psychiatric disorder in which the patient either fears AIDS or believes he has AIDS, since the term has little validity as a diagnostic entity. This has important implications for the future, as patients with psychiatric disorders may be more likely to present with symptoms concerning AIDS. The primary psychiatric disorder, of which the symptom is a feature, should be treated on its own merits following a comprehensive psychiatric examination.

My thanks to Dr P E Potter, Consultant Psychiatrist, Holywell and Whiteabbey Hospitals, for permission to report this case.

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Case report

Fulminant fat embolism associated with closed fracture reduction

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Accepted 3 February 1986.

Fulminant fat embolism is well recognised although uncommon. This case report of a patient developing pulmonary oedema during surgery and anaesthesia and dying four hours later is rare, unusual and disturbing. Two similar but non-fatal cases, both young males, of intraoperative pulmonary oedema associated with closed fracture reductions, where fat embolism was implicated, have been previously reported in the literature.[1]

CASE HISTORY

A 60-year-old woman was admitted following a road traffic accident, having sustained a fracture of the right tibia and fibula, a compound fracture of the right olecranon, a fractured right clavicle and fractures of the seventh, eighth and ninth ribs on the right side. She was conscious and fully orientated with a blood pressure of 120/60 mmHg and a pulse rate of 60/min. Chest X-ray showed a contused right upper lobe.

Three hours later she was referred for anaesthesia for reduction of her compound limb fractures. Pre-operative examination showed her to be drowsy and pale with cold cyanosed peripheries. There had been persistent oozing from her fracture sites since admission, with an estimated blood loss of 1.5 litres. Her haemodynamic status had deteriorated, with a blood pressure of 100/60 and a pulse rate of 96/min. Anaesthesia was deferred pending fluid replacement with 500 ml of gelatin solution (Haemacell), 840 ml blood and one litre of compound lactate solution over three hours while central venous pressure was monitored. Supplementary 40% oxygen by mask and 500 mg hydrocortisone intravenously was also given. Her clinical state improved but urinary output remained poor.

At this stage she was considered sufficiently improved for reduction of her badly displaced fractures. Anaesthesia was induced uneventfully with 2 ml intravenously of a mixture of alphaxalone and alphadolone (Althesin), and maintained by spontaneous ventilation of 50% nitrous oxide in oxygen via a face mask with intravenous increments of the induction mixture given as required. Throughout

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the 45-minute procedure, she was haemodynamically stable until the final 10 minutes when she suddenly became centrally cyanosed and tachypnoeic, even though the inspired oxygen concentration had been increased to 100%. Although the continuous ECG record and blood pressure were normal, the heart rate had risen to 120/min. A tracheal tube was inserted and pink frothy sputum was aspirated. There was no evidence of gastric contents on endotracheal suction, the trachea was centrally located and on auscultation diffuse bilateral pulmonary crepitations were audible.

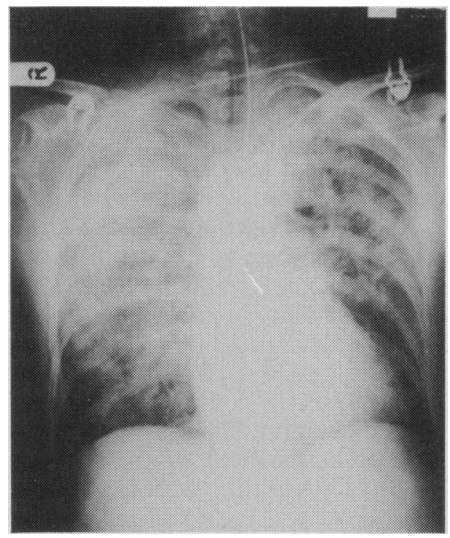

Fig 1. Post-operative chest film showing extensive 'snow-storm' infiltrates over both lung fields.

Post-operatively she remained cyanosed. Controlled ventilation with 100% oxygen was started and 80 mg frusemide and 1 g hydrocortisone were given intravenously. Chest X-ray (Fig 1) showed extensive bilateral pulmonary infiltrates consistent with pulmonary oedema. The differential diagnosis included aspiration pneumonitis, fat embolism syndrome, fluid overload, a severe hypersensitivity reaction to the induction mixture or acute cardiac failure secondary to possible cardiac contusion. Arterial blood gas analysis revealed gross hypoxaemia, PaO_2 3.8 kPa (normal range 12.6 – 13.3 kPa), PCO_2 5.23 kPa (4.5 – 6.1 kPa), pH 7.23 (7.36 – 7.44). Central venous pressure had risen from 9 cm H_2O pre-operatively to 14 cm H_2O. Urine output, scanty throughout, had now stopped. Peak inflation pressure was 25 cm H_2O and a positive end-expiratory pressure of 10 cm H_2O was applied without any improvement in oxygenation. Despite supportive treatment the patient had repeated episodes of asystole and died four hours post-operatively.

At autopsy, both lungs were grossly oedematous. Microscopic examination revealed massive pulmonary fat embolism (Fig 2) with the alveolar walls clearly outlined by fat-laden capillaries. Early reactive changes and interstitial oedema were also evident. There was also extensive deposition of fat in myocardial, renal glomerular and cerebral capillaries.

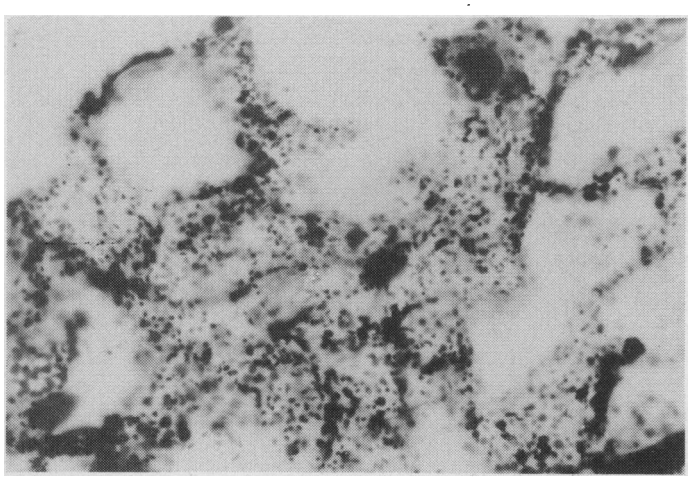

Fig 2. Histological section of lung showing fat emboli (black) in vessels of the alveolar walls, and early reactive changes.

DISCUSSION

Fulminant fat embolism appears to be an accelerated version of the classical fat embolism syndrome, differing only in severity and rapidity of onset. It occurs within a few hours of injury and is rapidly progressive with a fatal outcome in 90% of cases.[2] It can present with pulmonary hypertension, acute cor pulmonale, shock and sudden respiratory failure. The diagnosis is usually confirmed at autopsy.

In our patient, fat embolism was not initially suspected: the first arterial blood gas analysis was performed only after the clinical signs of hypoxaemia became apparent. The sudden onset of intra-operative cyanosis and pulmonary oedema in our patient supports the view that closed fracture reduction can produce repeated showers of fat emboli from traumatised tissues.[3] The filtering capacity of the lungs and the compensatory ability of the pulmonary circulation especially by capillary recruitment are normally very effective, so that moderate degrees of occlusion only produce a slight rise in pulmonary vascular resistance.[4] However, in a patient already compromised by fat embolism, additional fat emboli released by operative manipulations can overwhelm the compensatory capacity of the lungs, resulting in sharp rises in pulmonary vascular resistance and right ventricular pressure.[5] While operative interference in the presence of fat embolism exacerbates the syndrome, other studies have shown that early effective immobilisation can prevent fat embolism.[6] Herein lies the central dilemma. In retrospect it might have been wiser to delay surgery for at least 24 hours to allow further assessment of the patient in view of her initial hypovolaemia, lung contusion and rib fractures.

We would emphasise that a high index of suspicion for fat embolism and careful patient observation, including serial arterial blood gas analysis, is important in all severe trauma cases before operative intervention should be contemplated.

The authors would like to extend their thanks to Mr J Dempsey and Dr D Carson for preparation of the X-ray and histopathological photographs and to Dr D L Coppel and Professor J W Dundee for their advice and encouragement.

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Case report

Use of digoxin-specific antibody fragments (Fab) in the management of digoxin poisoning

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Accepted 26 February 1986.

Massive digoxin overdosage is an uncommon but serious medical emergency with a mortality of 20%.[1] It involves multiple management problems including hyperkalaemia, heart block and resistant ventricular arrhythmias. The management of the problems associated with severe digoxin overdosage has been assisted by two recent therapeutic advances, the use of digoxin-specific antibody fragments (Fab) to reverse toxicity,[2-4] and the relatively new Vaughan-Williams Class III antiarrhythmic, amiodarone, which has been of value in controlling the arrhythmias.[4,5] We report a case of severe digoxin poisoning in which both these agents were employed with a favourable outcome.

CASE HISTORY

A 41-year-old schizophrenic woman, who had been under treatment for a suspected cardiomyopathy, was admitted to hospital following the ingestion, in a suicidal attempt, of approximately one hundred 0.25 mg tablets of digoxin and a small quantity of thioridazine. Forced emesis and gastric lavage were performed less than three hours after the patient claimed she had ingested the tablets, but none were recovered. Serum digoxin concentrations subsequently became available and revealed a level of 22 μg/l on admission (therapeutic range 1 – 2 μg/l), rising to 32 μg/l twelve hours later.

On initial examination the patient was alert and orientated, with a pulse rate of 60/minute and a blood pressure of 100/60 mmHg, which subsequently fell to 80/60 mmHg. Cardiac monitoring showed variable atrio-ventricular block developing with an atrial rate of 120/minute and a ventricular rate ranging from 35 to 120/minute. The typical ST segment changes attributable to digoxin were also present. A bipolar transvenous ventricular pacing wire was inserted and connected to an external pacemaker (Devices Limited, England). Satisfactory pacing was achieved with an output of 3 volts, rate 80/minute on demand setting. Systolic blood pressure remained low at 80 mmHg despite dobutamine infusion, but a good urinary output was maintained.

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The serum potassium concentration on admission was 5.0 mmol/l but four hours later this had risen to 6.4 mmol/l and an intravenous bolus of 50ml of 50% dextrose and 10 units of monocomponent porcine insulin ('Actrapid', Novo) was administered. Hyperkalaemia persisted, the serum potassium concentration reaching a peak value of 7.3 mmol/l, but was successfully controlled by dextrose/insulin therapy, a total of ten doses being administered over a 40 hour period. (Fig 1).

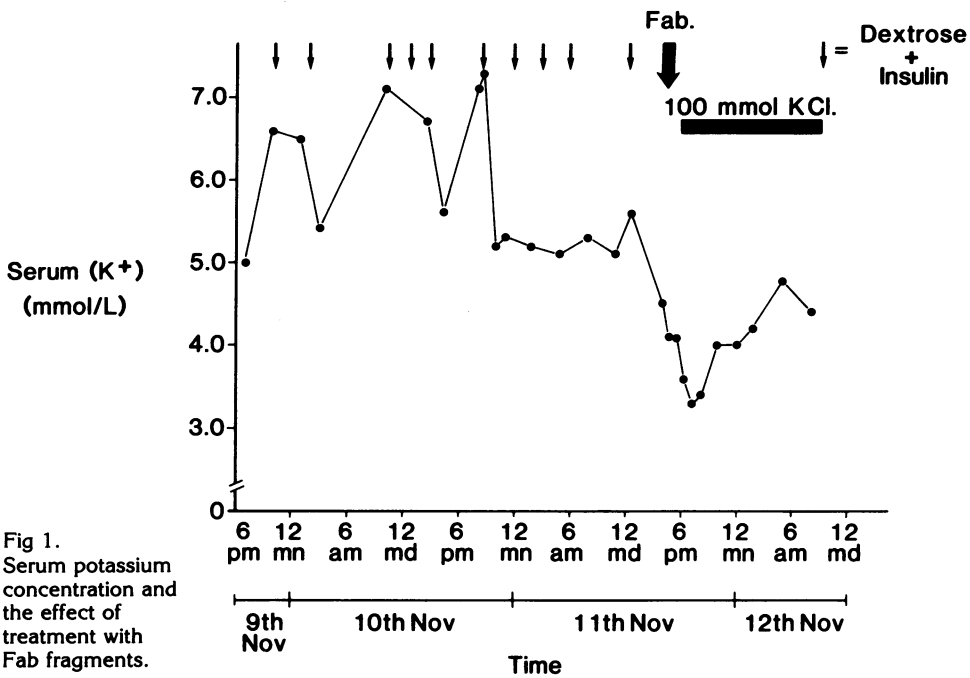

Fig 1.
Serum potassium concentration and the effect of treatment with Fab fragments.

Thirty-six hours after admission the first episode of ventricular fibrillation occurred — there had been no premonitary arrhythmias — and proved resistant to conventional therapy with DC conversion using energy levels up to 400J, both alone and following intravenous sodium bicarbonate and lignocaine. Six unsuccessful defibrillation attempts were made, but paced rhythm, with cardiac output, was eventually restored by a seventh 400J DC shock preceded by 300mg amiodarone intravenously. Continuous infusion of amiodarone 0.4mg/min was then commenced. The patient remained unconscious but respiration was spontaneous. Despite the amiodarone infusion, ventricular fibrillation recurred three hours later. On this occasion five 400J DC shocks, sodium bicarbonate and a further 150mg bolus of amiodarone were required to convert to paced rhythm. An hour later the patient arrested a third time and was even more difficult to resuscitate, a further 300mg bolus of amiodarone and twelve 400J DC shocks being required. After this arrest the infusion rate was increased to 0.6mg/minute and intermittent positive pressure ventilation instituted as spontaneous respiration had not returned.

Following the first episode of ventricular fibrillation, arrangements were made to obtain digoxin-specific antibody fragments (Fab) from the National Poisons Information Service, New Cross Hospital, London.[2] Four hours after the third

cardiac arrest, 320mg of these fragments were infused over a period of 40 minutes. The dose (to the nearest 40mg) was calculated using the following formula involving the weight of the patient and the most recent serum digoxin concentration: dose = 60 × 0.0056 × body weight (kg) × CPSS digoxin (g/l), (CPSS = concentration in plasma at steady state, more than 6 hours post ingestion). Within an hour of starting the infusion there was a dramatic improvement in both electrical and clinical parameters: the ECG returned to sinus rhythm with a normal PR interval, so that demand pacing was no longer needed. Her level of consciousness improved and the onset of spontaneous respiration permitted removal of the endotracheal tube. During the next few hours, serum potassium concentration fell to 3.3 mmol/l. An intravenous infusion of potassium chloride was instituted and a total of 100 mmol potassium chloride administered over the subsequent 15 hours. Twenty-four hours later, transient Wenckebach-type second degree heart block was noted; this responded to intravenous atropine. The effect of the Fab fragment infusion on the total and free serum digoxin levels is illustrated in Fig 2. Over the next few days the patient continued to improve and was able to return to psychiatric care within a week of admission.

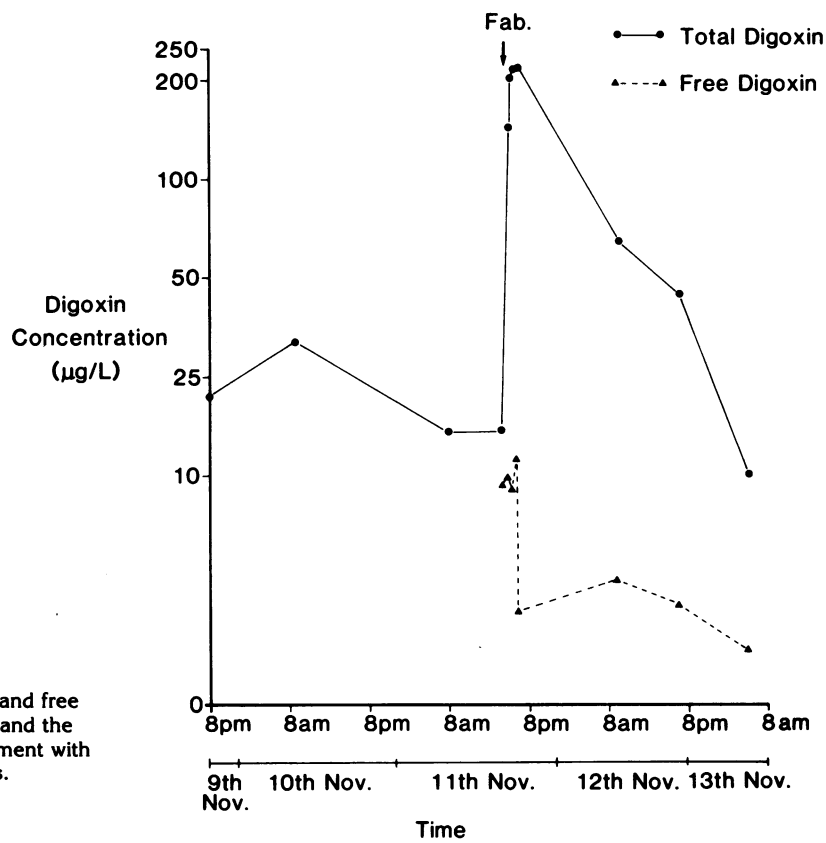

Fig 2. Total and free digoxin levels and the effect of treatment with Fab fragments.

DISCUSSION

Digoxin-specific antibody fragments remove digoxin from tissue binding sites and combine with circulating free digoxin.[2] The pharmacologically inactive complex of Fab and digoxin has a more rapid rate of excretion than the drug

alone, but more importantly the toxic effect of digoxin on the myocardial cell is rapidly removed.[6] The resultant effect is well illustrated by the total and free digoxin levels.

Amiodarone has proved beneficial in the management of digoxin-induced arrhythmias, where conventional agents such as lignocaine, phenytoin and procainamide and beta-adrenergic blocking drugs have been ineffective[4,5] though we felt the latter would have been inappropriate in the presence of atrioventricular block. The mode of action of amiodarone, and the reason for its efficacy in treating these arrhythmias is uncertain. It has been shown that amiodarone increases the serum digoxin concentration, possibly by displacing digoxin from myocardial binding sites.[7]

Hyperkalaemia occurs in massive digoxin poisoning as a result of inhibition of the sodium-potassium activated adenosine triphosphatase (Na-K ATPase) pump in the cell membrane which is responsible for maintaining a high intracellular level of potassium.[8] It has been shown that the development of significant hyperkalaemia in digoxin poisoning is a bad prognostic indicator,[9] and usually occurs in cases where the digoxin level is particularly high, although a direct correlation between digoxin concentration and potassium concentration has not been shown. Even in the presence of hyperkalaemia there is usually a total body deficit of potassium; hence, following treatment with Fab fragments, the reactivation of the Na-K ATPase pump often results in a significant hypokalaemia requiring replacement therapy.[2]

We report this case to illustrate the successful treatment with amiodarone of ventricular fibrillation occurring in severe digoxin poisoning, and we think that this antiarrhythmic should be employed as the drug of choice. Where cardiac arrhythmia and hyperkalaemia are resistant to conventional therapy, digoxin-specific antibody fragments are an extremely effective and in many cases life-saving therapeutic intervention.

We would like to acknowledge the contribution of the nursing and technical staff of the Mater Infirmorum Hospital, Belfast; we would also like to thank Dr David Holt of the National Poisons Unit for performing the digoxin assays and Mrs P Finn for typing the manuscript.

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Book reviews

The growth of a hospital, 1838 to 1948: Belfast and its Infirmary. By D H Craig. (pp vii, 99. Illustrated. £15.00). Belfast: Brough, Cox & Dunn, 1985.

In 1973 the author, in his presidential address to the Ulster Medical Society, and under the title *A history of the Belfast City Hospital,* gave a well researched and absorbingly interesting account of that hospital from its planning in 1838 to the beginning of this century (*Ulster Med J* 1974; **43**: 1-14). The narrative was skilfully interwoven with the social history of Belfast, since the hospital had throughout its existence to meet the many challenges of the poverty, hardship and rampant diseases of the time.

Mr Craig has continued his researches through the old minute books of the Poor Law Guardians and other documents, and this book contains much new material in a fresh and well organised form. It brings the record from the beginning up to 1948 and the advent of the Northern Ireland Hospitals Authority.

This is an important historical work and should be read by all interested in our town and the way in which thousands of our forefathers, driven by need and hardship, crowded into a booming industrial area, lived in appalling housing and suffered and died, often in thousands, in epidemics. It narrates how the Poor Law Guardians behaved, perhaps reasonably well and compassionately by the harsh standards of the time. It shows how a small number of our profession struggled to treat many seriously ill and dying patients in grossly overcrowded and insanitary wards and plagued by a chronic water shortage. It describes how nursing services, developed from the crude help available from workhouse inmates and through the often unselfish efforts of grossly overworked and underpaid nurses and attendants, managed to emerge with trained staff and a modern school of nursing. The human failings and shortcomings of many are recorded, but on the whole this is a heartening record of achievement.

The hospital story begins when the Guardians went beyond their remit and installed six beds for sick inmates in the workhouse. These were soon increased to over a hundred. Ultimately the hospital became one of the largest in the Kingdom. Dr Thomas Andrews, later to become the distinguished chemist and a Fellow of the Royal Society, was the first physician. Destitute people from the countryside and paupers returned by authorities in Scotland and England brought epidemics of typhus, relapsing fever and cholera and one in five of the population died. The workhouse infirmary shared these problems with the other hospitals of the town well into the mid-century.

In 1861 a new Poor Law allowed poor people, as distinct from destitute persons, to be admitted to the hospital. Smallpox became a relatively more important problem, and, as the century advanced, chest infections and tuberculosis, both products of overcrowding and poor working conditions, made increasing demands. The excessively high incidence of typhoid fever in Belfast around the turn of the century, and the related appalling housing and defective water supply are well described.

In this century conditions improved slowly. The heavy workload in surgery sustained by Dr Joseph Fulton is acknowledged, and there is a delightful account of his colourful personality. After the First World War, epidemics of measles and scarlet fever were followed by the more serious epidemics of influenza and acute encephalitis lethargica (sleepy sickness). A graphic description of the latter is given with what must be a firsthand account of the behaviour of survivors who remained convalescent in the hospital until 1948. Chapters record problems with mental disease and epileptics and with children in the care of the Guardians. In 1880 a quarter of those under the age of 12 died, but the Government Inspector considered this in no marked contrast with other large towns. Well balanced chapters on the development of the nursing services and on the struggles to admit medical students precede the final chapter. This describes briefly the appointment of specialists to the visiting staff and progress to a modern hospital.

The book is well illustrated with photographs of the other buildings and the new hospital and of many of the visiting medical staff over the years. The presentation, printing and binding are a credit to the printers. All who are interested in the history of this town and, especially, all interested in medical history here and elsewhere, must welcome without reservation this valuable book. JEM

Complications of total joint replacement. By Neville R M Kay. (pp 265. Illustrated. £18.50). London: Baillière Tindall, 1985. (Complications in surgery series).

This is the latest of a series of publications on the complications of surgery. The author is an orthopaedic surgeon in Sheffield who has been writing on complications of joint replacement since 1973. This topic is the subject of an increasing number of publications in keeping with the increase in

The Ulster Medical Journal, Volume 55, No. 1, pp. 93 – 96, April 1986.

Book reviews

The growth of a hospital, 1838 to 1948: Belfast and its Infirmary. By D H Craig. (pp vii, 99. Illustrated. £15.00). Belfast: Brough, Cox & Dunn, 1985.

In 1973 the author, in his presidential address to the Ulster Medical Society, and under the title *A history of the Belfast City Hospital,* gave a well researched and absorbingly interesting account of that hospital from its planning in 1838 to the beginning of this century (*Ulster Med J* 1974; **43**: 1-14). The narrative was skilfully interwoven with the social history of Belfast, since the hospital had throughout its existence to meet the many challenges of the poverty, hardship and rampant diseases of the time.

Mr Craig has continued his researches through the old minute books of the Poor Law Guardians and other documents, and this book contains much new material in a fresh and well organised form. It brings the record from the beginning up to 1948 and the advent of the Northern Ireland Hospitals Authority.

This is an important historical work and should be read by all interested in our town and the way in which thousands of our forefathers, driven by need and hardship, crowded into a booming industrial area, lived in appalling housing and suffered and died, often in thousands, in epidemics. It narrates how the Poor Law Guardians behaved, perhaps reasonably well and compassionately by the harsh standards of the time. It shows how a small number of our profession struggled to treat many seriously ill and dying patients in grossly overcrowded and insanitary wards and plagued by a chronic water shortage. It describes how nursing services, developed from the crude help available from workhouse inmates and through the often unselfish efforts of grossly overworked and underpaid nurses and attendants, managed to emerge with trained staff and a modern school of nursing. The human failings and shortcomings of many are recorded, but on the whole this is a heartening record of achievement.

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sacrificed a lot of his leisure time to the preparation of this book and it deserves to become the logical successor to *Physics for the anaesthetist* by Sir Robert McIntosh. I can highly recommend it to anaesthetists of all grades, theatre and intensive care technicians and nurses. DLC

Orthopaedics and trauma. Edited by S P F Hughes. (pp 118. Illustrated. £19.95). London: Baillière Tindall, 1985. (Current operative surgery).

It is a pleasure to receive this book for review and I cannot imagine that any orthopaedic specialist would think otherwise.

Professor Hughes and the publishers have amply fulfilled their remit in selecting, from established experts in their fields, a wide spectrum in current orthopaedic operative technology. That super-specialisation is inevitable is well demonstrated and it would be virtually impossible for any reader to evaluate each and every chapter critically from personal experience. Some chapters may even incur frank hostility!

That each team of orthopaedic surgeons (and we do work in teams) will require a copy is certain. It will be simply devoured by those in training with an appetite for further knowledge and innovative technology. The layout and standard of production is excellent, with a slight lapse in proof-reading at the word 'neoplasm'. The diagrams relating to operative technique are exceptionally clear and pleasing, the radiographs only occasionally indistinct, and clinical photographs are restricted, as (though restful to readers) they are seldom instructive. This excellent and varied collection of specialist articles is well worth the price and will find its way on to many individual bookshelves and into every departmental orthopaedic reading room — my own copy is already on its way there. PO

Blood transfusion and blood banking. Guest editor, William L Bayer. (pp 306. £13.75). London: Saunders, 1984. (Clinics in haematology, vol 13, no 1).

During the past few years there has been a very marked change in the pattern of blood transfusion therapy. Thus, whereas requirements for red cells have remained fairly constant, the usage of various blood components has increased enormously. The many developments in blood component therapy are well reflected in this volume of *Clinics in haematology.* The book comprises a series of review articles, mostly of North American authorship but also including a substantial input from the U.K. Most of the articles are directed towards haematologists and transfusion specialists, so that transfusion support in haematological malignancies, congenital coagulation disorders and haemolytic anaemias are each very fully covered by well established experts. Major omissions include the management of haemorrhage, acquired coagulation disorders and transfusion support of intensive care patients. Some parts will be of interest outside haematology and in this respect I particularly enjoyed the chapter on the clinical use of immunoglobulins. This growing field is very well reviewed by workers from Edinburgh who are actively involved in the development as well as clinical assessment of new immunoglobulins.

Another rapidly expanding area of transfusion medicine involves the use of apheresis techniques for therapeutic purposes and also for collection of blood components from donors. Both aspects of apheresis are discussed very fully in separate chapters, each extensively referenced. Description of the side effects of transfusion therapy is limited to one chapter on transfusion-related infections. The latter is disappointing because, while transfusion transmitted cytomegalovirus infection is discussed at great length, reflecting the interests of the authors, the more important areas of AIDS and particularly non-A non-B hepatitis are dealt with rather sketchily. In the case of AIDS, much of the information is unavoidably obsolete, having been written before the discovery of HTLV III as the causative agent.

WMMcC

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