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THE ULSTER MEDICAL JOURNAL

[continued on back cover

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The Ulster Medical Journal, Volume 57, No. 1. April 1988

Contents continued from front cover

Maxepa®
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References: 1. Bang HO, Dyerberg J. Adv Nutr Res 1980; 3: 1–22. 2. Saynor R, Gillott T, Doyle T, *et al.* Prog Lipid Res 1986; 25: 211–217. 3. Nestel PJ. Am J Clin Nutr 1986; 43: 752–757. 4. Dyerberg J, Bang HO, Stoffersen E, Moncada S, Vane JR. The Lancet 15 July 1978; 2: 118–119. 5. Saynor R, Verel D, Gillott T. Atherosclerosis 1984; 50: 3–10.

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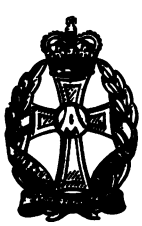
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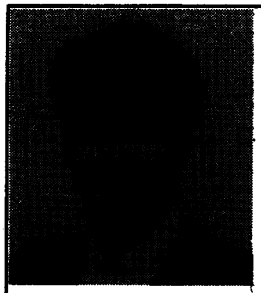

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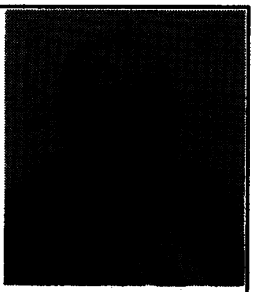

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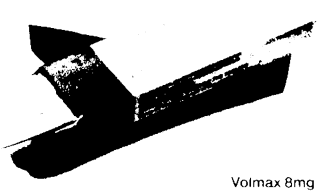

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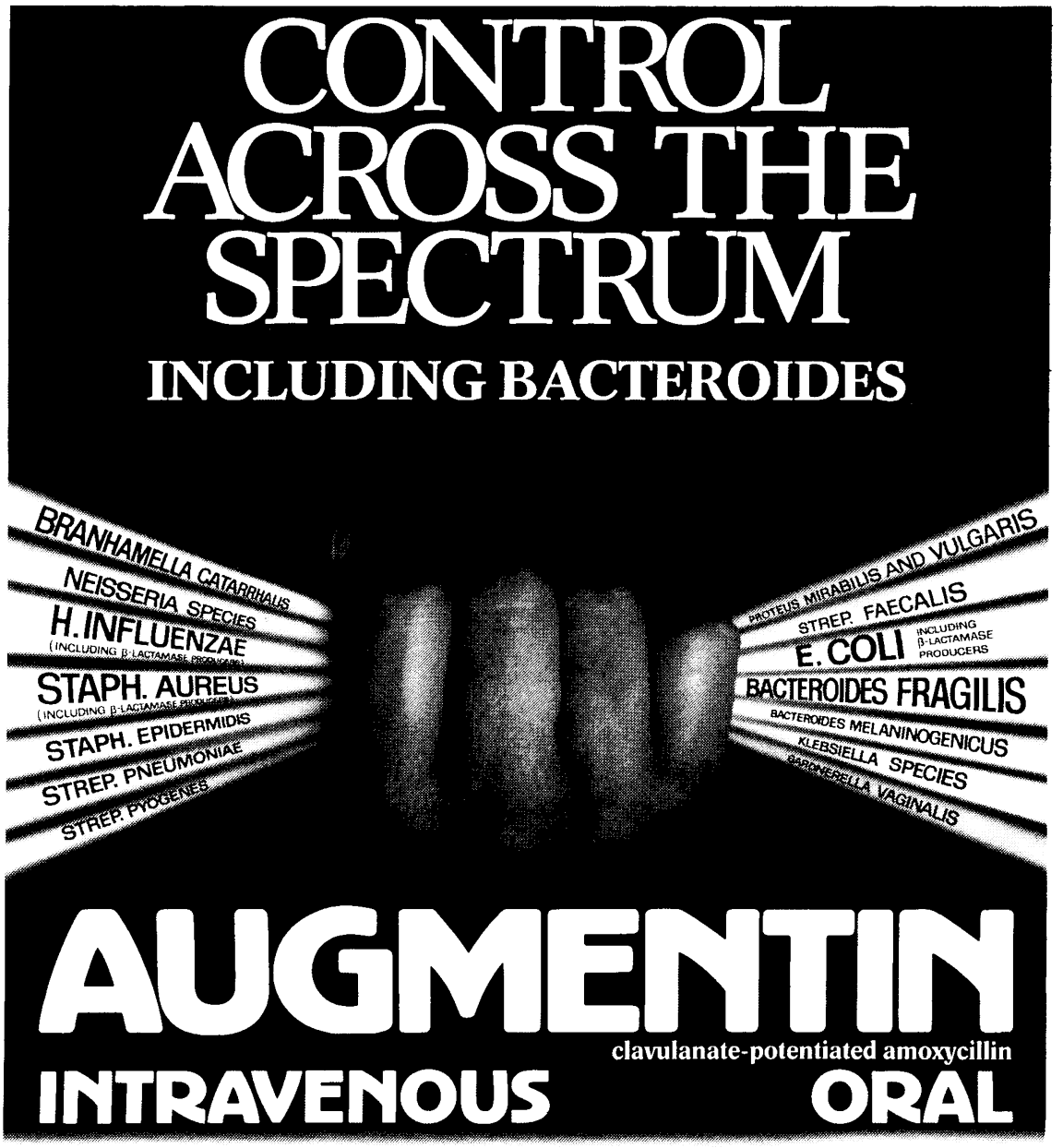

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THE ULSTER MEDICAL SOCIETY

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Penetrating head injuries

Presidential Address given to the Ulster Medical Society, 22 October 1987

D S Gordon

The story begins long before the dawn of recorded history, at the time when man learned to use sticks and stones as weapons. We shall trace the injuries caused by weapons, which became increasingly more deadly from the Stone Age, through the ancient civilisations of Egypt and Greece to modern times.

What do we mean by a penetrating wound of the head? There are three kinds of head injury. First, the blunt injury, often caused by a fall on a flat surface, which produces generalised brain damage without an open brain lesion. Secondly, the comminuted depressed fracture of the skull caused by a falling object such as a brick: indriven bone fragments may damage the cerebral cortex but the patient seldom loses consciousness and usually recovers without serious handicap. Thirdly, the penetrating head injury, where a sharp object or missile fractures the skull and enters the brain, driving fragments of bone before it. The spears and arrows of earlier civilisations frequently penetrated the brain. Nowadays most penetrating injuries are sustained in road traffic accidents or in assaults with sharp weapons; but the most severe penetrating wounds are caused by bullets or bomb fragments.

PRE-HISTORY

Our knowledge of the earliest penetrating wounds is derived from archaeological sources. Little trace remains of most ancient operations, but preserved skulls from the Neolithic Age provide clear evidence of surgical treatment of skull fractures. Pieces of comminuted bone were elevated, sometimes by making a hole beside the fracture not unlike the modern surgeon's burr hole. Admittedly, we don't know the depth of these wounds or the extent of brain damage, but new growth of bone at the edge of the defect shows that some patients survived the operation. The head was bandaged and occasionally the defect was repaired with a piece of shell, or even a sheet of gold.

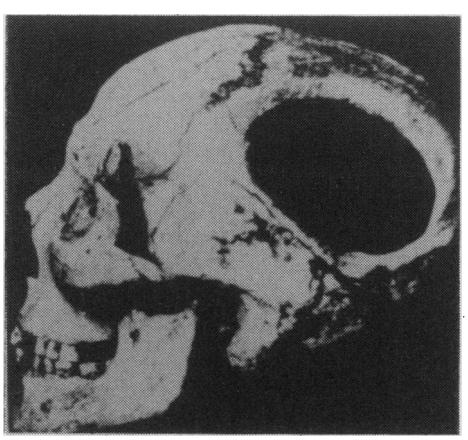

Fig 1. A trepanned skull. (From *A history of neurological surgery*; ed. A Earl Walker. New York: Hafner, 1951).

A greater number of pre-historic skulls have defects from the operation known as trepanning or trephining.[1] The deliberate fashioning of a hole in the normal skull was carried out in many parts of the world. In Neolithic times a flint or obsidian knife was used; in the Bronze Age more sophisticated

D S Gordon, OBE, MCh, FRCS, Consultant Neurosurgeon, Royal Victoria Hospital, Belfast BT12 6BA.

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instruments appeared and the holes in the skull reached several inches in diameter. The finding of new bone growth at the edges proves that the defect was not the result of disease or post mortem damage. We don't know how or why trepanning of the skull developed; perhaps the early doctors believed insanity or epilepsy might be helped by the operation. It did not die out in the Bronze Age and indeed was recorded in the early twentieth century in Albania and Bolivia. No trace of this apparently ritualistic practice has been found in Egypt and Mesopotamia, the countries in which civilisation first appeared.

THE EARLY CIVILISATIONS

Several times in his history, man has made a rapid, inexplicable leap forwards. The first great leap was about 3000 BC in Mesopotamia and Egypt, another was the fifth century BC in Greece, another a few hundred years later in Rome. The Renaissance period, and the scientific revolution of the nineteenth and twentieth centuries are further examples. What has this to do with head injuries? I would suggest that in most of these periods of change we can trace an advance in medicine in general and sometimes in the treatment of head injuries in particular.

The first documentary record of surgical practice comes from Egypt, the Edwin Smith papyrus named after the explorer and archaeologist who discovered it in the nineteenth century.[2] Imhotep, physician to the Pharaoh Yoser about 3000 BC probably wrote the document, which is almost entirely devoted to neurosurgery and reveals the firm grasp the Egyptians had of neuroanatomy, the scope of head injuries and the significance of their complications. It illustrates the management of 48 patients who had head or spinal injuries. A laceration of the scalp with an underlying simple fracture was to have an application of fresh meat to the wound on the first day and thereafter dressings with honey and lint till it recovered. The Egyptians appreciated the importance of the dura mater: a depressed fracture with an intact dura mater was to be treated. However, a patient with a wound which penetrated the dura leading to a discharging laceration of the brain was regarded as hopeless — 'Thou should say of him, an ailment not to be treated'. In spite of their considerable knowledge and experience, operative neurosurgery does not seem to have been widely practised by the Egyptian doctors. Indeed, in the Edwin Smith papyrus we find no mention of the trepan.

Perhaps surgical practice in Egypt was regulated by law, as in Babylon where the *lex talionis* (the principle of retaliation) became encapsulated in the code of Hammurabi, sixth King of Babylon, over 2000 years BC. In this we are given some insight into the life of the surgeons. 'A physician who makes a wound and saves a freeman shall receive ten pieces of silver, five pieces of silver for a plebeian and for a slave two pieces of silver. However, if a doctor treat a patient with a metal knife for a severe wound and has caused the man to die his hand shall be cut off'. Perhaps that explains our lack of knowledge about surgical techniques in Babylon or Egypt. With the threat of retaliation hanging over them the doctors were probably afraid to operate.

If the physicians on the banks of the Nile, the Tigris and the Euphrates were conservative in their treatment of head injuries, the Greeks some 2000 years later adopted a much more aggressive approach. 'War,' said Hippocrates, 'is the only real school for the surgeon'. In the fifth century BC, Greece emerged from the Bronze Age. The development of steel made possible new instruments such as saws and a trephine of a design used in Europe until the seventeenth century AD. In the school of Cos, dominated by the Hippocratic corpus, we find the basis of

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operative neurosurgery. The Greeks perfected the technique of trephining the skull. Their incisions and methods of elevating skull fracture were in use 2000 years later. Earlier civilisations had practised elevation of depressed fractures but the Greeks advocated operation on linear fractures as well. The reasons are not quite clear. Perhaps they hoped to anticipate the development of an extradural haematoma. However, they accorded the linear fracture a status shared today only by the plaintiff's legal advisers in a High Court action. Although their operative skills improved immensely, the outlook for patients with penetrating wounds remained appalling, and if brain tissue exuded from the wound death was to be expected.

Celsus (25 BC – 37 AD) kept alive the practical neurosurgical skills developed in Greece; his chief contribution was his extensive writings which, together with those of Hippocrates formed the standard surgical textbooks until the seventeenth century. Galen, the last medical writer of note in the ancient world, developed new instruments for head injury operations. However, even before the fall of the Roman Empire in the West, scientific medicine had gone to sleep, not to awaken for 1000 years when Europe would emerge from the Dark Ages. The Greek and Roman manuscripts went east to Byzantium where Arabs preserved medical knowledge for a time. Later the manuscripts came west again, first to the monasteries and later to the great mediaeval universities of Paris, Oxford, Padua, Bologna and Montpellier.

THE SIXTEENTH CENTURY

By this time the Mongols had brought gunpowder to Europe from China and a new era of penetrating head injuries dawned. A ball from a Spanish musket could now penetrate the thickest armour. At first the devastating effect of bullet wounds on the brain was attributed to poisoning from the saltpetre in gunpowder. Surgeons hoped that boiling oil poured into the wound would counteract the poison. By the mid-sixteenth century Ambroise Paré[3] discarded cautery realising that the bullet caused mechanical disruption of the brain. He encouraged the patient to strain against a closed nose and mouth to force out 'sanious matter and filth'. Paré was probably treating brain abscesses when he used the trephine to evacuate 'sanies or matter poured forth upon the membranes'. We now know that retained bone fragments promote infection. The mortality remained high.

THE SEVENTEENTH CENTURY

Even at this time the treatment of missile wounds followed the teaching of the Greeks and the Romans. Surgeon Wiseman,[4] an army surgeon on the side of the Roundheads at the battle of Worcester (1651) dressed wounds using powders concocted by Galen or sometimes used red meat followed by red wine. Wiseman's incisions were those devised by Hippocrates 2100 years earlier. By now the crown saw and circular bit had been invented and Wiseman followed the Greek practice of operating on all fractures, linear and depressed alike. The instrument was to be lubricated with milk and the surgeon was 'to proceed warily not listening to the prattling of standers by'. Wiseman reports that rapier wounds of the head were easy to treat but wounds caused by 'a poleaxe, or halberd or other obtuse weapons were more difficult'. Fractures made by musket wounds 'for the most part beat pieces of the skull inside the brain and so may be considered mortal, but be the hurt what it will, if it penetrate not further than the dura mater it is curable if it be timely laid open and dressed'. Here in the seventeenth century was a clear

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recognition of the importance of the dura mater, a point that the Egyptians had stressed 4500 years earlier. Wiseman was the first surgeon of his time to insist, perhaps for the wrong reasons, that fragments must be removed from the brain 'but if they will not come easily away, leave it to nature, lest the patient die under your hands, and you be thought to hasten his death'.

THE EIGHTEENTH CENTURY

Our information on penetrating head injuries at this time is derived from army surgeons. Their views depended largely on whether they worked at the front line or in a base hospital. In 1761 John Hunter,[5] then surgeon to the British troops at Belle Isle, well away from the front line, felt that 'injuries in consequence of a musquet-ball' were very little different from other head injuries and should 'require no peculiar mode of treatment'. He didn't appreciate that he was seeing the less severe cases, the slow journey to hospital having 'selected out' good survivors. Hunter wrote 'it was hardly necessary for a man to be a surgeon to practise in the army' — not much progress was made at that time.

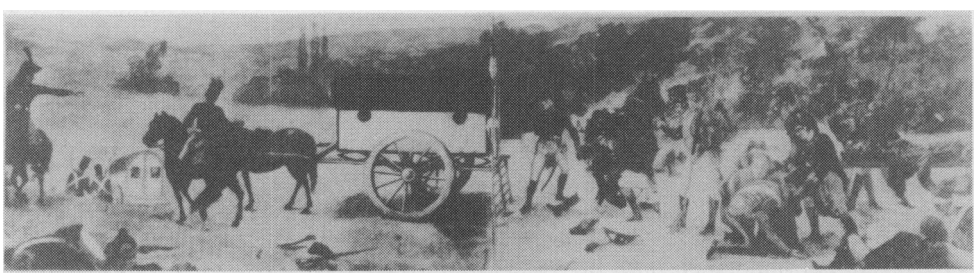

Fig 2. Baron Larrey's flying ambulance.
(From *A near run thing,* by D Howarth. London: The Literary Guild, 1968).

On the other hand, Napoleon's doctor, Baron Larrey, was recognised, even by the British, as the foremost of army surgeons. He appreciated the need for swift action at the front line and invented what he called the 'ambulance volante' — the flying ambulance. It was a well-sprung, two-wheeled cart fitted with litters. Each division had 12 of these ambulances and 130 mounted medical staff, providing an evacuation service, vital to patients with head injury, and probably unequalled till the 1939–45 War. At Waterloo the British surgeon had no ambulance and operated near the battle. He brought his own equipment including a canteen of good wine and spirits; for, as a medical handbook said, 'many men sink beyond recovery for want of a timely cordial before, during and after operations'.

Towards the end of the late eighteenth century the policy of operating on all skull fractures was being challenged. A rational approach came from Sylvester O'Halloran, surgeon to the Limerick County Hospital, and descendant from one of the oldest families in Connaught. His training had been in Paris, Vienna and London. He must have cut a conspicuous figure in Limerick. Sir William Wilde, father of Oscar Wilde, describes him as 'a tall thin doctor in his quaint French dress, with his gold-headed cane, beautiful Parisian wig and cocked hat, turning out every day to visit his patients'.[6] Pride in his ancestry led him to revive the old family motto 'I destroy and I kill', not a very suitable motto for an eminent surgeon. He was elegant, well known all over the continent and, according to Sir

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Fig 3. Sylvester O'Halloran, MRIA.[6]

Lucius O'Brien, Bt, President of the Antiquarian Society, 'he was never more at home than when abroad' an aphorism that could be applied to many of his academic successors.

Although he was an Honorary Fellow of the Royal College of Surgeons in Ireland, O'Halloran appreciated that, as a surgeon practising in Limerick, a city which had neither a philosophical nor a medical society, he must establish his qualification to write with authority. In 1793 he prefaced one of his books on head injuries 'that I may not be deemed presumptious in thus assuming the style of a master and, by way of engaging the favourable opinion of the public, I beg leave to submit to the candid and critical reader my pretensions to this character. Without doubt there is not part of the habitable globe as for half a century past has afforded such an ample field for observations on the injuries of the head as Ireland in general and this province of Munster, in particular. A slight offence is frequently followed by serious consequences and sticks, stones and every other species of offence are dealt out with great liberality. To this add the frequent abuse of spirited liquors, particularly whiskey, which has unhappily for the morals and constitutions of the people found its way to every part of the kingdom. Many of our fares, patrons and hurling matches terminate in bloody conflict. From this it appears what superior advantages Irish surgeons have long possessed in this department of their profession over those of the neighbouring nations. I have had no less than four fractured skulls to trepan on a May morning and frequently one or two other injuries resulting from the activities of the Whiteboys. The infirmary becomes as necessary as the gaol of which it seems but the outer porch'.[7]

O'Halloran's first achievement in the management of head injuries was to lay to rest the notion that all patients with fractures of the skull automatically require an operation. His work was soon recognised in Paris and in London and the practice of speculative trephining of the skull in patients with linear fractures had ceased by the early nineteenth century, bringing to an end the Hippocratic tradition of 2400 years. His next achievement was to improve the treatment of penetrating head wounds. He quotes the case of Patrick Casey, aged 18, who sustained a penetrating head injury when he fell from a horse. Another doctor had performed a delayed and inadequate operation leaving bone fragments in the brain, O'Halloran pointed out that the patient was 'free from pain and fever at the beginning'. Delay led to deterioration and 'the substance of the brain poured forth

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and he expired the next morning'. That patient almost certainly died from a brain abscess. Although the relevance of pus was not appreciated in those days, O'Halloran, working at the same time as Baron Larrey, appreciated from his own experience that early and meticulous wound débridement was important. O'Halloran in Limerick and Dease in Dublin were having some success with their improved surgical technique. We find the first note of optimism in the management of these open brain injuries from a contemporary, Abraham Colles, in his lecture notes from Trinity College, Dublin, published posthumously in 1844: 'Suppose you are called in immediately after a man has received a severe injury of the head, and you find a part of his brain in his hat, or that it comes away in his night cap after he is laid in bed — does it make any difference in the case? None whatever, many such cases have done very well'. That was the Irish contribution from the civilian field at the turn of the eighteenth century, but few of these patients had gunshot wounds.

THE NINETEENTH CENTURY

The writings on war surgery throughout the nineteenth century convey unrelieved gloom. In the Crimean War of 1856, the mortality from penetrating head injuries in soldiers who were evacuated from the front line was over 90%. In the American Civil War (1861–65) 80% died.[8] In the Franco-Prussian War of 1871, few operations were performed, presumably because of the remote chance of success. Slow evacuation and inadequate surgery no doubt contributed to the bad results. There was still no knowledge of bacteriology: infection was attributed to miasma. One new benefit to the injured appeared just before the American Civil War — the introduction of general anaesthesia by Morton.

Lister's introduction of the antiseptic method in 1867 coupled with new techniques developed by British surgeons of the Royal Army Medical Corps in the Anglo-Boer War (1899–1902) opened the modern era of the surgery of penetrating head wounds. With these advances one might have expected an improved chance of survival. However, the South African war also saw the introduction of high velocity, small calibre bullets fired from modern rifles. At first it was thought that these small missiles might cause less brain damage than the larger Martini-Henry low velocity bullet. It soon became clear that survival from a penetrating high velocity bullet was rare unless the bullet was fired at long range. The South African war was the only modern military conflict in which bullet wounds were more common than wounds caused by metallic fragments from shells or mortars,[9] and bullet wounds carry a much higher mortality. Transport of the wounded to hospital was, as usual, difficult. A front line artist has left an impression of the ambulances which conveyed the British wounded during the battle of Lombard's Kop at Ladysmith. We see a cumbersome unsprung oxen cart, comparing very unfavourably with Baron Larrey's 'ambulance volante'. The cart has been struck by a shell; we see the felled oxen and wounded men staggering in bewilderment and agony around the wreckage.

Comparison of results between one conflict and another is difficult because the results depend on the interval between wounding and treatment and on the severity of the wound. Nevertheless, surgical progress seems to have been made: of the patients who reached hospital in South Africa the mortality was only 33%. The essential improvement in technique lay in gaining wide access to the track of the wound by large incisions, identifying and removing indriven bone fragments and blood clots, suturing the dura mater and closing the wound without drainage.

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Not enough credit has been given to the Boer War surgeons, notably Makins,[10] Bowlby and Wallace.[11]

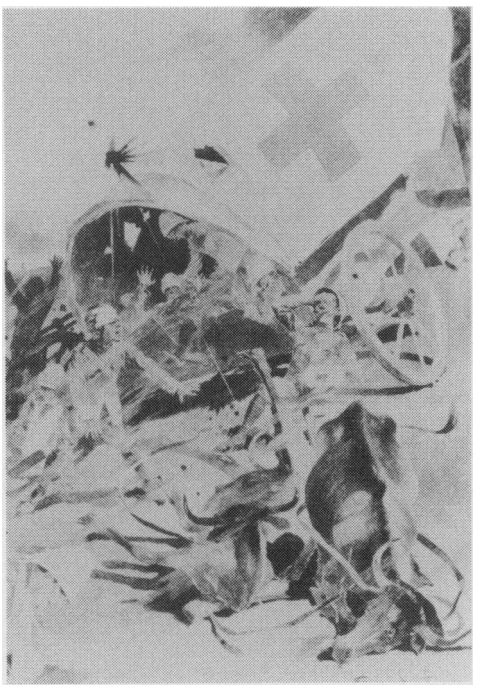

Fig 4. Firing on the ambulance: a scene during the battle of Lombard's Kop at Ladysmith, 1899. (From *Frontline artists*, by Peter Johnson. London: Cassell, 1978).

THE TWENTIETH CENTURY

The history of war surgery is that the lessons of earlier conflicts are forgotten. The 1914–18 War was no exception. The lessons from South Africa were 'rediscovered' in 1916, but only after two years of disastrous experience. In 1916 Cushing,[12] who had had the benefit of meeting Sir George Makins, Sir Anthony Bowlby and Sir Cuthbert Wallace, redefined the essential operative techniques which soon reduced the mortality from penetrating wounds from over 60% to about 40%. Even so definitive neurosurgical treatment was often delayed for 48 hours.

In the 1939–45 War, history was again repeated. In 1942 Ascroft found that 27% of penetrating head wounds in the Middle East developed brain abscesses.[13] Neurosurgeons were by then available but many patients were operated on first by general surgeons and evacuation was slow. The lessons from 1914–18 had been forgotten; Cairns sent to the United States for Cushing's papers to guide him in establishing the neurosurgical forward units which ultimately proved so successful. More rapid evacuation, better neurosurgical débridement and the introduction of penicillin, after Ian Fraser's field trials of the drug,[14] brought the infection rate down to only 3% by 1945. Surgical techniques for the most difficult cases were further refined by Cecil Calvert and Hugh Cairns.[15] Many patients with missile wounds, especially those injured by metallic fragments rather than bullets, made an excellent recovery. Since these techniques were developed, the risk of serious infection from penetrating injuries sustained in road traffic accidents has been almost eliminated provided the patient reaches neurosurgical care early.

Helicopter evacuation was used in Cyprus[16] and in Korea where the patient reached neurosurgical care within eight hours of injury.[17] The time interval came down to 1–1½ hours in Vietnam,[18] where the incidence of serious wound infection fell to only 1–2%. Of the 49 British soldiers with penetrating head injuries who received definitive neurosurgical care in Korea, there were no brain abscesses, but three patients developed meningitis.[19]

NORTHERN IRELAND

The Northern Ireland experience with missile wounds is in some respects unique.[20] It differs from military conflicts in that patients arrive at hospital much sooner after injury, their wounds are more severe and better facilities are available

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for resuscitation and treatment. Most patients, especially those injured in Belfast, reached hospital within 30 minutes of injury. Almost immediate resuscitation, which was seldom possible in military conflicts, now became possible. On the other hand the rapid ambulance service led to many moribund patients reaching hospital only to die shortly after admission. Forty-two per cent of hospital deaths took place within six hours of injury.

In twentieth century warfare over 80% of head wounds are caused by metallic fragments from explosions, These injuries are usually less severe than bullet wounds. In 1947, Russell had reported that 43% of patients with penetrating head wounds in the 1939–45 War did not even lose consciousness.[21] In Vietnam, 86% of metallic fragment wounds were operable as compared with 56% of bullet wounds.[18] In Northern Ireland the pattern of injuries has been different: over 80% of penetrating head wounds have been caused by bullets; because of the severity of these wounds and the rapid transport to hospital, the hospital mortality is high, at 56%.[20]

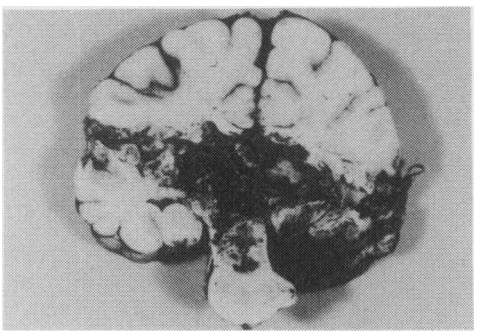

Fig 5. High velocity bullet wound of the brain.

In the Anglo-Boer War the difference between wounds caused by high velocity bullets and low velocity missiles of all kinds became obvious. The effect of a bullet passing through the body depends largely on its kinetic energy rather than on its size, shape or flight characteristics. As the kinetic energy of a bullet is a function of its mass and the square of its velocity ($E = \frac{1}{2}\ mv^2$), it follows that a small high velocity bullet from a rifle carries more energy than a larger low velocity bullet from a hand gun. High speed photography of bullets fired into gelatine blocks or animal tissues demonstrates the effect of the radial forces generated by a high velocity bullet.[22] The pictures reveal a large, almost spherical, temporary cavity which exists for only micro-seconds before giving way to the smaller, more fusiform permanent cavity. A low velocity bullet leaves a track of a diameter not much greater than that of the bullet itself.

The first hour of treatment

A patient with a cranial missile injury is in a highly labile state. He may be conscious on admission, only to deteriorate rapidly and die within an hour. Clinical deterioration is often associated with hypoxia due to epileptic fits, bouts of coughing or inhalation of vomitus or blood. Hypovolaemic shock from other injuries, or due to bleeding from cerebral vessels, can also cause cerebral ischaemia which may prove fatal in a patient with a severely injured brain. Prevention of secondary brain damage from hypoxia and hypotension is the chief aim of early management. This was the chief challenge facing us in Belfast in the early 1970s. We soon realised that practically every patient with a cerebral missile wound, conscious or unconscious, needed immediate tracheal intubation and mechanical ventilation. The blood O_2 and CO_2 levels could be controlled and excessive rises of intracranial pressure prevented.

Adequate resuscitation demands the presence of appropriate medical staff. A team of three doctors is ideal: one to examine and record and to assess

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priorities of treatment; the second, preferably an anaesthetist, to take care of the airway, and the third to replace fluid loss, which may be substantial, by intravenous infusion. Patients with cerebral missile wounds who remain in deep coma after cardio-pulmonary resuscitation do not survive. The fact that 72% of patients with a mean blood pressure under 90mmHg on admission died, gives an indication of the importance of early and adequate transfusion. One of the advances in the management of these injuries in Northern Ireland has been the involvement of anaesthetists in resuscitation, in subsequent intensive therapy and, outstandingly, in transporting patients from other hospitals using mechanical ventilation in the ambulance. Clinical deterioration in the ambulance, which can be a problem with any kind of head injury, has been almost eliminated in Northern Ireland.

Surgery

The aim of surgery is to arrest haemorrhage, to prevent infection by removing bone and any accessible metal fragments and to repair the dura mater and scalp in a way that facilitates cranioplasty at a later date. No patient treated in the neurosurgical department has developed a brain abscess.

Extensive débridement of a missile wound often leaves a very large skull defect which has to be repaired to protect the brain and to improve the patient's appearance. None of the conventional cranioplasty techniques — bone grafting or insertion of acrylic or metal plates — proved satisfactory. A high pressure moulding technique developed in the Royal Victoria Hospital School of Dentistry has proved ideal for producing accurately contoured titanium plates.[23] Titanium was chosen because of its low density, radiolucency, malleability and, most important of all, its lack of reactivity with the tissues. Titanium cranioplasty has now been adopted in many centres as the method of choice.

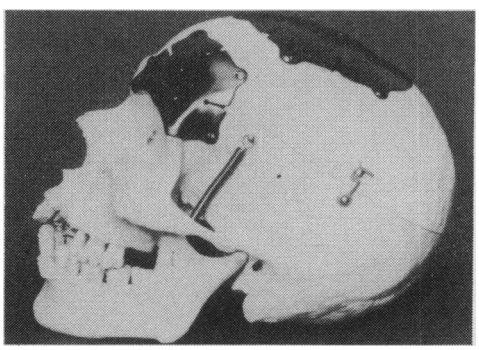

Fig 6. Titanium cranioplasty: a demonstration specimen which is now in the Science Museum, London.

The challenge of the missile injury is increasing in western society. To meet this challenge, the emergency services must be organised to provide immediate resuscitation and intensive therapy and, when necessary, expert care during transportation to the neurosurgeon. Many patients with severe head injuries from any cause die from primary brain damage. Our experience in the past 18 years indicates that emphasis must be placed on preventing secondary brain damage brought about by hypoxia and hypotension. Immediate action can save lives and prevent some of the after-effects of brain injury.

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Not strangers, but pilgrims

Annual Oration at the opening of the 1987 – 1988 teaching session, Royal Victoria Hospital, delivered on 1 October 1987.

D Burrows

I had some difficulty with both the subject and the title, bearing in mind that this oration is a welcome to the new students to the Hospital. It is common practice to deliver a lecture of some historical interest, often on one's specialty. I have resisted that for three reasons. Firstly, Dr Reginald Hall has dealt with the history of dermatology in an earlier paper in a most distinguished manner.[1] Secondly, while those who cannot remember the past are condemned to repeat it, it is probably more true that we learn nothing from history except that we can learn nothing from it. I cannot think that anyone here is consumed with the desire to know the origins of dermatology. Thirdly, though I feel the temptation to take this opportunity to advance a couple of my obsessions, I feel I should resist this.

One of these obsessions is to tell you why I have such an affection for dermatology — an affection that is shared with young doctors in the United States where it is the second most popular specialty. We deal with a young age group — 27 years on average, younger than ophthalmology patients, for instance. All our patients are ill: that is, have something physically wrong with them. It is probably the largest medical specialty, comprising 8% of all patients seen by doctors, and 75% of all industrial disease.

My second temptation is to tell you how dermatology as a specialty could have been better and how younger specialties could learn from this. In the rest of the world, dermatology has followed an independent specialty line and has prospered, with full university departments, larger numbers, funding for research, etc. In the United Kingdom a different path was taken some years ago by those who led us at that time, who felt that dermatology's best path was as a sub-specialty of medicine: that is, that dermatologists were physicians with an interest in skin, rather than forming a specialty group in their own right. The results of this controlled experiment of the UK versus the rest lead me to believe that the rest had chosen more wisely.

So, a path chosen can have importance for your own career but it may also influence the career of those who succeed you. I have for many years, had a great affection for Robert Frost's poem *The road not taken*:

> 'Two roads diverged in a wood, and I —
> I took the one less travelled by,
> And that has made all the difference.'

So I see medicine as setting out on a journey. The idea of life as a journey is common to many cultures and ages, both in mythology and in real life. Many of the epic journeys seem to have been by sea: Marco Polo, Columbus, Jason with

Department of Dermatology, Royal Victoria Hospital, Belfast BT12 6BA.
D Burrows, MD, FRCP, Consultant Dermatologist.

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his Argonauts who set out to capture the Golden Fleece, and Ulysses who made his long way home on a journey that has become a legend for man's journey through life. I think probably the greatest story of a journey is *Pilgrim's Progress,* written in 1670, of which Bunyan sold 100,000 copies in his lifetime, and which was translated into 190 languages.

My second difficulty was the title. I am worried lest my present title might sound rather like a sermon, as it is taken from Hebrews 11, verse 13. It refers to Abraham as 'a stranger and a pilgrim who went out not knowing where he was going'; which I think describes the young doctor's dilemma today and you may feel like the saints described in that same chapter: 'They all died not having received the promises but having seen them afar off'. I hope you don't die before the promise of a senior job is fulfilled though some of you may feel like it. I changed it to 'Not strangers' because I consider medicine one of the best fellowships one could enter. Perhaps a better title would have been 'Hats off to the past, coats off for the future'.

So, I see you as embarking on a journey rather as Pilgrim did. The journey has three parts — setting out, the journey itself, and the final destination, probably the most important part. It is important to have an aim in life.

SETTING OUT

TS Eliot wrote a poem on his graduation, in which he described the future:

'Although the path be tortuous and slow,
Although it bristle with a thousand fears,
To hopeful eye of youth it still appears
A lane by which the rose and hawthorn grow.'

So really the simple purpose of this Oration is to point out a few roses which will cheer your path and warn you about some thorns.

You have set out in medicine. What are your reasons for doing so? I thought it would be interesting to look at the expectations of youth setting out and to compare it with the experience of a mature doctor. So I sent a questionnaire to five groups, as shown in Table I.

TABLE I

Number of questionnaires sent out and returned

	Sent out	*Returns*
Students	157	157
Junior doctors	150	69
Northern Ireland consultants	150	106
General practitioners	150	96
RVH consultants	170	116

The questions were: 1. What made you choose medicine, and have your expectations been fulfilled? 2. What qualities do you consider make a good doctor? 3. What do you think could have been added to the medical curriculum to improve your medical education? Those questioned were asked to score 0–5, 0 being of no importance, 5 very important. Table II is made up by adding together those who put down '4' or '5' in the column.

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TABLE II

When you applied to study at university, what made you choose medicine? The percentages replying 'important' or 'very important' to the different modalities (4 and 5 on a scale 1 to 5)

	Students (%)	*Doctors (%)*	*Doctors' expectations fulfilled (%)*
Job satisfaction	86	76	91
Helping people	66	62	92
Challenging job	55	47	92
Scientific interest	49	39	82
Comfortable income	40	29	86
Job security	38	43	76
Personality suited to profession	37	28	93
Opportunity for leadership	30	7	76
Social status	19	16	83
Potential for very high earnings	14	5	42
Did not know what else to do	12	10	—
Parental influence	11	17	—
Peer group pressure	5	3	—
School careers advice	5	4	—

One of the interesting outcomes was that in every modality the fulfilment of expectations had been much higher than the expectations themselves. There cannot be many professions in which that is the case. I was very gratified to see that the reasons which we might consider to be the rather better ones, such as helping people, job satisfaction and looking for a challenging job, came out highest. I was especially pleased that this also applied to the students. For a number of the modalities it was interesting to look for differences between the various groups of doctors. Those who became consultants had higher expectations of scientific interest, which were generally fulfilled. Otherwise there was remarkably little difference between senior house officers, general practitioners and consultants.

On setting out on the medical journey, one's luggage consists only of three things — intellect, character and training — and two of these one can do nothing about, because they are handed to us: it is difficult to put weight between character and intellect. As the years have gone on, I have become more and more convinced that hard work and dedication will outlast and outrun intellect. Any consultant in this room, could name without thinking too hard, not a few with a mediocre undergraduate performance but a brilliant postgraduate career. HJ Walton, Professor of International Medical Education at Edinburgh, confirms that this is not just an impression.[2] To summarise his evidence, intellectual ability accounts for about 35% of the observed variance, and the addition of personality and motivation measures accounts for up to 75%.

The answers to the second question are shown in Table III. Most people thought that high intelligence was not one of the most important things, and that the most

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important characteristic of all was common sense, followed closely by keeping up to date with the subject, integrity and being a good communicator. As before, students and doctors were very similar in their perceptions.

TABLE III

What do you think makes a good doctor?

	Students %	*Doctors* %
Commonsense	97	98
Good communicator	94	92
Keeping up to date with subject	93	96
Readily available to patient	89	81
Bedside manner	89	86
Ability to get on with others	88	83
Integrity	85	92
Hard worker	82	88
Most effective at age 35–45	62	52
Efficiency at seeing patients	54	54
Having time for patients	52	65
Interests outside medicine	52	46
Leadership qualities	40	39
Good academic background	40	28
Good at administration	26	31
Most effective at age 45–55	22	37
High intelligence	19	18
Interest in research	13	6
Retiring manner	6	3

The only piece of luggage we have a choice about is our training. You are now committed to this medical school. We all say that Queen's is one of the best medical schools, but how do we know? How can it be assessed? Is it the amount or quality of research? One couldn't say there have been many discoveries made in Belfast to revolutionise medical care or medical teaching. Is it the quality of the graduates? How can we assess quality? It is easy with a football team or a business. As CS Lewis said, the purpose of education is not to cut down jungles but to irrigate the desert. How do we know if we are growing the right plants, or applying the best fertiliser, or doing it better than other people? One thing I have little doubt about is that Queen's University has been the most caring medical school. Though I cannot get figures to prove it, I believe it has made the greatest contribution to the Third World of any University because of the strong Christian commitment of its medical intake from both communities.

In Professor Roddie's inaugural lecture *An excellent medical school,* he deals with all these points, and in my opinion this should be required reading for all medical staff and medical students.[3] I think the worst fault that people in my generation fell into was to convince ourselves that we had an excellent medical school and

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not question, as Professor Roddie has done, whether indeed we did, or if we did, what was necessary to retain its excellence. The things which my respondents considered would improve medical education are shown in Table IV. It would seem that most people felt there was not great room for improvement. It is interesting that computer skills, avoiding litigation and an illness of your own, came high, but highest of all came interests outside medicine.

TABLE IV

Do you feel experience/knowledge of any of the following would be helpful in your medical curriculum?

	Students and doctors (%)
Interests outside medicine	48
Computer skills	45
In illness of your own	41
Advice on avoidance of negligence claims	37
Administration/organisation	36
Teaching how to get on with colleagues	36
Statistics	32
Court witness technique	27
Experience in nursing	23
Teaching in leadership	21
Typing	20
Shorthand	13
Social sciences	12
A foreign language	10
Photography	5
Other (please specify)	(70)

Most people probably do not know that the Medical Library Association was inaugurated in Belfast on July 28, 1909,[4] and the distinguished first president, who gave the inaugural lecture, was Sir William Osler, and in Belfast he said, 'One of the best features I find in my "old country" colleagues is the frequency with which they have hobbies. No man is really happy or safe without one, and it makes precious little difference what the outside interest may be — botany, beetles or butterflies, roses, tulips or irises, fishing, mountaineering or antiquities — anything will do so long as he straddle the hobby and ride it hard'. His lecture was packed with good advice: 'In the case of our habits, we are only masters of the beginning, the growth by gradual stages being imperceptible like the growth of a disease'. In this he was referring to reading. It is interesting to see how little times have changed, because he said, 'How can a busy man read, driven early and late, tired out and worried? He cannot. It's useless to try, unless he has got into the habit when he was not so busy. Then it comes easy enough and the hardest workman in the land may read his journals every week, even if he has to do it in his carriage'.

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The future may seem bleak to you with over-intake into medical schools but this has only increased by 0.35% from 1979. You are in a vastly superior position to medical students in other countries. For instance, Italy has 30,000 unemployed doctors, Spain has 20,000, Mexico has a national union of unemployed physicians with a membership of 50,000, and one medical school in Europe, Naples, admits over 7,000 students a year. You are joining a profession with 88,000 members in Britain, of whom 24,000 are in general practice, 11,000 are senior hospital doctors and 20,000 junior doctors: 56% of you will finish in general practice, 27% as senior hospital doctors, 5% as academics and 5% in community health. About one person every other year in the medical school will not bother to register, but, once registered, only about 0.2% leave the practice of medicine which must be a very much lower proportion than in other professions. Bearing in mind that many of those who leave do so to occupy distinguished positions in other professions, this would suggest that doctors are well fulfilled in their profession.

THE JOURNEY

Having started, we think about the journey. In my view the best guide is a Christian faith, but there are others, and we will concentrate on those today. There are a number of roses and thorns which I wish to discuss, and they are not meant to be exclusive.

ROSES

Colleagues. The best help you have are good colleagues and friends and a happy environment. I think one of the unique features of the medical profession is the support and friendship which one receives from colleagues over the years. I look round other professions and I do not see the same degree of colleague support. Perhaps the best asset we have is our happy relationship with the nursing profession. Some are working hard to destroy this. Don't let them. Nevertheless, that does not mean to say that we are always in agreement, but healthy disagreement with one's colleagues is valuable. It is said, when two men in business always agree, one of them is unnecessary.

Beware of advice from so-called wiser experienced colleagues. I have seen some catastrophic advice given to young men by so-called older wiser colleagues. The notion that as a man grows older his illusions leave him is not quite true. What is true is that early illusions are supplanted by new and, to him, equally convincing illusions.

Etiquette and ethics. You might think these should be considered among burdens, but, in my opinion, the reverse is true. I am sad to see that the word 'etiquette' is being dropped from the new BMA handbook on medical ethics. It is said to have virtually disappeared from current usage in the English language, and we now talk about professional behaviour. Professional behaviour is really good manners towards one's colleagues. It may be that the hardest thing young people today have to do is learn good manners without seeing any.

Medical traditions themselves are important. Don't disparage them. As a result of your forebears you are going to be placed in a privileged place in society. This is a sweet-smelling rose. It is impossible to over-estimate the benefit of public goodwill towards the medical profession. I hope younger colleagues will continue that, and I see no reason to suspect that they won't.

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Health. Doctors have significantly good health. Standard mortality rate for those aged 20–64 in the last decennial supplements of occupational mortality show that doctors have a standard mortality rate of 66, as opposed to the normal of 100. There are, however, two diseases from which doctors suffer a statistically significant excess in mortality. Firstly, suicide — the standard mortality rate for doctors is 172 and there are about 65 suicides per year in the UK. It may be that this relates to the stress of the medical profession, but an important factor may also be the ready availability of methods of suicide. Secondly, diseases of alcoholism — Professor Rawnsley has shown that the rates for doctors for admission with alcoholism to Scottish psychiatric units is about three times those for controls.[5] That this is a problem is confirmed by the standard mortality ratio for physicians and surgeons for liver cirrhosis (311 in 1971). We probably have a duty to two groups in this regard. Firstly, to medical students and younger doctors we should point out that the medical profession has a problem which many believe begins in student days, including Professor Anthony Clare, as evidenced in his Snow Lecture to the British Association of Anaesthetists in 1986 (unpublished). Perhaps we should make it more difficult rather than less difficult for students to consume large quantities of alcohol. Our second duty is to our patients who take their example from us. Doctors who gave up smoking have had a major effect on the general population. It has been said that a doctor only regards a person as an alcoholic when he drinks more than himself. If they take their standards from us, then they are in trouble. You are entering a stressful occupation, as a very recent paper has shown, more so than any other profession, but it is an enjoyable one, even at a junior level.[6]

Doctors have a much better rate of health in most things, eg, carcinoma of the bronchus, standardised mortality 25, ischaemic heart disease, standardised mortality 22, and the lowest standard mortality for doctors, 12, is for ulcer of the stomach and duodenum. There is a very common belief that, if one retires early, it increases one's expectation of life. It has been said that, if one retires at 60, one may have a life expectation of 10 years, as opposed to one year when retiring at 65. The belief is widespread and pervades the whole UK. The figures from the Registrar-General (England and Wales) are shown in Table V. These figures may be slightly skewed by those retiring early from ill health, but the message is clear. If you are retiring early to enjoy a longer life, you won't. An argument has been put forward that it would cost the pension fund too much to allow doctors to retire early. These figures do not support this.

TABLE V

Life expectancy of medical pensioners in the United Kingdom

Age at retirement	*Life expectancy in years*	
	Males	*Females*
30	20.0	34.3
35	19.3	32.0
40	18.3	29.6
45	17.2	26.9
50	15.8	24.1
55	14.1	21.0
60	14.9	22.5
65	14.2	18.4

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It would be interesting to know if different specialties have different health rates. I was not able to find any figures except those in a study of 1020 United States physicians who died between January 1978 and March 1979 (Table VI).[7] There are more octogenarians among the ENT and eye specialists, with the dermatologists in third place. No family doctors survived to this age.

TABLE VI

How specialty relates to longevity in the USA

	Percentage of doctors who died at this age				
	Less than 50	*50–59*	*60–69*	*70–79*	*80 plus*
Otolaryngology	0	5.6	18.1	34.7	41.7
Ophthalmology	3.1	9.4	17.2	40.6	29.7
Dermatology	8.3	8.3	25.0	29.2	29.2
Internal medicine	8.9	14.9	28.6	26.8	20.8
Paediatrics	4.2	22.5	28.2	25.4	19.7
Obstetrics/Gynaecology	13.7	9.8	28.4	32.4	15.7
Orthopaedics	8.3	8.3	39.6	29.2	14.6
Preventive medicine	0	3.2	38.7	45.2	12.9
Surgery	10.8	21.6	33.1	21.6	12.8
Family practice	25.0	41.7	29.2	4.2	0

From: Mostafa MFD, Freeman RA.[7]

A FEW THORNS

Litigation. Litigation is a worrying problem, increasing not only in numbers but in the amount of awards given. It is very difficult to get exact figures (the Defence Unions don't give theirs). The figures for England are shown in Table VII, which works out about one claim per 25 doctors. In Northern Ireland there is a higher rate (1980 — 77 cases; 1984 — 197 cases): with about 2,500 doctors this is one case per 12.5 doctors per year. If you multiply this by 40 (the average working life of a doctor), it would suggest that, if the present rate continues, and we have no reason to suppose it is going to get any less, then a doctor in Northern Ireland can expect to be sued on average three times in his lifetime. One cannot imagine that medical practice is any worse here than in England, so this difference must be due to two things — the proclivity of the local population for litigation, and the zeal of certain solicitors to look for new business. An interesting sideline is that a hospital has three times the chance of being sued by its staff than it has by the patient.

TABLE VII

Claims against hospitals — England

	No. of cases	*Payments £*
1981/82	2,791	3,437,363
1982/83	2,990	3,569,019
1983/84	3,266	4,839,175

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Administration. Having had some experience of medical administration, I believe it is essential for doctors to be involved. However, I think I should say that it can be a snare and a delusion — a delusion that you are doing any good or in any way helping the patient. Administration, be it medical or other, only redistributes resources. It doesn't create medical wealth. One can very quickly suffer the delusion that one is actually improving the lot of patients. Nothing could be further from the truth.

There are only three groups of people who create medical wealth — those who finance it, those who deliver it, and those who move the frontiers forward in research. The Government allocates most of the resources and in this country there is a very low total health expenditure (Table VIII). With the exception of Sweden, public health expenditure as a percentage of the gross domestic product in the UK is not greatly different from the average in other countries. The difference is in the amount contributed by private sources. Even in Sweden 0.8% is supplied from the private sector as opposed to only 0.7% in Britain. Perhaps we do need more private input to health. I find it hard to know whether to consider private practice as a rose or a thorn in the body medical. I think, on balance, it has a greater effect for good than bad.

TABLE VIII

International comparisons.
Health expenditure as percentage of gross domestic product, 1983

	Total health expenditure	*Public health expenditure*
USA	10.8	4.5
Sweden	9.6	8.8
France	9.3	6.6
Netherlands	8.8	6.9
Germany (GFR)	8.2	—
Australia	7.5	4.9
Italy	7.4	6.2
Austria	7.3	4.6
Norway	6.9	6.2
Japan	6.7	5.0
Finland	6.6	5.2
Denmark	6.6	5.6
Belgium	6.5	6.0
UK	6.2	5.5
Greece	4.7	?

Administration is also a snare because it takes time, and some get involved in it when they should be committed to other things. It is a particular snare to younger doctors for it may detract them from research, which should be a main function in a teaching hospital after duties to patients and teaching. I don't know what makes one a success in administration, but I do know what makes a failure, and that is

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trying to please everyone. The best advice I know is the best is the enemy of the good — the advice that Sir John Harvey Jones, the most successful chairman Imperial Chemical Industries ever had, gave to his younger staff. What I mean is that the relentless pursuit of the perfect solution to a problem often means that nothing gets done. Walter Lippmann expressed it better than I can when he said: 'A rational man acting in the real world may be defined as one who decides where he will strike a balance between what he desires and what can be done. It is only in imaginary worlds that we can do whatever we wish'. This is not only true of administration but of research. I have found more research abandoned because it aimed too high rather than too low. It is also true that the pursuit of the best for one unit means abandonment of the good for someone else.

Age. Another thorn is age — your own and the patient's. It astonishes me that anyone can find merit in the argument that there should be no retiring age. I feel the problem is one for the General Medical Council. I find it surprising that, over the age of 70, one has to undergo a medical examination to carry on driving a car, and yet one is allowed to go on doing a much more dangerous thing, treating patients, without any check on mental health, eyesight or other faculties. My knowledge of doctors further leads me to the view that a person over 70 is more likely to have problems than somebody just finishing their resident year. The General Medical Council figures confirm this, and it is extraordinary that this body should be considering a second year of compulsory training, while doing nothing about control of ageing and perhaps even dementing doctors. The other problem is the age of your patient: 15% of the population are over 65, and require 50% of the health resources. Quite clearly, if you don't like looking after old people you shouldn't be taking up medicine.

There are other burdens which will be added to your shoulders, which we have avoided, such as investigation about your cost-effectiveness and your performance indicators. I wish I had time to give you my views on these.

JOURNEY'S END

'What are you aiming for?' Bunyan's Christian said, 'Whither must I fly?' Then said Evangelist pointing with a finger over a very wide gate, 'Do you see yonder wicket gate?' The man said 'No'. Then said the other, 'Do you see yonder shining light?' He said, 'I think I do'. Then he said, 'Keep that light in your eye'.

What shining light can be kept in your eye? Is it money, a successful department, personal fame, contribution to medical science, service to patients? I feel It is important to have some guiding principle. Thinking about a standard or aim leads us to the original symbol of medicine, namely the snake on a pole. The staff of Aesculapius has represented medicine since about 800 BC. This is a staff with one snake wound round it. Greek mythology describes how he discovered a magical herb when he observed a snake rejuvenate a previously dead companion by placing the herb in its mouth. No one knows why the Caduceus, which is two snakes intertwined, has gradually been chosen to represent medicine, though it didn't appear until after 1800. It is particularly significant when two wings are added on top of the snakes to make the medical symbol of that into Hermes or Mercury. Everyone knows that Hermes is the God of Commerce. So you can choose whether you wish to have a single snake or the double snake with the wings as your guiding light.

One cannot discuss the snake without referring to the serpent which Moses set up on a pole, which we might like to think of as the basis of the medical symbol,

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although it does not appear to be so. But I think it is relevant to us. Perhaps it is a symbol of the faith which patients have in the medical profession, but it is interesting that having given the people this symbol of healing, God should tell Heziakah after many years to destroy it. I think there are two things we can learn from that. Perhaps the snake had served its usefulness and we should not hesitate to cut out anything that has ceased to be useful and move on to the new. I think the medical profession has been exemplary in moving with the times. However, I believe the greater meaning for us is that the snake was to be destroyed because the people came to worship it and to burn incense to it. In an oration such as this, one is in danger of worshipping the profession and the art of medicine, so that something which was given to us for the good of our patients can be a thing to be worshipped and admired for itself.

Our primary purpose in medicine, therefore, should not be to improve the profession, improve our hospital, or to improve our medical standing or income, which are legitimate and right in their place, but come under the worship of the snake. These all can be taken from you, but what you have done for your patients, never. It seems to me we have been given gifts and trained in healing, and that the shining light towards which we should aim should be the simple task of doing the best for our patients. Our success and failure may be judged on that. So, I welcome you to a wonderful profession where the roses greatly outnumber the thorns, and on behalf of the medical staff, may I wish you a safe, satisfying and successful pilgrimage.

I should like to express my sincere thanks to Professor Robin Shanks and Dr Noel Wright for their help during this survey.

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Considerations for smoking advice in pregnancy

A E Hill

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SUMMARY

Cigarette smoking in pregnancy was the single most important preventable factor identified when determinants of birth weight were studied in 375 pregnancies. Current professional intervention was not effective in reducing cigarette consumption in pregnancy in these mothers in Londonderry, of whom only 19 (5%) became ex-smokers. Present health education, which emphasises impaired fetal growth and wellbeing, had its greatest effect amongst primigravid smokers of whom 32% made some reduction in cigarette consumption. Maternal expectation of birth weight differed significantly between non-smokers, light to moderate, and heavy smokers (8.2 lb, 7.9 lb, 7.3 lb respectively). 54% of multiparous smokers expected the birth weight to be similar to the birth weight in previous pregnancies. Of the women who reduced smoking, 57% did so for the baby, 23% because they found the habit less pleasurable during pregnancy and 13% because of professional advice. Maternal expectation of birth weight is one factor which negates the slant of current health education advice in pregnancy. An anti-smoking programme aimed at protecting the fetus from the harmful effects of cigarettes may produce optimal results when targeted at primary school-aged children in whom the smoking habit is less firmly established.

INTRODUCTION

Cigarette smoking in pregnancy was the single most important preventable factor identified when determinants of birth weight were considered in Irish newborns.[1] Most adults in Britain, irrespective of social class, are now non-smokers.[2] From 1972 to 1982 the proportion of men who were heavy smokers (20 or more cigarettes per day) decreased but no such change has been seen in women who smoke heavily, who are mostly in social class five.[2] The most effective way of persuading mothers to curtail smoking in pregnancy is not known. Methods used have largely concentrated on informing mothers of the hazards of smoking to the baby. It has been shown that those who gave up smoking during pregnancy in a study of London mothers were significantly better informed about fetal problems than those who continued to smoke.[3]

In Londonderry, an area with a high proportion of older, parous mothers, cigarette smoking in pregnancy is prevalent. Current professional advice does not appear effective in reducing the habit. This paper examines the expectations of birth weight in pregnant women in relation to cigarette consumption and considers maternal explanations for reduced smoking in pregnancy.

Altnagelvin Hospital, Londonderry.
A E Hill, MB, MRCP, DRCOG, Senior Registrar.
Correspondence to Dr A E Hill, The Royal Belfast Hospital for Sick Children, Belfast BT12 6BE.

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METHODS AND PATIENTS

Three hundred and seventy-five mothers of consecutive, singleton, term deliveries at the Altnagelvin Hospital, Londonderry, were interviewed on the first or second postnatal day. The mothers were asked to complete a questionnaire under supervision which was designed to record maternal smoking habits and attitudes to smoking during pregnancy, the birth weight the mother had expected prior to the infant's birth and the reason for the estimated weight, and additional socio-cultural details regarding alcohol, education and employment. Maternal weight, height and other obstetric details were obtained from the medical records. Infant gestational age and anthropometric measurements were taken by methods previously described.[1] The mothers and infants were divided into three groups according to the average number of cigarettes smoked per day: non-smoker, mild to moderately heavy smoker (1–19), heavy smoker (20 or more). Where a range of cigarettes smoked was given, the upper figure was used. Statistical analyses were applied using chi squared and the one way analysis of variance.

RESULTS

Data from 375 mothers was analysed. Twenty-seven (7.2%) of the population were under 20 and 52 (13.8%) over 35 years. One hundred and forty-one (37.6%) were primigravid and 66 (17.6%) had had four or more pregnancies (grand multiparity). Other maternal characteristics are shown in Table I. When obstetric factors were considered, there was no statistical significance between the smoking groups for parity, previous large or small for gestational age infants or the incidence of pre-eclampsia. Respiratory illness occurred more frequently in the heavy smokers ($p = 0.004$). Although maternal height and weight at booking were similar in the three groups, the weight at delivery, the mid upper arm circumference at delivery and the weight gain per week were significantly different ($p = 0.0004$, $p = 0.0006$, $p = 0.003$ respectively) with the heavy smokers having lowest values. Adverse socio-cultural factors (young age, unmarried, unemployed, from a large family, no school examinations, alcohol consumption during pregnancy) were over-represented in the heavy smoking group and reached statistical significance. (On chi-squared tests, $p = 0.03$, 0.008, 0.003, 0.015, 0.001, 0.000 respectively). The heavy smoking group was least likely to want to breast-feed.

TABLE I

Maternal characteristics: 375 mothers. Mean ± SD, or percentage of total group

Age (years)	27.2 ± 61
Parity	1.7 ± 2
Married	89.6%
Sibs	6.0 ± 3.5
Gestation at booking (weeks)	19.9 ± 8.3
Smoking prior to pregnancy	50.0%
Smoking during pregnancy	45.0%
Alcohol during pregnancy	32.0%
Husband unemployed	36.6%
Social class 4 or 5	30.0%

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The anthropometric profile of the infants in relation to the pattern of smoking is shown in Table II. There was a significant effect of smoking in reducing birth weight, length and head circumference, and this was not accounted for by any possible difference in gestational age. The birth weight differential between non-smokers and smokers for gestational ages 37 to 41 weeks is shown in the Figure.

TABLE II

Anthropometric profile of infants (mean ± SD)

	Non-smokers	*Smokers*		*p value**
		light to moderate	*heavy*	
	n = 205	*n = 120*	*n = 50*	
Birth weight (kg)	3.5 ± 0.5	3.3 ± 0.4	3.2 ± 0.4	0.0000
Length (cm)	51.7 ± 2.2	50.6 ± 2.2	50.2 ± 2.2	0.0000
Head circumference (cm)	35.4 ± 1.3	35.0 ± 1.2	34.9 ± 1.4	0.004
Arm circumference (cm)	10.6 ± 0.9	10.2 ± 0.8	10.2 ± 0.8	0.03
Gestational age (wks)	39.4 ± 1.2	39.4 ± 1.1	39.4 ± 1.2	0.92

*One-way analysis of variance.

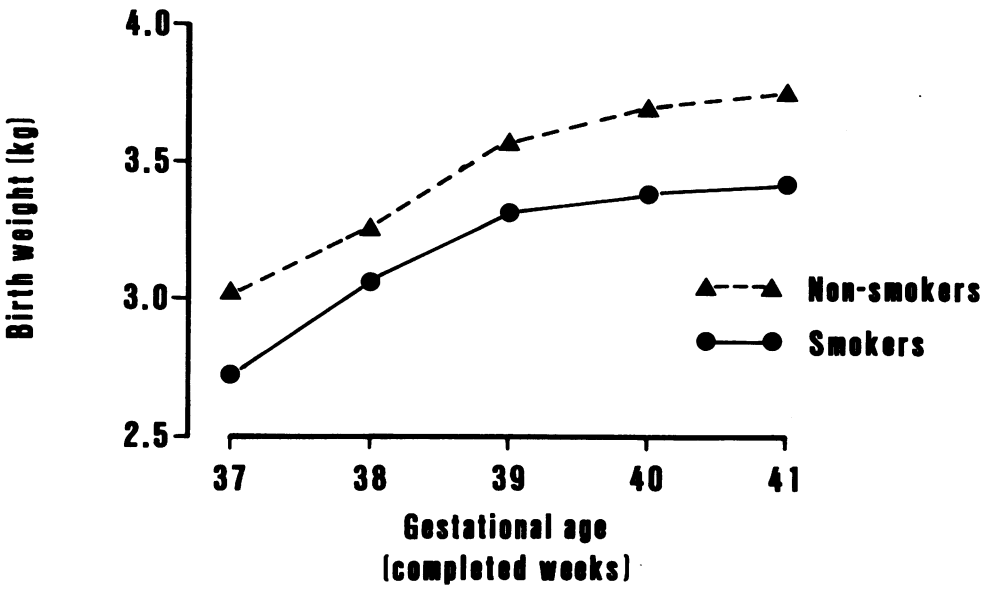

Figure. Graph of birth weight in relation to gestational age for non-smokers and smokers.

The average number of cigarettes smoked per day fell from 18 before pregnancy to 13.5 during pregnancy. Smoking profile in relation to parity is shown in Table III. Of the 109 mothers who reduced smoking, only 19 became non-smokers. A significantly larger number of primipara decreased smoking as compared with mothers with one to three children, or those with four or more, 32%, 29% and 24% respectively, ($p < 0.05$).

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TABLE III

(i) *Smoking profile by parity and* (ii) *Explanation by parity for smoking decrease in 88 and 109 women who reduced cigarette consumption*

	Parity			
	0	*1–3*	*⩾4*	*Total*
(i)				
Smoking profile	n = 141	n = 168	n = 66	n = 375
Non-smoker	73 (52%)	86 (51%)	29 (44%)	188 (50%)
Smoking unchanged	15 (11%)	25 (15%)	17 (26%)	57 (15%)
Increased smoking	8 (5%)	9 (5%)	4 (6%)	21 (6%)
Decreased smoking	45 (32%)	48 (29%)	16 (24%)	109 (29%)
(ii)				
Reason smoking decreased	n = 35	n = 40	n = 13	88 (81%)
'For baby'	20 (57%)	22 (55%)	8 (62%)	50 (57%)
'Didn't feel like it'	9 (26%)	8 (20%)	3 (23%)	20 (23%)
'Professional said'	4 (11%)	7 (17%)	1 (7.5%)	12 (13%)
Other reason	2 (6%)	3 (7%)	1 (7.5%)	6 (7%)

Maternal expectation of birth weight was analysed in relation to smoking pattern. The non-smokers expected significantly higher birth weights than either of the smoking groups: estimated mean birth weights 8.2 lb ± 1.2, 7.9 lb ± 1.4, 7.3 lb ± 1.5 ($p = 0.0001$) for non-smokers, light/moderate and heavy smokers respectively. The reasons most frequently given for the estimated birth weight by primigravid patients were 'The doctor said so' or 'It is an average or normal birth weight' (72%). The reason most frequently given by multiparous patients was 'Same as last time' (54%). The frequency of the reason 'Same as last time' varied with the smoking pattern — in multiparous non-smokers 57%, in those who either increased smoking or smoked the same 54% and 50% respectively, and in those who decreased smoking 37%. Only one woman said she expected her infant to be small because of smoking during pregnancy.

Responses to the questions 'Why did you cut down your cigarettes?' were received from 88 (81%) of the 109 who did reduce smoking and are shown in the second part of Table III; the majority reduced their habit 'for the baby'.

DISCUSSION

The questionnaire format, which was used post-natally to document the number of cigarettes smoked both before pregnancy and after the mother knew she was pregnant, has limitations posed by the truthfulness and recall of the patient. Variations in cigarette consumption, which are known to exist during pregnancy, have not been assessed. Effects of cigarette smoking on pregnancy have been reported in the first trimester, with an increase in spontaneous abortions amongst smoking mothers.[4] Late fetal and neonatal mortality rates are similarly increased[5] and studies on birth weight patterns from various populations have consistently demonstrated that the distribution shifts downwards in proportion to the number

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of cigarettes smoked.[6] This study confirms this effect in babies carried to term. The socio-economic background of heavy smokers in this population differed significantly from the non-smokers. It has been shown that the effects of cigarette smoking on fetal weight are evident after allowance has been made for age, parity and social class differences which exist between smokers and non-smokers.[7]

In the present study 10% of the smokers completely curtailed the habit during pregnancy. There was no reduction in the mean birth weight when non-smoking and ex-smoking mothers were compared (3551g and 3555g respectively). Martin has noted that 13% of mothers stopped smoking in pregnancy[8] and Butler and Alberman reported that the average birth weight and perinatal mortality rate in mothers who gave up smoking by the fourth month of pregnancy was the same as in non-smokers.[7] The most effective way of persuading mothers to curtail smoking in pregnancy is not known. Sixty-four per cent of smokers reduced cigarette consumption during pregnancy, and, when asked why, most commonly said it was for the sake of the baby. Almost a quarter reduced smoking, however, because the habit was less enjoyable in pregnancy and the heavy smokers gave this as the reason as often as any other. Dalton et al[3] in a study of low social class mothers in London noted that health education posters and leaflets did little to increase the percentage of women admitting knowledge of the dangers of smoking or the proportion who stopped smoking. Those who gave up smoking during pregnancy were significantly better informed about fetal hazard than those who continued to smoke. Donovan, in a randomised controlled trial of anti-smoking advice in pregnancy, saw a reduction in cigarettes smoked in the test group, but no significant increase in birth weight, despite intensive individual smoking advice at each antenatal visit.[9] The reasons for the failure to influence birth weight were not clear but it may have been that those counselled did not reduce their smoking sufficiently.

The multiparous women in the present study were less likely to alter their smoking pattern in pregnancy than the primigravida. The majority of parous mothers (54%) anticipated that the birth weight of the infant would be similar to that in previous pregnancies. Part of the present health education message which highlights compromised growth as a result of maternal cigarette smoking, in itself, is unlikely to motivate such a mother to curtail her smoking. Graham has found that one of the critical variables in the continuation of smoking in pregnancy is not ignorance of 'the facts', but rather the credibility the individual accorded to these facts in particular and to scientific knowledge in general.[10]

Maternal attitudes to cigarette smoking in pregnancy are complex. The way smoking mothers perceive their smoking habit during pregnancy remains relatively uninvestigated but must have important implications for the timing and the content of health education. Co-ordinated, concentrated smoking advice from general practitioner, health visitor, midwife and obstetrician from early pregnancy has had limited success.[9] Any programme of intervention aimed at altering a mother's smoking habit once she is pregnant seems doomed to failure, because attitudes and expectations which work to negate professional advice appear to be well established long before this. A health education programme is needed which will influence the potential mother before she ever starts to smoke. The timing of this needs to be brought forward to precede the teenage years and to become an increasing part of primary education. Social class trends in birth weight are due, for the greater part, to heavier smoking during pregnancy of women in the lower social classes. If we seek to ameliorate some of the influences

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of home on smoking behaviour, we will need to establish non-smoking behaviour as the norm in society. To this end I would envisage a primary school-based health education programme. Before this or any new method was operational, the psychological aspects of the approach towards the potential mothers and fathers of tomorrow would need to be evaluated.

I should like to thank the mothers, the midwives and the consultants in the obstetric and paediatric departments of Altnagelvin Hospital for their help and encouragement during this study and the Department of Computing and Medical Statistics, Queen's University, Belfast, for assistance with statistical analysis.

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Multidisciplinary assessment of applicants for residential accommodation

M J P Power, I C Taylor, J G McConnell

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SUMMARY

Fifty of 62 applicants for residential accommodation underwent assessment at a geriatric day hospital. Twenty-five were suitable, 11 were suitable following rehabilitation and 14 were unsuitable for placement in residential accommodation. Around 35% of all applicants were not assessed. Seventy-nine per cent of assessed applicants, without dementia, either were unsure of how their application had been initiated or did not understand the implications of a move to residential accommodation. Twenty-two per cent of all applicants assessed were taking four or more drugs. To maximise the use of residential accommodation, all applicants should be assessed to reduce inappropriate referrals.

INTRODUCTION

Current population trends indicate an increasing number of very old people in our community for at least the next two decades.[1] This will lead to an increasing burden on both hospital and community resources. Residential homes managed by the Department of Health and Social Services in Northern Ireland form an important part of community care for the elderly. Inefficient use of this resource will not only affect the elderly in the community but will increase the length of stay of the elderly in hospital. It therefore becomes increasingly important that the limited resource of residential accommodation is closely matched to need. Recent studies in England and Scotland have shown the benefits of assessing applicants.[2, 3, 4, 5] A district social services department, with the permission of local general practitioners, was asked to refer all applicants for residential accommodation from home for a multidisciplinary assessment at a geriatric day hospital. Present criteria for admission to residential accommodation include that the elderly person be independently mobile with or without mechanical assistance, be independent in activities of daily living (washing, toileting, dressing), or require minimal assistance or supervision only. The aim of the study was to see if multidisciplinary assessment is useful both to the applicant and to those responsible for making the final decision to admit the elderly person to residential accommodation.

MATERIALS AND METHODS

Between August 1985 and January 1987, 62 applicants for residential accommodation were referred for multidisciplinary assessment. Their mean age

Geriatric Medical Unit, The Ulster Hospital, Dundonald, BT16 0RH.
M J P Power, MB, MRCP, Senior Registrar.
I C Taylor, BSc, MD, MRCP, Consultant Geriatrician.
J G McConnell, BSc, MD, MRCP, Consultant Geriatrician.
Correspondence to Dr Taylor.

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was 82.6 years (range 72–94). Applicants were followed up at six to nine months after initial assessment to determine the outcome.

The initial multidisciplinary assessment included:

(a) Interview with a social worker to discuss the applicant's personal situation, the reasons for the application and their understanding of residential accommodation.

(b) Medical screening to assess mental function and any underlying medical problems, especially remediable ones.

(c) Physiotherapy and occupational therapy assessment to consider problems of mobility or ability to perform activities of daily living such as dressing and attending to personal hygiene.

Standard assessment forms were used to reduce inter-observer variation and to provide a framework for decision-making. For confused applicants, information was obtained from relatives and/or social services. For all applicants, information was sought from their general practitioner and local social services. Each applicant had the following tests carried out: mental test score,[6] full blood count, erythrocyte sedimentation rate, a thyroid function test, serum urea and electrolytes, vitamin B12 and folate, calcium, phosphate and alkaline phosphatase levels, and a chest X-ray. A decision on suitability for residential accommodation was taken after discussion among the assessors. Any necessary treatment or rehabilitation was carried out.

If an applicant was deemed unsuitable, it was recommended whether they should remain at home with or without increased social services support, or obtain sheltered accommodation.

Following these decisions the applicants were placed in one of four groups, A, B, C or D (Table I).

TABLE I

Assessment groups decided by the multidisciplinary assessment team after the initial attendance at the geriatric day hospital

	Number of subjects	*Number with dementia*
A — Suitable for residential accommodation	25	16
B — Suitable for residential accommodation after rehabilitation at geriatric day hospital	11	
C — Unsuitable for residential accommodation	14	5
D — Did not attend	12	

RESULTS

Assessment groups

Fifty of the 62 applicants referred had multidisciplinary assessment (Table I). There were 8 males and 42 females. Four were living with relatives, the remainder were living alone — 27 with support from family and/or social services, 18 in sheltered accommodation and one with no support. Fourteen subjects were assessed to be unsuitable for residential accommodation (Group C), 13 being considered to be too fit. Of these, seven required no change in their

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accommodation or level of social services support, three were recommended for sheltered accommodation and three required increased social services support at home. The remaining subject in Group C was too dependent for residential accommodation. Group B subjects (rehabilitation) attended the geriatric day hospital for a mean of 46 days (range 10–95). Eight had medical treatment as well as rehabilitation. Of the 12 applicants who failed to attend (Group D), three were dead, two had been admitted to residential accommodation as emergencies, three were in hospital, three refused to attend and one was in private care.

Follow-up

Ten of the 36 applicants in Groups A and B were in residential accommodation six to nine months after assessment, while 15 were still on the waiting list (Table II). Five of the remaining 11 were dead at follow-up. Of those in Group C, one was in residential accommodation and four were on the waiting list contrary to our recommendations. Thirty (60%) of all those assessed (Groups A, B and C) were either on the waiting list or in residential accommodation at follow-up and 20 of them attended for re-assessment. Only one showed functional deterioration and required a further course of rehabilitation. The 10 who did not attend for re-assessment were still at home.

TABLE II

Placement of applicants for residential accommodation 6–9 months after their initial assessment

Group	*Residential accommodation*		*Home*	*Dead*	*Private care*	*Total*
	Placed	*Waiting list*				
A	8	10	3	4	0	25
B	2	5	1	1	2	11
C	1	4	7	0	2	14
D	4	0	2	4	2	12
TOTAL	15	19	13	9	6	62

Mental test score

Twenty-one (42%) of all those assessed had dementia (mental test score six or less out of 10). Five of them were considered more suitable for residential accommodation for the confused elderly and one was referred for psychogeriatric assessment. Thirty-four per cent of those without dementia were considered to be too fit for residential accommodation compared with 19% of those with dementia. Fifteen of the 29 without dementia had a poor understanding of what a residential home was, and eight were unsure why or how the application had come about.

Medical screening

This revealed a number of previously undiagnosed problems, the majority of which were treatable (Table III). No applicant required hospital admission at initial assessment, but six of those who failed to keep their appointment were either dead or in hospital when called for. Eleven (22%) of all applicants assessed were

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taking four or more drugs and 24 drugs were discontinued in these applicants because of side effects, drug interaction or lack of indication. The majority of discontinued drugs were either diuretics, or drugs prescribed for 'dizziness' — notably prochlorperazine which is a potent cause of hypotension, Parkinsonism and falls in the elderly.[7]

TABLE III

Medical problems discovered at assessment in 50 applicants for residential accommodation

Diagnosis	*Number of subjects*
Inappropriate drug therapy	11
Falls due to gait abnormalities	6
Poor vision	4
Anaemia	3
Transient ischaemic attacks	3
Deafness	2
Faecal impaction	2
Biochemical osteomalacia	2
Osteoarthrosis	2
Hypothyroidism	1
Carcinoma of the lung	1

Non-referral

It was clear that in a home-based assessment not all applicants for residential accommodation would be assessed. Because geriatric and social services catchment areas are not co-terminous, it was not possible to obtain precise figures. From figures obtained from social services it would appear that around 35% of all applicants for residential homes in our area had not been referred to us for assessment during the study period.

DISCUSSION

In many areas the availability of places in residential accommodation is exceeded by demand.[8] With increasing numbers of elderly people, especially those over 85 years of age in the community, this situation is likely to worsen. DHSS-recommended norms for places in residential homes and homes for the confused elderly are 24 and three per thousand over 65 years respectively. In our catchment area the actual corresponding figures are 10.8 and 4.5 per thousand. In order to use this limited resource more efficiently there is an urgent need for careful assessment of applicants prior to permanent placement in residential accommodation.

In contrast to other studies,[2, 3, 4, 5] we assessed thirteen (26%) of our applicants as too fit for residential accommodation. Some otherwise 'fit' elderly people require placement in residential accommodation for important psychological reasons. This figure strongly suggests that people are being referred for residential accommodation without due consideration of alternative means of community support. When an elderly person experiences difficulty in living at home,

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residential accommodation should not necessarily be the first response. All applicants in this study were living at home at the time of the initial assessment, whereas in previous studies some or all subjects were already in residential accommodation or in hospital at assessment.[2, 3, 4, 5] This only partly explains why our group of applicants seemed fitter than those of other studies. Criteria for admission to residential homes can vary according to number of available places, staffing and dependency levels of residents. Unlike other reports this study revealed only one subject who was too dependent for residential accommodation.

There was a significant level of undiagnosed medical illness in applicants, most of which was treatable. Many applicants were on inappropriate drug therapy, confirming findings in previous studies.[2, 3, 4, 5] Since the reason for non-attendance in three applicants was death and in a further five was emergency admission to hospital or residential accommodation, early assessment may be important to reduce morbidity and mortality. The numbers with dementia in this study were similar to other studies.[3, 9] The presence of dementia increased the likelihood of need for admission to residential accommodation.

An important finding in this study was that 79% of screened applicants without dementia were unsure how their application had come about or did not understand the implications of a move to residential accommodation — sometimes a relative had taken the decision-making out of the elderly person's hands. This, along with the finding that some applicants were too fit for residential accommodation, points to the need for a careful review of how applications are initiated and processed. Current discussion by the Review Group on Residential Accommodation of the Eastern Health and Social Services Board is therefore timely. Any regular visitor to a residential home will see increasing numbers of frail elderly people. We recommend a change in the philosophy of residential care with a greater nursing input and increased staffing levels to deal with these changes. The alternative is an inappropriate burden of care being placed on the hospital services. Private residential and nursing homes can provide a suitable alternative in some cases, but there is no proper assessment procedure and, despite DHSS supplementation, the cost can be prohibitive.

Placement panels for residential accommodation found the multidisciplinary assessment reports helpful in allocating places although five subjects whom we considered unsuitable were accepted. Around 35% of applicants referred were not assessed by us. There are several reasons for this: those who had emergency admission to residential accommodation or admission from hospital and those referred for residential homes for the confused elderly were not included in our study. Of more immediate concern are applicants who did not wish to be assessed and/or whose general practitioners did not give consent. If multidisciplinary assessment is to be worthwhile it should cover all residential home applicants. For this to happen, assessments might have to be mandatory. Assessments at hospital are stressful so we would recommend home assessment where possible. Good social and medical assessments are important, with referrals to other members of the multidisciplinary team where appropriate. The whole purpose of assessment is to establish a knowledge base which will allow effective, appropriate care which meets the elderly person's real needs.[10] Such assessments should extend to DHSS-supplemented private sector care of the elderly.

In conclusion we recommend:

1. All applicants for residential accommodation should be seen at an early stage by experienced social work staff for discussion and counselling.

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2. All applicants should be assessed by medical and social work staff experienced in care of the elderly to avoid inappropriate admissions and reduce morbidity. The main philosophy of this assessment should be to assess the elderly person's needs and to try and maintain them in their own home or other appropriate setting for as long as possible, rather than merely to assess their suitability for residential accommodation.
3. Since occupational therapy assessment provides pertinent information on ability to cope with everyday life it should be part of the assessment process in the majority of applicants.
4. Physiotherapy assessment should be carried out on a more selective basis.
5. Where possible, assessments should be carried out in the applicant's home.
6. All elderly people admitted as an emergency to residential accommodation and being considered for permanent placement should be assessed as soon as possible as they may be suffering from a treatable condition and therefore be inappropriately placed.[5]
7. All DHSS-supplemented applicants for private residential and private nursing homes should be assessed — this will have manpower implications.

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Renal replacement treatment for diabetic nephropathy in Northern Ireland 1979-1987

A P Maxwell, J F Douglas, C C Doherty, W E Nelson, R A Donaldson, Mary G McGeown

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SUMMARY

Twenty-three patients with end-stage renal failure due to diabetic nephropathy received renal replacement treatment. All patients had insulin-dependent diabetes mellitus. Nineteen transplants were performed in seventeen patients. Two-year graft survival for all transplants was 74% with a two-year patient survival post-transplantation of 81%. Overall two-year patient survival was 73%, compared with 82% in non-diabetic patients receiving renal replacement treatment. In diabetic patients accepted for treatment there was a high incidence of non-renal complications, particularly vascular disease. An aggressive approach to the treatment of vascular disease in these patients may improve overall survival rates.

INTRODUCTION

The diagnosis of diabetic nephropathy in an individual diabetic patient is usually presumed in the presence of proteinuria and impaired renal function in association with diabetic retinopathy. Hypertension is almost invariably present at some stage and common histological features are diffuse and nodular glomerulosclerosis. The nephropathy may be compounded by other diabetic renal complications such as atheromatous renovascular disease, pyelonephritis, papillary necrosis and neurogenic bladder.

The prevalence of diabetic nephropathy increases with the duration of diabetes to a peak of around 20% after 20 – 25 years.[1] After 25 years the annual incidence declines, indicating that the development of nephropathy is not solely dependent on the duration of diabetes.[2] Renal failure secondary to diabetic nephropathy was responsible for over one quarter of the deaths in one study[3] of patients in whom insulin-dependent diabetes mellitus was diagnosed before 31 years of age. This and other similar studies[4, 5] emphasised the need for more liberal extension of renal replacement programmes to diabetic uraemic patients. The proportion of new patients with end-stage renal failure due to diabetes mellitus referred for renal replacement treatment is rising in Northern Ireland. To date, all patients have had insulin-dependent (type 1) diabetes mellitus. In the United Kingdom

Renal Unit, Belfast City Hospital, Belfast, Northern Ireland, BT9 7AB.
A P Maxwell, MB, BCh, MRCP, Registrar.
J F Douglas, MA, BCL, MB, BCh, FRCP, Consultant Nephrologist.
C C Doherty, MD, MRCP, Consultant Nephrologist.
W E Nelson, MD, MRCP, Consultant Nephrologist.
R A Donaldson, BSc, MB, BCh, FRCS, Consultant Urologist.
Mary G McGeown, CBE, DSc(Hon), MD, PhD, FRCP, FRCPEd, FRCPI, Consultant Nephrologist, Physician in Administrative Charge.
Correspondence to Dr Maxwell.

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overall, the proportion of diabetics with end-stage renal failure accepted for treatment had risen to 11.1% in 1984,[6] a figure still well below those of 25% in the USA[7] (although more elderly non-insulin dependent diabetic patients are treated there) and over 15% in some European countries;[6] this may reflect a lower prevalence and/or under-referral of diabetic renal failure. In diabetic patients with end-stage renal failure who receive renal transplants, the outcome compares favourably with that of non-diabetic patients in some centres.[8] However, sub-groups of diabetic patients with severe vascular disease have shown a clear excess of graft failure and patient mortality.[9]

This paper outlines the experience of the Renal Unit at the Belfast City Hospital in treating diabetic nephropathy between April 1979 and May 1987.

PATIENTS

Twenty-three patients (14 male, 9 female) were accepted for renal replacement treatment. Their ages ranged from 23 to 62 years (mean 37.3) at time of referral. Renal replacement treatment commenced at a mean age of 38.3 years. The duration of diabetes ranged from 8 to 38 years (mean 21.7). Assessment for renal replacement treatment was generally late in the progression of renal failure: the mean serum creatinine was 773 μmol/l at referral, and mean creatinine clearance 10 ml/min. Twenty-one percent of patients were referred as an emergency with life-threatening illness including pulmonary oedema. Diabetic nephropathy was a presumptive clinical diagnosis in all patients based on the presence of diabetic retinopathy, heavy proteinuria and impaired renal function. Nephrectomy and post-mortem pathological evidence confirmed the presence of diabetic nephropathy with typical histological features of diffuse and nodular glomerulosclerosis, arteriosclerotic lesions and interstitial fibrosis in all five cases examined; atheromatous renovascular disease was present in addition in three of these cases.

TABLE

Complications present at renal referral in 23 insulin-dependent diabetic patients

	Number	
EYES		
Retinopathy	23	100%
Proliferative retinopathy requiring laser therapy	16	70%
Blindness	5	22%
CARDIOVASCULAR		
Abnormal ECG	18	78%
ECG criteria for myocardial infarction	7	30%
Hypertension (BP > 150/90)	22	96%
Angina	5	20%
CEREBROVASCULAR		
Previous stroke	2	9%
PERIPHERAL VASCULAR DISEASE		
Intermittent claudication/ischaemia	8	34%
Amputation	2	8%
PERIPHERAL NEUROPATHY	13	57%

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There was a high prevalence of non-renal diabetic complications in patients at the time of referral (Table). Retinopathy was present in all and 70% had received laser photocoagulation for proliferative retinopathy; 22% were registered blind. Cardiovascular disease was common: ECG recordings were reviewed by one investigator (APM) — 78% had an abnormal ECG, and 30% had the electrocardiographic criteria for a previous myocardial infarction. Angina pectoris was present in 22%, and hypertension (BP >150/90) in 96%. The prevalence of cerebrovascular disease was lower, 9% having had a completed stroke while carotid bruits were heard in 9%. Evidence of peripheral neuropathy was found in 57% based on clinical symptoms and signs, and nerve conduction studies in 13% of these patients confirmed the clinical diagnosis. Peripheral vascular disease had produced considerable morbidity in 34% of patients, and above-knee amputations had been required in 8% (two cases). Three further patients had above-knee amputations shortly after commencing renal replacement treatment.

RENAL REPLACEMENT THERAPY

The mean age for commencing treatment was 38 years. Dialysis therapy was started in 60% of patients for refractory pulmonary oedema or hypertension, and in a further 30% for uraemic symptoms. Initial treatment was haemodialysis for the majority (61%), peritoneal dialysis in 30% and transplantation in 9%.

Haemodialysis. Fourteen patients initially started treatment with haemodialysis. Three patients had been commenced on intermittent peritoneal dialysis and were later changed to haemodialysis for reasons of infection and availability. Two patients changed from haemodialysis to chronic ambulatory peritoneal dialysis following assessment of chest pain. Both patients underwent coronary artery bypass grafting for multi-vessel coronary artery disease and were managed post-operatively by peritoneal dialysis. Vascular access has proved difficult in several patients.

Intermittent peritoneal dialysis. Five patients began intermittent peritoneal dialysis: one was subsequently transplanted, one was trained for chronic ambulatory peritoneal dialysis and three patients were transferred to haemodialysis.

Chronic ambulatory peritoneal dialysis. Two patients started this as their initial treatment and three are currently maintained on this mode of dialysis. Two of these patients manage the technique successfully despite being registered blind.

Transplantation. Nineteen renal transplants have been performed on 17 patients. Eighteen transplants were cadaver grafts, and one patient received a graft from a living related donor which never functioned because of intra-operative hypotension. The mean duration of dialysis pre-transplant was 9.9 months. Thirteen patients survive (11 with functioning grafts and two having returned to dialysis). Two-year graft survival is 74% including one operative death and two cases of primary non-function due to graft artery thrombosis. Comparable results in this unit for two-year graft survival in non-diabetics was 81%, and two-year patient survival 82%.

Operative problems encountered included atheromatous pelvic vessels in five recipients, in one case necessitating formal revascularisation at the time of transplant. Wound infections occurred in 38% post-operatively. Early transplant rejection (within three months) occurred in 10 grafts and required treatment with

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high dose oral prednisolone. In three cases plasma exchange was included in the management of rejection. Eight of the 10 grafts continued to function. Diabetic transplant recipients were treated with a combination of low dose prednisolone (20 mg daily reduced in stages over 18 months to 10 mg daily) and azathioprine (3mg/kg).

Mortality. Seventeen of the 23 patients accepted for treatment survived to May 1987. Four transplant recipients died and post mortem examinations were obtained in three cases. One patient died from a combination of bacterial infection and transplant rejection; the other two suffered myocardial infarctions. The fourth transplant patient died suddenly at home after recent onset of atrial fibrillation, presumably also on the basis of ischaemic heart disease. The two deaths in non-transplanted patients occurred during haemodialysis, one from a myocardial infarction (confirmed by post-mortem examination revealing extensive triple vessel coronary artery disease which had been clinically silent), the other due to a cerebrovascular accident. Overall the two-year patient survival for all modalities of treatment during the period studied was 73.5%.

DISCUSSION

In Northern Ireland the incidence of new cases of diabetic renal failure has been estimated at 7.6 cases/million/year, which is lower than in other UK regions reported.[10] The number of patients who have received treatment to date is less than the estimated number for a variety of reasons including limited resources and under-referral. The absence of older patients with non-insulin-dependent diabetes mellitus in our treatment group probably represents under-referral of such patients because of perceived bias towards treatment of younger patients with end-stage renal failure — a policy initially reflecting medical opinion [11] but latterly reflecting constraints on the expansion of the renal failure service. From 1979 the rate of referral of patients with diabetic nephropathy to the Renal Unit has been rising, and many insulin-dependent and non-insulin-dependent diabetic patients will reach the renal replacement programme this year. In view of the expected increase in the number of diabetic patients for assessment we have reviewed our preliminary experience in treatment of end-stage diabetic nephropathy.

The diabetic patients reported in this paper represent 7.3% of the total number of new patients accepted for renal replacement therapy in Northern Ireland between 1979 and 1987. However, over the last two years (June 1985 to May 1987), diabetic patients accounted for 12% of the total treated, indicating that increasing numbers of diabetic patients are receiving renal replacement therapy. Some diabetic patients who were referred in the period reported were not treated for reasons of advanced age, dependency and overwhelming medical complications. These criteria were equally applied to non-diabetic patients referred for assessment and reflect earlier restrictions on the provision of care for patients with renal failure. The number of diabetic patients with renal impairment in Northern Ireland is unknown as the recent survey [10] only selected for study diabetics with advanced renal failure (serum creatinine >500 µmol/l and urea >25 mmol/l). Most of the patients reported here were referred at a late stage of their renal disease and required renal replacement, on average, within a year. Pulmonary oedema and hypertension were the main indications for starting treatment. A high incidence of non-renal diabetic complications was evident which posed many management problems. While vascular disease did not prove a contraindication to renal replacement treatment, our experience suggests that an aggressive approach to

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this frequently co-existing problem is essential. Myocardial infarction was implicated in 50% of the deaths in diabetic patients in the period reported. Significant ischaemic heart disease may be present [12] without symptoms, often declaring itself only after treatment for end-stage renal failure has commenced. Two patients with multi-vessel coronary artery disease, which became symptomatic on haemodialysis treatment, underwent coronary artery bypass grafting procedures. These patients were managed post-operatively by peritoneal dialysis and later trained for continuous ambulatory peritoneal dialysis. Some centres have recommended coronary artery angiography as a routine pre-transplant screening procedure in diabetic uraemic patients [13] and others have reported that diabetic patients with significant coronary artery disease had a worse prognosis for all modalities of treatment but a trend to better survival following transplantation.[14] In those patients with significant peripheral vascular disease transplantation may worsen lower limb ischaemic by a 'vascular steal' phenomenon resulting in amputation in some cases shortly after a transplant procedure. Rimmer and colleagues [9] found higher mortality and graft loss following transplantation in patients with pre-existing atherosclerotic vascular disease and concluded this should influence the choice between therapeutic alternatives in diabetics with end-stage renal failure. Revascularisation procedures might favourably alter the survival characteristics of this group following transplantation. The vascular disease may lead to difficulty with vascular access.[11] Other problems encountered include autonomic neuropathy (urinary retention, hypotension), gastrointestinal symptoms and a wasting syndrome.[15] The two-year graft survival of 74% and two year patient survival of 81% post transplantation must in this series be interpreted with caution in view of the relatively small numbers of patients involved and the relatively short period of follow-up. Diabetic transplant recipients were treated with conventional immunosuppressive therapy (azathioprine and low dose prednisolone) and it is still our policy to reserve cyclosporin therapy for patients (diabetic or non-diabetic) who are intolerant of azathioprine.

The treatment of diabetic patients with end-stage renal failure by dialysis [16, 17] and transplantation [8] improves patient survival. The cost of renal replacement treatment for diabetic patients is higher than that for non-diabetic patients, one estimate suggesting transplantation is one-third more expensive.[18] New patients with end-stage renal failure due to diabetic nephropathy are now forming an increasing proportion of the numbers accepted for treatment in the United Kingdom,[6] and concern has been expressed that many patients who could benefit from treatment are probably not receiving it.[19, 20] The survey of diabetic renal failure for 1985 conducted by the Joint Working Party of the Renal Association, the British Diabetic Association and the Royal College of Physicians concluded that up to 40% of patients suitable for treatment were not receiving it.[10]

While prevention of diabetic nephropathy remains the ultimate goal, current aims are earlier referral and joint management of the patient by nephrologists and diabetologists, with stricter blood pressure [21, 22, 23] and metabolic control [24] attempting to reduce the rate of progression of diabetic nephropathy. End-stage renal failure in diabetic uraemic patients can be managed successfully with a combination of dialysis and transplantation. Vascular disease, whilst an important factor determining eventual outcome, has not proved a contraindication to treatment. The quality of life for the individual must also be considered in choosing between therapeutic alternatives in renal replacement treatment for diabetic patients.

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Royal Victoria Hospital

FRIENDS OF THE ROYAL

The formation of a 'Friends of the Royal' group is currently under way. In order to explain the aims and object of the Friends and elect a Committee, an inaugural meeting is being held at 3.30 pm on Wednesday, 15 June, 1988 in Bostock House Ballroom, RVH. It is hoped that this organisation will promote the work of the Royal through the Ladies Committee, Working Mens Committee and Ex-Patients Guild and will in addition take on other projects to assist the Hospital. Anyone interested in our endeavours is welcome at this meeting. We look forward to your support and ideas.

Ingrid Allen

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Distribution of properdin factor B and glyoxalase I (further genetic markers) in Northern Ireland

P Hamilton, D Middleton

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SUMMARY

The normal distribution in Northern Ireland of the genetic markers properdin factor B (Bf) and glyoxalase I (GLO) are described. These markers are available for comparison in disease studies, family studies and paternity studies.

INTRODUCTION

The HLA chromosomal region, or major histocompatibility complex — located on the short arm of chromosome six — is a cluster of at least three gene families. These are class I (HLA-A, HLA-B, HLA-C), class II (HLA-DP, HLA-DQ, HLA-DR) and class III (C2 and C4 of the classical complement pathway and properdin factor B (Bf) of the alternative complement pathway). HLA antigens have been associated with susceptibility to certain diseases, for example HLA-B27 and ankylosing spondylitis, HLA-DR2 and multiple sclerosis, and HLA-DR3 and HLA-DR4 in insulin-dependent diabetes.[1]

The genetic marker Bf has been reported to be associated with certain diseases. There are four alleles of Bf which can be distinguished by their electrophoretic mobilities — BfS1, BfS, BfF and BfF1. In insulin-dependent diabetes, Kirk noted a marked association between the early onset of the disease and BfF1.[2] If the patients were 10 years or less at onset of the disease the BfF1 frequency was 19.1%, whereas the normal BfF1 frequency of 1.3% was present in patients acquiring the disease at 20 years or later. Dyer, in a study of 37 patients with idiopathic membranous nephropathy, demonstrated an increase in BfF1, 21.6% of patients being positive compared with 1.7% of controls.[3]

The gene which regulates the enzyme glyoxalase I (GLO) is positioned at a distance of five centimorgans from the MHC loci. This enzyme, which is present in both red blood cells and leucocytes, catalyses the irreversible conversion of reduced glutathione (GSH) and methyl glyoxal to S-lactoyl glutathione. Two major GLO alleles exist, GLO1 and GLO2, giving rise to three phenotypes, GLO-1, GLO-2 and GLO 2-1.

MATERIALS AND METHODS

Determination of Bf was made on 189 serum samples from blood donors who attended the Northern Ireland Blood Transfusion Service, using the method of

Northern Ireland Tissue Typing Service, Belfast City Hospital, Belfast, BT9 7AD.
P Hamilton, FIMLS, Senior Medical Laboratory Scientific Officer.
D Middleton, PhD, Top Grade Scientist.
Correspondence to Dr Middleton.

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Alper in which high voltage electrophoresis is followed by immunofixation with specific antibody to Bf.[4] It was also possible to test 272 heterogeneous serum samples taken from people who had been HLA – A, – B typed. These sera samples were taken from kidney recipients and donors, multiple sclerosis patients and members of staff.

Thin layer gels (0.5 – 1.0mm thick) of 1% agarose in barbitone/calcium lactate buffer pH8.6 were prepared on sheets of Gelbond (124 x 258 x 2mm) (Pharmacia, Uppsala, Sweden) supported by glass plates. The gels were electrophoresed at 20v/cm for two hours. Following separation, anti-factor B (Atlantic Antibodies, Westbrook, Maine, USA) was applied evenly to the gel, which was then incubated for one hour at 37°C. Four saline washes were carried out on the gel to remove excess antibody and unfixed protein bands before staining with coomassie blue R for 15 minutes. The excess stain was removed with water, and the gels were allowed to dry before reading the Bf phenotypes directly from the gels.

A modification of the starch gel electrophoretic technique[5] was used to determine glyoxalase (GLO). Red cell haemolysates, obtained from 200 citrate phosphate dextrose normal blood samples were applied to 1% agarose gels made up in tris-barbital buffer pH8.0. Each gel was electrophoresed for one hour at 17v/cm. Following separation, the gel was incubated for 20 minutes at 37°C in a buffered methyl glyoxal and GSH mixture, then overlaid with a second gel containing dichlorophenol-indophenol (DCIP) and 3 – (4,5,dimethylthiazol – 2yl) – 2,5,diphenyltetrazolium bromide (MTT). The GLO phenotypes were read according to the position of the clear bands in the blue background after approximately 20 minutes. The development of the background colour is based on GSH, in the absence of GLO, being involved in the non-enzymatic reduction of DCIP, which is then oxidised by MTT to produce a blue formazan precipitate. Where GLO is present, the above reaction does not occur, as GSH is utilized as a co-factor in the irreversible enzymatic conversion of methyl glyoxal to S-lactoyl glutathione, resulting in the clear bands in the blue formazan background.

RESULTS

It was only possible to demonstrate six of the 10 possible Bf phenotypes in the 189 samples tested because of the low prevalences of F1 and S1. The distribution of these phenotypes and the Bf gene frequencies in Northern Ireland is given in Table I. The absence of silent alleles in the Bf system which allows homozygosity to be attributed to BfS and BfF individuals permits direct determination of the gene frequencies.

TABLE I

Distribution of Bf phenotypes and gene frequencies in a Northern Ireland population (n = 189)

Phenotypes		*(%)*	*Gene frequencies*	
SS	105	55.6		
SF	65	34.4	S	0.749
SS1	5	2.6	F	0.228
SF1	3	1.6	S1	0.013
FF	10	5.3	F1	0.011
FF1	1	0.5		

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In the HLA – A, – B typed samples, all HLA – B7 (n = 76) and HLA – B8 (n = 89) positive individuals were positive for BfS, and of the 31 individuals positive for HLA – B35, 30 were positive for BfF. The Bf phenotype distribution in this heterogeneous group was very similar to that in the normal population: the phenotype SS was present in 60% of the samples and the phenotype SF in 35% of the samples.

The distribution of the GLO phenotypes and gene frequencies is presented in Table II. Calculation of the gene frequency was based on there being two alleles of GLO present at an autosomal locus. No observations contradicted this hypothesis.[5]

TABLE II

Distribution of GLO phenotypes and gene frequencies in a Northern Ireland population (n = 200)

Phenotypes		*Gene frequencies*	
GLO – 1	47		
GLO 2 – 1	92	GLO1	0.535
GLO – 2	61	GLO2	0.465

DISCUSSION

The frequency of each of the four Bf alleles in a Northern Ireland population was compared with normal populations in Manchester,[3] Wales[6] and north-east England.[7] No significant difference ($p > 0.05$) in the frequency of BfS1, BfS, BfF or BfF1 was detected. Previous reports have shown that linkage disequilibrium exists between some HLA – B antigens and Bf variants: established examples are HLA – B8 and BfS, HLA – B35 and BfF,[8] and B7 and BfS.[9] Although in this study it has not been possible to determine if these HLA – B antigens are on the same haplotype as the Bf variant, these associations appear to be present in our population.

No significant difference ($p > 0.05$) was demonstrated in the frequency of GLO – 1 or GLO – 2 in this Northern Ireland population when compared with a normal Caucasoid population studied in the VIIIth Histocompatibility Workshop.[10]

We are now able to study the frequency of Bf and GLO, as well as the HLA antigens, in various diseases. The determination of Bf and GLO markers will also be of use in family studies, with reference to recombination of genes, and in paternity studies.

We thank Miss Denise Fulton for typing the manuscript.

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THE ROYAL MEDICAL BENEVOLENT FUND

I would like to introduce you to the Royal Medical Benevolent Fund. This charity — *YOUR CHARITY* — has been helping members of the medical profession and their families for 152 years. Its help is generous and unfortunately the need is still great. Please help us to help others.

Lesley E E Donaldson (Area Visitor/Guild Secretary N.I.)
19 Cabin Hill Gardens, Belfast BT5 7AP.

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Cellobiose/mannitol sugar absorption test in patients with dermatitis herpetiformis: a preliminary report

J G M Bridges, T J Robinson, K W Scott, H N Pearson

Accepted 26 January 1988.

SUMMARY

An abnormal cellobiose/mannitol ratio is present in new patients presenting with dermatitis herpetiformis and in patients who have never adhered to a strict gluten-free diet. In patients with dermatitis herpetiformis in remission on a strict gluten-free diet, small bowel absorption as measured by the cellobiose/mannitol ratio is normal. It is suggested that cellobiose/mannitol ratio absorption is a useful screening test for the intestinal abnormality associated with dermatitis herpetiformis and failure of the test to return to normal could suggest poor dietary compliance.

INTRODUCTION

Dermatitis herpetiformis was shown to be associated with jejunal villous atrophy by Marks et al[1] and changes similar to those in coeliac disease have been found.[2,3] Although between one-third and one-fifth of dermatitis herpetiformis patients do not demonstrate jejunal villous changes,[3-5] most patients show an improvement on a gluten-free diet. Tests of absorption tend to be normal or only slightly abnormal.[5-7] The mucosal changes are said to be patchy,[8] so that simple screening tests have not been found useful in assessing mucosal recovery and repeated jejunal biopsies may not give accurate information. A reproducible absorption test would be invaluable in the clinical management of these patients, as it would appear that clinical remission occurs only after the mucosa has returned to normal.[9,10]

Abnormal sugar absorption has been shown to be a useful measure for screening jejunal function in patients with coeliac disease.[11,12] Cellobiose/mannitol absorption has been well studied with reports of clear separation between active coeliac disease and coeliac disease in remission.[13] We felt that cellobiose/mannitol absorption might be a useful test in patients with dermatitis herpetiformis. Abnormal persistent defects in permeability have been demonstrated in coeliac disease and dermatitis herpetiformis patients, even if the disease is in remission and the mucosa has returned to normal using the 51 chromium-labelled

Craigavon Area Hospital, Craigavon, Co. Armagh BT63 5QQ.
J G M Bridges, MB, BCh, Senior House Officer.
T J Robinson, MD, FRCP, Consultant Physician.
K W Scott, MB, FRCP(I), Consultant Dermatologist.
H N Pearson, BSc, Senior Scientific Officer.
Correspondence to Dr Robinson.

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ethylene-diamine tetra-acetate (51 Cr/EDTA) absorption test.[14] However, in the clinical management of patients, a simple demonstration of recovery of absorptive ability is all that is required.

The present study comprises a group of patients with dermatitis herpetiformis who have been followed up for a number of years, and five patients, newly diagnosed, who have not yet started on the gluten-free diet. We measured the cellobiose/mannitol ratio, assessed the clinical response to a gluten-free diet, and looked for a correlation between the original jejunal biopsy and clinical response.

MATERIALS AND METHODS

A total of 20 patients with dermatitis herpetiformis was investigated of whom five were newly diagnosed cases (two men and three women; mean age 42 years; range 23–62 years) taking a normal diet. The remaining 15 patients (10 men and 5 women of mean age 45 years; range 19–78 years) had been on gluten-free diet treatment for a mean of eight years (range 1–13 years) at the time of the study. A jejunal biopsy had been taken before the start of gluten restriction. The diagnosis of dermatitis herpetiformis was based on the characteristic body distribution of an intensely pruritic, polymorphic and vesicular skin rash together with the demonstration by immunofluorescence of IgA deposits in the papillary dermis of clinically uninvolved skin.[15, 16] In a few patients presenting before the availability of IgA immunofluorescence, a classical skin rash associated with abnormal histology and a clinical response to dapsone was accepted as diagnostic of dermatitis herpetiformis.

Biopsy of the upper jejunal mucosa was carried out using a Watson capsule placed endoscopically in the second part of the duodenum and the capsule then advanced 25 cm distally from the end of the endoscope. The mucosa was examined under the dissecting microscope, following which routine microscopic sections were stained with haematoxylin and eosin. The biopsies were graded as normal, partial villous atrophy and subtotal villous atrophy. Increased intra-epithelial lymphocytic infiltration in the presence of otherwise normal villi has been accepted as partial villous atrophy.

Cellobiose / mannitol absorption

A sugar solution composed of 2 gm mannitol, 5 gm cellobiose, 20 gm lactose, 20 gm sucrose, made up to 150 ml was the test solution. After an overnight fast, the patient emptied his bladder and drank the solution over a period of five minutes. All urine passed within the next five hours was collected. The amount of mannitol and cellobiose passed in the urine was measured as outlined by Strobel et al.[13] The results were expressed in percentages and the final ratio of percentage recovery of cellobiose to percentage recovery of mannitol was calculated as the cellobiose/mannitol ratio. A cellobiose/mannitol ratio of less than 0.037 was accepted as normal.

RESULTS

The cellobiose/mannitol ratio in the five untreated dermatitis herpetiformis patients was at or above the normal range (0.037–0.19). The remaining 15 patients had been diagnosed one to 13 years previously, and cellobiose/mannitol ratio varied from 0.001–0.22 (Table I).

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TABLE I

Dermatitis herpetiformis: cellobiose / mannitol ratio (normal < 0.037) in five newly diagnosed (untreated), and 15 previously diagnosed patients

Cellobiose / mannitol ratio			
Newly diagnosed	*Previously diagnosed*		*Never on gluten restriction*
	Gluten-free diet	*Stopped diet*	
0.037*	0.024	0.001	0.220*
0.190*	0.006	0.019	0.064*
0.064*	0.005	0.014	0.011
0.110*	0.002		0.039*
0.047*	0.010		
	0.023		
	0.031		
	0.047*		

*Abnormal result.

Seven of the previously treated patients had no skin symptoms and a normal cellobiose/mannitol ratio: six were on a gluten-free diet, but one who had been on a diet for a number of years had found it inconvenient and continued on dapsone (Table II). Eight previously treated patients had persistent or intermittent blistering and itch of the skin. Two had been on a strict gluten-free diet for seven years, but had stopped, and they had a normal cellobiose/mannitol ratio at the time of study. Two patients continued on a strict gluten-free diet — one of these had a normal ratio and the other who had been on a gluten-free diet for only two months, with noticeable improvement of the skin, had an abnormal ratio. Four of these 15 patients had never been on a gluten-free diet and had persisting skin problems; three of these had an abnormal ratio.

TABLE II

Skin response and cellobiose / mannitol ratio

Cellobiose / mannitol ratio	
Complete remission	*Active skin disease*
0.024	0.019
0.006	0.014
0.005	0.031
0.002	0.047*
0.010	† 0.220*
0.023	† 0.064*
† 0.001	† 0.011
	0.039*

*Abnormal results.

† Patients on dapsone.

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Thus, of nine patients (five new, four previously treated) not on a gluten-free diet at the time of study, eight had an abnormal cellobiose/mannitol ratio, and of 11 patients who had been or were on a strict gluten-free diet, 10 had a normal ratio. The one patient with an abnormal ratio had been on a diet for only two months.

The initial jejunal biopsy was reported as showing partial villous atrophy in all five newly diagnosed patients who were on a normal diet and in seven of the 15 previously diagnosed patients. Four of the old patients had sub-total villous atrophy and the remaining four had a normal or minimal change mucosa. Subsequent response to a gluten-free diet did not depend on the initial biopsy. Eleven patients had used dapsone 100mg daily with relief of symptoms at the onset of the disease. Three patients with poor dietary compliance continued to use it intermittently with some benefit and two out of three have an abnormal cellobiose/mannitol ratio. One patient who had been well controlled on diet has a normal ratio and continues to use dapsone, as he finds it more convenient than a strict gluten-free diet.

DISCUSSION

This study indicates a possible clinical use for the measurement of the cellobiose/mannitol ratio. The abnormal ratio in new untreated patients could be used as a screening test either prior to or instead of a jejunal biopsy. There is very good separation between patients well controlled on a gluten-free diet and new patients. Repeated tests of patients who are on a gluten-free diet could measure both dietary compliance and an improvement in jejunal mucosa. Poor dietary compliance appears to be linked to poor control of the skin condition.[9] The actual defect in dermatitis herpetiformis is not clear but there is evidence to suggest that an abnormal leaky jejunal mucosa leads to an absorption of immune complexes which, deposited in the skin, can lead to intense pruritis.[17, 18]

Published results tend to be confusing about the benefit of a gluten-free diet.[9, 19, 20] In this study, however, it would appear that strict gluten exclusion leads to good control of dermatitis herpetiformis. The speed of response varies in that the rash responds rapidly to gluten exclusion in some patients, perhaps within a few weeks, while in other patients a gluten-free diet is necessary for many months before remission is obtained.

If jejunal 'healing' is necessary for a remission of dermatitis herpetiformis it would be interesting to follow up a group of patients with dermatitis herpetiformis taking serial measurements of the cellobiose/mannitol ratio to see if permeability has to return to normal before skin remission occurs. Our preliminary results suggest that cellobiose/mannitol absorption studies may be a good measure of jejunal healing and presumably of dietary compliance. Further investigation is necessary to find if skin improvement occurs at the same time as, or subsequent to jejunal recovery.

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The effect of exposure to mupirocin on the survival and recovery of sensitive *Staphylococcus aureus* strains

J G Barr, G M Hogg, E T M Smyth

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SUMMARY

A significant percentage survival, representing a large number of viable cells, can occur in a Staphylococcus aureus *population exposed to concentrations of mupirocin up to 1,000 times the minimum inhibitory concentration. An elevated ratio of minimum inhibitory to minimum bactericidal concentration of mupirocin with staphylococci was recorded. Staphylococci which survive exposure to mupirocin may be less sensitive to the subsequent bactericidal action of the antibiotic. The observations may explain the re-colonisation with staphylococci seen in some studies.*

INTRODUCTION

The predominant reservoir of *Staphylococcus aureus* is man: about 15 - 35% of the population are colonised with *S aureus* in the anterior nares from which the organism can readily be disseminated to other parts of the body and to the environment. A decrease in host resistance to infection with *S aureus* can be associated with a wide variety of clinical syndromes of differing severity. The management of these infections may be complicated when multi-resistant staphylococci are encountered. Although the occurrence of multiple-antibiotic resistant strains of *S aureus* is still only about 5%, they can cause serious problems [1, 2] especially in association with hospital special care units where the opportunities for cross-infection and emergence of multiple resistance are high.

Any infection control policy aimed at reducing the incidence of staphylococcal infections due to multiple resistant strains must include attempts to eradicate nasal carriage. The topical use of antibiotics or skin disinfectants has had variable success, is unreliable, and may have inherent disadvantages in respect of resistance development.[3] Resistance to chlorhexidine, commonly used as a topical agent to reduce staphylococcal carriage, has recently been reported.[4]

A new compound, mupirocin (derived from *Pseudomonas fluorescens*, pseudomonic acid) has been shown to be effective in the eradication of *S aureus* from the anterior nares [5, 6] and the skin [7, 8] when applied as a 2% ointment. Casewell and Hill [9] have also shown that *in vitro* mupirocin behaves as a bactericidal antibiotic giving 99.9% reduction in the number of viable staphylococci within

Department of Bacteriology, Royal Victoria Hospital, Belfast BT12 6BA.
J G Barr, PhD, MRCPath, Top Grade Microbiologist.
G M Hogg, BSc, FIMLS, Scientist.
E T M Smyth, MB, MRCPath, Consultant Bacteriologist.

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24 hours' exposure. The aim of this study was to investigate the effect on staphylococci of various concentrations of mupirocin at differing inoculum concentrations and at different stages of growth and to examine the characteristics of surviving bacteria.

MATERIALS AND METHODS

Mupirocin (Beecham Pharmaceuticals, Worthing) was supplied in pellet form as the sodium salt. Each pellet, weighing 20.5 mg, was dissolved in distilled water to give the appropriate antibiotic concentration, sterilised by filtration (0.22μ, Millipore UK) and used on the day of preparation.

Eighteen clinical isolates of *S aureus* were examined, in addition to two type cultures, *S aureus* (NCTC 6571) and a methicillin resistant *S aureus* (NCTC 10442). All strains were grown on nutrient agar slopes for 24 hours at 37°C, stored at 4°C and sub-cultured weekly. Mueller-Hinton (M-H) agar (CM 337), blood agar (Gibco 152-0600) containing 5% (v/v) defibrinated horse blood, and quarter-strength Ringer's solution (Oxoid BR 52) were prepared according to the manufacturer's instructions. Nutrient agar slopes (Oxoid R2) were supplied by the manufacturer.

Minimum inhibitory concentrations were carried out using the agar incorporation method. Four-hour peptone water cultures were used as inocula, and dispensed using a multipoint inoculator (Denley Instruments, Ltd) on to M-H agar plates containing two-fold serial dilutions of mupirocin. Inoculated plates were incubated aerobically at 37°C and examined for growth after 24 hours. The lowest concentration which gave a complete absence of growth was recorded as the minimum inhibitory concentration. Drug-free plates were inoculated as controls and *S aureus* NCTC 6571 and *S aureus* NCTC 10442 were included in each test set as control organisms.

Minimum bactericidal concentrations were determined using the velvet pad transfer method. The surface of each agar plate used in the previous determinations were sampled with a sterile velvet pad. The pad was then transferred on to the surface of an M-H agar plate. Following incubation at 37°C for 48 hours the minimum bactericidal concentration was calculated as the lowest concentration of antibiotic yielding no growth after velvet pad sub-culture.

Viable bacterial counts were determined by the spread plate method using quarter-strength Ringer's solution as diluent and 5% blood agar as plating medium. After incubation of plates at 37°C, results were expressed as the number of colony-forming units per ml of the bacterial suspension. To remove mupirocin from bacterial suspensions prior to enumeration of viable bacteria, unit volumes of suspension were centrifuged at 3,000 rpm for five minutes, the supernatant decanted, the cellular deposit washed once in broth, and then resuspended in broth to the original volume.

Staphylococci were grown in either M-H broth or quarter-strength Ringer's solution in the presence or absence of specified concentrations of mupirocin. Two inoculum levels were investigated, approximately 10^7 and 10^5 colony-forming units per ml. Cultures were sampled at intervals during the incubation period and viable counts performed. All experiments were carried out in triplicate.

The ability of *S aureus* to initiate growth in liquid medium after exposure to mupirocin was measured by conductance using the 112L microbiological growth analyser (Malthus Instruments, UK, Ltd). Staphylococci were exposed to

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mupirocin at a concentration of 256 mg/l for different periods. The antibiotic was removed from each sample by the methods described and 1 ml of this suspension was used to inoculate 9 ml broth contained in 10 ml capacity growth cells. Identical cells containing antibiotic only or bacterial suspension only were incorporated as controls. All tests were incubated at 37°C and performed in duplicate. Changes in conductance of the growth medium due to bacterial growth were monitored and recorded; conductance measurements were made in microsiemens (μs). The interpretation of the conductance measurements in relation to microbial growth were as described in detail by Baynes, Comrie and Prain.[10] As growth proceeds, the conductivity of the medium increases, and a growth curve can be plotted of time (hours) against conductivity (μs). To test the stability of the mupirocin throughout each investigation, an aliquot of culture was taken before and after completion of each experiment. Each aliquot was filter-sterilised (0.22μ) and divided into two portions. One portion was treated with esterase in the ratio of one volume porcine liver esterase suspension (Sigma Diagnostics E-3128) to one volume of test suspension for 30 minutes in a 37°C waterbath. The other portion was untreated. Both treated and untreated samples were titrated in quarter-strength Ringer's solution (two-fold dilutions) beyond the minimum inhibitory concentration of the organism used. Single drops of 25 μl of each titration dilution were dispensed on to the surface of pre-dried blood agar plates and allowed to absorb into the medium undisturbed. After absorption, the plates were flooded with a log phase broth culture of *S aureus* NCTC 6571 incubated 18 hours at 37°C. Plates were examined for zones of clearing indicating the presence of the antibiotic.

RESULTS

The survival of *S aureus* NCTC 6571 in M-H broth when exposed to concentrations of 2, 16 and 256 mg/l mupirocin is shown in Fig 1. Using both high and low inoculum levels it was shown that after an initial increase in viable count over a three-hour period, a stationary phase of growth was established for a period of 24 hours. Where the initial inoculum level was exposed to 16 or 256 mg/l mupirocin, the viable count fell to less than 10 colony-forming units per ml, representing at least a 99.9% reduction of viable cells. For cells exposed to 2 mg/l mupirocin a 99.8% reduction in viable count was achieved. Where the higher inoculum level was examined, there was a reduction in viable count of 0, 98 and 99.6% after exposure to concentration of 2, 16 and 256 mg/l mupirocin. Under the conditions described with the higher inoculum level, reductions in viable count of 98% and 99.6% corresponded to counts of surviving bacteria after 120 hours' exposure to mupirocin of 2×10^5 and 4×10^4 colony-forming units per ml respectively.

The survival kinetics for a further four strains of *S aureus* showed a similar pattern to that obtained for *S aureus* NCTC 6571. The influence of these inoculum sizes on the percentage survival of three multiple-resistant (including methicillin resistance) staphylococci and *S aureus* NCTC 10442 was studied after 64 hours' continuous exposure to 256 mg/l mupirocin, and significant numbers of all strains survived this exposure. A higher percentage survival was found with the higher inoculum level. No significant difference in results was obtained with inocula prepared from lag phase, logarithmic phase or stationary phase cells. The effect of mupirocin (256 mg/l) on the survival of staphylococci at different inoculum levels in M-H broth and in quarter-strength Ringer's solution is shown

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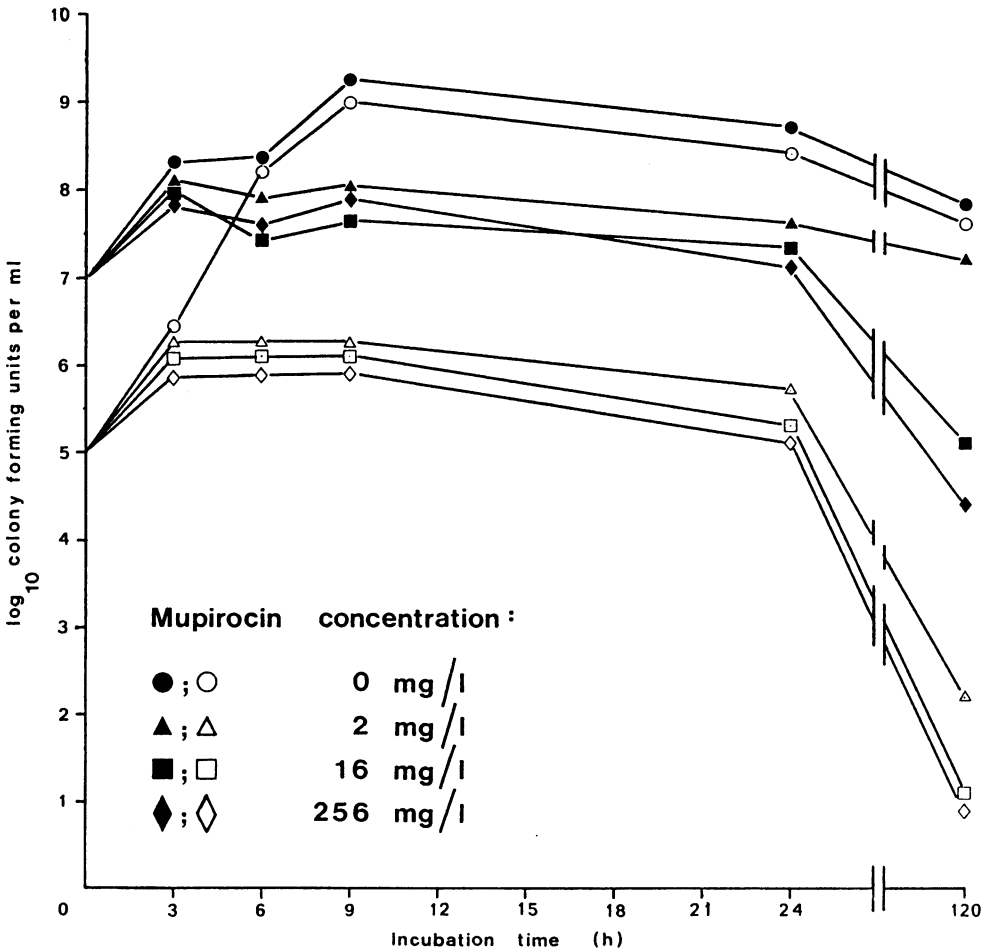

Fig 1. The effect of mupirocin on the survival of *S aureus* NCTC 6571 at two inoculum levels (1×10^7 colony-forming units per ml, black symbols; 1×10^5 colony-forming units per ml, open symbols).

in Fig 2. Cells grown in M-H broth alone (Fig 2B) demonstrated a normal growth curve for uninhibited cells, whereas cells exposed to 256 mg/l mupirocin in M-H broth showed a stationary phase of growth established over 24 hours followed by a decline in viable count. The decline in viable count shown in M-H broth confirmed the effect of inoculum on the percentage survival after 24 hours' and 120 hours' exposure to mupirocin. The viability of cells in quarter-strength Ringer's solution began to decline, even in the absence of mupirocin (Fig 2A). This was most marked with the lower initial inoculum level. However, in the presence of mupirocin, no surviving cells could be detected after 48 hours with the lower initial inoculum level or after 120 hours with the higher initial inoculum level.

A relationship was established between the inoculum size of *S aureus* NCTC 6571 and time of detection of conductance change using the Malthus microbiological growth analyser. The detection time varied between three hours and 15 hours for

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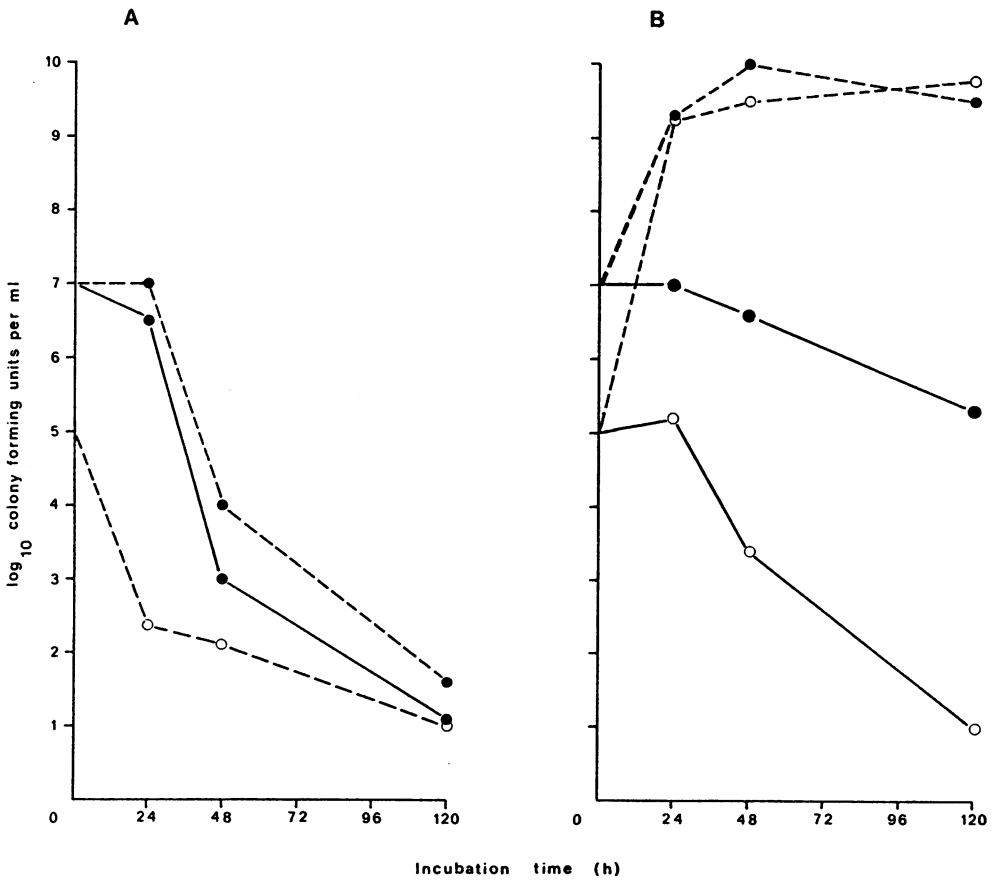

Fig 2. Survival of *S aureus* NCTC 6571 at two different inoculum levels with and without concomitant exposure to mupirocin at a concentration of 256mg/l. Both a non-nutritional (quarter-strength Ringer's solution — Fig 2A) and nutritional (M-H broth — Fig 2B) environment were tested. The initial inoculum contained cells in a logarithmic phase of growth.

Inoculum levels: 1×10^7 colony-forming units per ml (black symbols), 1×10^5 colony-forming units per ml (open symbols). Solid line ——— with mupirocin, dotted line — — — without mupirocin.

inocula varying from 2×10^9 to 2×10^2 colony-forming units per ml. *S aureus* cells exposed to mupirocin (2mg/l, 16mg/l, or 256mg/l) for different periods (24, 48 and 72 hours) showed no residual effect of mupirocin. In each case the time of detection of conductance change was as expected from the calculated viable count of mupirocin-exposed cells, and the pattern and magnitude of conductance of cells pre-exposed to mupirocin was the same as the control unexposed organism.

The minimum inhibitory and minimal bactericidal concentrations of mupirocin were determined before and after treatment of *S aureus* cultures for 24 hours with the same drug (Table). When the pre-treated cells were recovered on antibiotic-free M-H agar after overnight incubation at 37°C, no change in their susceptibility to mupirocin was noted. The minimal bactericidal concentration was generally four-fold higher than the minimal inhibitory concentration. When

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TABLE

The effect of previous exposure to mupirocin on the minimum inhibitory concentrations (MIC) and minimal bactericidal concentration (MBC) of mupirocin on Staphylococcus aureus *(18 clinical isolates, and two control cultures, NCTC 6571 and 10442)*

	*Study A**		*Study B*		*Study C*	
	Previous exposure		*Previous exposure with cells washed*		*No previous exposure*	
	MIC	MBC	MIC	MBC	MIC	MBC
18 clinical isolates (mean and range)	0.5 (0.25-64)	2.0 (2-64)	0.13 (0.03-32)	64 (16-64)	0.5 (0.25-64)	2.0 (2-64)
2 control cultures (mean)	0.4	2.0	0.03	64	0.4	2

*The previously exposed staphylococci had been grown in a medium containing 256 mg/l mupirocin for 24 hours; the cells were then grown on M-H agar for 16 hours without the antibiotic before further inoculation (Study A), or simply washed free of the antibiotic (Study B). The cells in Study C had not previously been exposed to mupirocin.

mupirocin-treated cells were washed to remove the antibiotic and used directly as an inoculum, the minimal inhibitory concentration generally decreased 1–2 fold; the bactericidal concentration however, exceeded the inhibitory concentration by a factor of 500 for all clinical isolates and type cultures.

No variation in assays of mupirocin activity before and after each experiment was found. This confirmed the stability of the antibiotic in the presence of both quarter-strength Ringer's solution and M-H broth and at 37°C incubation. Bacterial degradation of the antibiotic was not apparent even when broth cultures were tested after several days' continuous incubation of 37°C. Treatment of culture filtrates with esterase negated the titration value in all cases, indicating the continued presence of the antibiotic.

DISCUSSION

S aureus is one of the major causes (20%) of hospital infection[11] which can become life-threatening in debilitated patients.[12] The re-emergence of multiple-resistant, including methicillin-resistant, staphylococci has placed further burdens both on the patient and on hospital personnel with its associated problems in the control of cross-infection.[11, 13] Such were these problems that recently detailed guidelines for the prompt control and management of methicillin-resistant *S aureus* infections were published by the Hospital Infection Society and British Society for Antimicrobial Chemotherapy (1986).[14]

Although staphylococci can be found associated with a wide range of hospital-acquired infection from skin sepsis to endocarditis, the main reservoir for these organisms is the anterior nares, and recently the importance of gastro-intestinal carriage in hospitalised patients has been reported.[15] The problem of carriage of *S aureus* has been reflected in the varied treatment regimens and measures used to control or eliminate this carriage, often resulting in organisms becoming antibiotic-resistant.[3]

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The new topical antibiotic mupirocin seems to satisfy a number of criteria expected of a topical agent. It has a novel mode of action, selective anti-bacterial activity, a total lack of cross-resistance to other antibiotics, and is metabolised and excreted quickly as a non-toxic compound. Another favourable property is its action against multiple-resistant coagulase-positive and coagulase-negative staphylococci.[9] White et al (1984) stated that using experimental inocula of 10^5 colony-forming units per ml, more than 99.9% of *S aureus* were killed in 24 hours.[16] For a number of strains, using similar inoculum concentrations, Casewell and Hill (1984) agreed with this observation, demonstrating that at concentrations of 16mg/l a 99.9% reduction in viable count was achieved by 24 hours and sterility of the culture by 120 hours.[9] In this study we found that with inoculum levels of 10^5 to 10^7 colony-forming units per ml, a significant percentage survival, representing a large number of viable cells, could occur in an *S aureus* population exposed to concentrations of mupirocin up to 256mg/l (equivalent to 1,000 times the minimum inhibitory concentrations). This was demonstrated with a number of strains, including methicillin-resistant *S aureus.*

Surviving cells, if transferred to an environment free of mupirocin, showed the same growth characteristics as unexposed cells. With high inoculum levels, even exposure to 256mg/l for 72 hours could be followed by a rapid recovery when removed to an antibiotic-free environment.

A significant difference was noted between the effect of mupirocin in *S aureus* grown in broth and in quarter-strength Ringer's solution. In quarter-strength Ringer's solution, the slope of the curve for both exposed and non-exposed cells were very similar, suggesting a direct effect of the Ringer's environment. However, no viable cells were recovered after 120 hours, when the initial population of cells were exposed to mupirocin (256mg/l) in a Ringer's salts environment; this suggested that the action of the antibiotic was not altogether dependent on the presence of bacteria in a phase of rapid growth and protein synthesis. This was confirmed by the findings that the antibiotic effect on viable counts of bacteria was not significantly different using lag, logarithmic or stationary phase cells. The ratio of minimal inhibitory to minimal bactericidal concentration of mupirocin with staphylococci is quoted at between 1:8 and 1:32. Our findings demonstrate a ratio of at least 1:128 for cells that have been treated with mupirocin and used directly as inoculum. It appears that cell growth in the presence of mupirocin ceases and will only resume on removal of the antibiotic. The change in the ratio in this case seems to confirm that cells surviving exposure to mupirocin at high concentrations are less sensitive to the subsequent bactericidal action of mupirocin. It is possible that in an *in vitro* situation the cells which survived an initial challenge with high concentrations of mupirocin could be less sensitive to the bactericidal action of subsequent exposure.

These results do not differ very much from the observations of other workers, although with similar inocula we cannot demonstrate such a high percentage loss of viability in the presence of mupirocin. This may be due to some carry-over of mupirocin on plating media. Cells are sufficiently damaged to be inhibited from rapid growth on solid media, but viability can often be demonstrated by velvet pad transfer to fresh media or by prior centrifugation of treated organisms to remove the antibiotic. At inoculum levels higher than 10^5 we find that, even at high mupirocin concentrations, high numbers of organisms can survive for 120 hours and retain their ability to grow again in a manner similar to untreated cells, when environmental conditions become favourable.

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Although the antibacterial properties of mupirocin support its use as a topical antibiotic, we feel that, in some clinical applications (where high concentrations of mupirocin locally challenge high numbers of staphylococci at a micro-environment level), sufficient numbers of viable organisms may remain to initiate re-colonisation. This may explain the low percentage re-colonisation with the same strain of staphylococcus seen in some studies.

The authors would like to thank Beecham Laboratories for supplies of mupirocin ointment, Professor A M Emmerson for his assistance and Mrs C McIlhatton for typing the manuscript. Mupirocin is marketed as Bactroban (Beecham), which is a 2% ointment in a water-miscible macrogel base.

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Sexual problems and their management: a survey of general practice in Northern Ireland

E C O'Gorman, L E Thompson

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SUMMARY

This survey suggests a similar prevalence of sexual problems in general practice in Northern Ireland compared with England and Wales. Of the respondents, 9.6% reported a much higher prevalence which may indicate a greater ability in detecting sexual problems and therefore implies that many cases are being missed. The majority of problems appear to be dealt with in general practice, although referral is often considered, with the Psychosexual Clinic being the preferred option. Difficulties encountered with referral are highlighted by the survey. It is suggested that provision of more information about the clinic, visits by therapists to practices and further training opportunities would help general practitioners in managing sexual problems.

INTRODUCTION

The rate of referral to the Psychosexual Clinic at the Belfast City Hospital is slightly lower than to similar clinics in England and Scotland.[1] Since two-thirds of the referrals to the Belfast clinic come from general practitioners, a questionnaire survey was undertaken to determine whether similar numbers of psychosexual problems are encountered in general practice in Northern Ireland compared with that estimated for Great Britain. This survey also estimated the proportion of psychosexual problems which general practitioners would refer to the clinic, found out the preferred management of these problems, and identified reasons that might hinder general practitioners from referring psychosexual problems.

METHOD

The questionnaire was designed and sent to one hundred general practitioners chosen at random from the list of general practitioners in Northern Ireland. The questions are listed in the results section.

RESULTS

Seventy-three general practitioners completed and returned the questionnaire; another two replied but felt unable to submit results, and twenty-five did not reply.

The Psychosexual Clinic, 68 Fitzwilliam Street (Belfast City Hospital) and the Department of Mental Health, The Queen's University of Belfast.

E C O'Gorman, MD, DPM, FRCPsych, Consultant Psychiatrist and Senior Lecturer in Mental Health, Inishowen, Crumlin, Co. Antrim.

L E Thompson, MB, BCh, DRCOG, MRCGP, General Practitioner.

Correspondence to: Dr E C O'Gorman, 68 Fitzwilliam Street, Belfast BT9 6AX.

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Question 1: Approximately how many cases of sexual dysfunction (impotence, premature ejaculation, frigidity, vaginismus) or deviant sexuality (homosexuality or paedophilia) present to you in practice in a year? Only one doctor replied that he had no cases, and eight (11%) had over 10 cases per year.

TABLE I

Number of cases of sexual dysfunction or deviation presenting to each general practitioner per year

Number of cases	*Number of doctors*	*Percentage*
0	1	1%
1	12	16%
2—5	39	53%
6—10	13	18%
11—20	7	10%
More than 20	1	1%
TOTAL	73	100%

Question 2: What proportion of cases would you refer to the Psychosexual Clinic? Seven doctors (9.6%) would refer all cases, 13 (17.8%) more than half, 39 (53.4%) less than half, and 14 (19.2%) would refer none.

Question 3: Methods of management. Place in order preferred method of management: referral to a psychosexual clinic, to a medical or social agency (physician, endocrinologist, marriage guidance agency or the social services) or management within the practice. Forty-two doctors gave first preference to management within their practice, and 29 preferred referral to the Psychosexual Clinic.

TABLE II

Preferred methods of management

Methods of management	*Preference of management*		
	1st	*2nd*	*3rd*
Referral to Psychosexual Clinic	29	24	9
Referral to a medical or social agency	4	12	34
Management in practice	42	17	6

Three doctors marked two methods as first choice, 19 did not give a second choice, 23 did not give a third choice and one did not indicate any choices.

Question 4: Indicate up to three reasons which might prevent a general practitioner from referring a patient to a psychiatrist or psychosexual clinic if you feel they would strongly influence your decision not to refer. The most important reasons for non-referral were thought by this sample of doctors to be patient dislike of the psychiatric or psychosexual connotation, although distance and delay were also mentioned.

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TABLE III

Reasons which might prevent a general practitioner from referring a patient with a psychosexual problem to a psychiatrist or psychosexual clinic (up to three reasons to be listed)

	Number of doctors	*Percentage*
1. Patient's dislike of referral to a psychiatrist	43	59%
2. Patient's embarrassment at attending a psychosexual clinic	36	49%
3. Disadvantages of the patient being labelled a 'psychiatric case'	32	44%
4. Distance to clinic	31	42%
5. Delay in obtaining an appointment	28	38%
6. No faith in psychiatry or psychosexual clinic	2	3%
7. Exacerbation of the problems by medical intervention	1	1%
8. The problem is felt to be incurable	0	0%

DISCUSSION

Nearly 90% of the general practitioners estimated that they saw between one and 10 cases of sexual dysfunction or deviant sexuality in a year, and about 50% thought that they saw between one and five. Using the 1971 National Morbidity Survey for general practice in England and Wales, Hodgkin[2] calculated a prevalence of 0.3 cases of male sexual dysfunction for 1,000 patients per year and 0.7 cases per 1,000 for female sexual dysfunction. In Northern Ireland the average practice list size at the time of the survey was 1,879, so that one or two cases per year would be expected on that basis. Although the survey can only provide an estimate of the prevalence of sexual problems presenting in general practice in the Province, it would appear that similar numbers of cases of sexual dysfunction present to general practitioners in Northern Ireland as do in England and Wales.

Sixteen per cent of the doctors saw only one case per year, while 9.6% saw between 10 and 20 cases. This might suggest a very large difference in the presentation of sexual problems in these two groups, but may in fact indicate that some general practitioners are more able to detect problems of this type than others. There is evidence to suggest that, while sexual problems are common, being present in 12% of attendants at a family planning clinic in Edinburgh,[3] they are often difficult to identify and only one in 10 patients will spontaneously present.[4] In addition to this evidence, Jachuck[5] found that, even when general practitioners asked their patients on anti-hypertensive therapy if they had sexual difficulties, the patients invariably said 'No', and it was only when the spouses were interviewed that substantial evidence of sexual difficulties was found.

About 10% of general practitioners said they would refer all cases; about 20% said they would refer none, while the largest group said they would refer less than 50%. These results, along with the preferences given for management options, indicate that family doctors in Northern Ireland usually deal with sexual problems

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in their practices, but would often consider referral to the Psychosexual Clinic. Very few said they would refer to another type of specialist or agency. Additional comments indicated that two general practitioners did not know of the Clinic's existence. While these results would give support to the view that general practice is the most appropriate place for the management of the majority of sexual problems, it is clear that specialist help is often required. Although the survey did not uncover any large body of dissatisfaction with the type of service which the Psychosexual Clinic offers, a substantial number of doctors agreed that there were disadvantages involved with referring: these were psychiatric labelling and patient embarrassment, as well as travelling difficulties and appointment delays. It might be of help if more information were available about the specialist services that can be provided by the Clinic.

The problems associated with travelling distances to the Clinic would be alleviated by visits by therapists to practices and health centres. This could support the general practitioners who wish to treat such patients in the primary care setting. These measures would particularly help those doctors who see smaller numbers of sexual problems to acquire clinical competence in this area. More training opportunities for general practitioners in the management of sexual dysfunction could increase awareness of the extent of these problems and emphasise that patients may not need intensive treatment, but can obtain great relief from simply having the 'permission' to talk about their difficulties. They may also be helped by being given small amounts of information as suggested in the PLISSIT model of management.[6] This model is a description of different types of management ranging through P—Permission; LI—Limited information; SS—Specific suggestions; IT—Intensive treatment. The majority of sexual problems, it is suggested, can be treated without the need for specialist intensive treatment.

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Increasing mortality from malignant melanoma among women in Northern Ireland

C M McKee

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SUMMARY

Deaths from malignant melanoma in each part of the British Isles between 1969 and 1984 have been examined. There has been a substantial increase of deaths among women in each part, and an increase of similar magnitude among men in all areas except Northern Ireland. It is suggested that this difference requires further study. The increasing number of deaths from this disease indicates a need for greater public and professional awareness of this potentially preventable and curable condition.

INTRODUCTION

Deaths from malignant melanoma in the United Kingdom have increased substantially in the last 20 years.[1]

A recent report on the incidence of malignant melanoma in Northern Ireland drew attention to an excess of cases among females, and in this review of specimens submitted for pathological examination a female-to-male ratio of almost three-to-one was found.[2] There is a widespread international variation in the female-to-male ratio. British and American studies have reported a female excess,[3, 4] but in Belgium, Holland and Scandinavia, in the period 1955–1974, there was a considerable male excess.[3] I have examined deaths from malignant melanoma over a 16-year period in order to see whether mortality has varied over time in Northern Ireland, and to compare this with the experience in the remaining parts of the British Isles.

METHODS

Deaths from malignant melanoma (ICD-9 Code 172) were extracted from the annual reports of the Registrar-General for Northern Ireland for the years 1969 to 1984. It was not possible to determine the histological type from this source. Corresponding figures for England and Wales, Scotland, and the Republic of Ireland were extracted from OPCS *Mortality statistics,* reports of the Registrar-General for Scotland, and *Reports on vital statistics* from the Central Statistical Office, Dublin. Data for the Republic of Ireland were only analysed for the period 1969 to 1982. The standardised mortality ratios (SMR) were calculated by comparing the annual average number of deaths throughout the period with the age-specific death rates for England and Wales during 1976, the mid-point of

C M McKee, MB, MSc, MRCP, Senior Registrar/Lecturer.
Department of Community Health, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT.

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the period, related to the population estimate for Northern Ireland in that year. The variation of deaths over time were examined by plotting a scattergram and by regression analysis, and the significance of the regression equation was examined by analysis of variance.

RESULTS

One hundred and twenty men and 176 women died from malignant melanoma in Northern Ireland during the period studied. Deaths among males were slightly less common and among females were significantly so in Northern Ireland as compared with England and Wales. The SMR is 0.90 ± 0.16 for males and 0.84 ± 0.12 for females (95% confidence intervals). (British Isles = 1.00).

There was no significant trend of deaths among males ($F = 0.083$, 14 df). There was a significant increase in female deaths throughout the period ($F = 8.09$, 14 df, $p < 0.05$). The regression coefficient is 0.605. The trend represented an increase of 173% or from approximately 6 to 16 deaths per year among females while male deaths remained constant at about eight per year.

When analysed by ten-year age groups, there was no significant difference between the age at death of males and females (Chi squared = 7.43, 14 df).

There was an increase in deaths from melanoma among females in each part of the British Isles, but the increase is greatest in Northern Ireland. This is the only part of the British Isles in which there was not a significant increase in male deaths, and is the only one in which there was a significant difference between the trend of deaths among males and females ($t = 2.25$, $p < 0.05$) (Table).

TABLE

Percentage increase in deaths from malignant melanoma in the British Isles 1969–1984

	Male percentage increase 1969–84		*Female percentage increase 1969–84*		*Ratio of female-to-male deaths*
Northern Ireland	8%	NS (n = 120)	173%	$p < 0.05$ (n = 176)	1.47
England and Wales	96%	$p < 0.001$ (n = 5203)	70%	$p < 0.001$ (n = 6580)	1.26
Scotland	56%	$p < 0.05$ (n = 458)	49%	$p < 0.01$ (n = 703)	1.53
Republic of Ireland*	100%	$p < 0.05$ (n = 188)	101%	$p < 0.05$ (n = 266)	1.41

Increases have been calculated from the equations of the trends.

Sources: Annual reports of the Registrar-General for Northern Ireland, OPCS *Mortality statistics*, Reports of the Registrar-General for Scotland, and *Reports on vital statistics* from the Central Statistical Office, Dublin.

*1969–1982 only.

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DISCUSSION

There has been a rapid rise in the incidence of malignant melanoma among people of Northern European descent in many parts of the world.[5] Total mortality in the British Isles has almost doubled since 1969 and elsewhere: a report from Sweden documented an increase of 7% in both sexes in the period 1959–68.[5] The relationship between lentigo malignant melanoma and ultra-violet light is well recognised, although the association with superficial spreading melanoma and nodular melanoma is less well established.

People of Celtic descent may have a genetic predisposition and studies from Australia[6] and the United States[7] have found that the incidence is significantly higher among those with names suggesting Irish or Scottish ancestry.

In Northern Ireland there is a marked difference in deaths from melanoma in males and females. As it is not necessarily a fatal condition, this may reflect either a difference in incidence or in prognosis. In Gordon and Lowry's comprehensive study of the incidence of melanoma in Northern Ireland in 1974–78 there were 170 females and 61 males.[2] During the same period there were 28 male and 49 female deaths. This suggests a higher case fatality rate among men, which is consistent with the observation that they have a higher proportion of thicker, and therefore more advanced, lesions among biopsies.[2] A higher male case fatality has also been found in England and Wales.[3] Taken with the evidence of increasing mortality, this suggests that the rise in incidence in females in the same population may be very much greater. Walsh and Bharucha[8] in their study of samples submitted for histological examination in 1930, 1955 and 1980, found that the female-to-male ratio in their sample had increased from 1.5:1 in 1955 to 2.3:1 in 1980. It would seem that melanomas are much more common in women but are also likely to be diagnosed earlier.

The reason for the absence of an increase in male deaths in Northern Ireland is uncertain, especially since it is at variance with findings from other parts of the British Isles. It is possible that it may be related to differences in behaviour of male and female holidaymakers from Northern Ireland. A case control study would be useful to examine this hypothesis further. A further possibility is incorrect certification of the cause of death. Although considerable inaccuracies in death certification have been found,[9] a large study of 9,501 deaths[10] comparing clinicians' diagnoses and post-mortem findings established substantial agreement in deaths from many types of neoplasms. The disagreements were more likely to occur in conditions which tend to present with secondary spread, and in which the primary site may be difficult to locate, or among neoplasms which are recognised to present occasionally with non-metastatic complications, such as bronchial neoplasms. It would seem reasonable to suggest that neoplasms of skin would be less likely to be misdiagnosed in comparison with neoplasms of other sites. It is difficult to see how misdiagnosis could consistently and systematically affect one sex to a greater degree than the other over a 16-year period.

Unfortunately, cancer registration in Northern Ireland is incomplete and, when examined in 1983, it was found to have included less than 50% of skin malignancies identified from laboratory records. Coverage of the Province is variable with under-representation of the western part of the Province.[11] It is hoped that improvements in the register will make it possible to investigate future trends.

The observation of lower overall mortality in Northern Ireland as compared with other parts of the United Kingdom is consistent with studies of incidence of

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melanoma based on pathological specimens which have reported an annual incidence in Northern Ireland of 3.12/100,000 population[2] compared with 5.1/100,000 in Scotland.[4] The reasons for this are uncertain and may be related to differences in foreign travel. The expenditure on holidays by Northern Ireland residents is one of the lowest in the United Kingdom and is substantially lower than expenditure by residents of Scotland.[12]

Increasing mortality from melanoma has been actively confronted in several countries. Intensive public health campaigns in Queensland[13] and in Scotland[14] have increased both public awareness and the number of cases that are diagnosed early. Where they are still relatively uncommon they may often be missed by doctors and only recognised at a late stage.[15] Walsh and Bharucha[8] have shown that, although there was some improvement between 1955 and 1980 among the samples in their study, by 1980 50% of lesions involved the reticular dermis or subcutaneous fat, and there had been no increase in the percentage which were confined to the epidermal layer.

Leaflets giving advice on exposure to the sun are now being distributed with inclusive tour tickets through travel agents in Northern Ireland. The leaflets contain advice on the use of sunscreens and recognition of malignant melanomas. This should increase knowledge of the risk from melanoma, and promote earlier detection. There is a need for greater public and professional awareness of this potentially preventable and curable condition.

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Diabetic ketoacidosis in a district general hospital, 1981 – 1986

D J Eedy, A J McNeill, W J Andrews

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SUMMARY

The outcome of therapy of poorly controlled insulin-requiring cases of diabetes mellitus needing admission to a district general hospital from 1981 to 1986 was examined. There were 156 admissions to the hospital, 17 of these classified as severe diabetic ketoacidosis (serum standard bicarbonate less than 14 mmol/l). A 'low dose' insulin regimen was used in each case of severe ketoacidosis. No patient who was admitted died within a six-month period. These figures emphasise the value of a policy of direct hospital admission for poorly controlled diabetics and suggest that early diagnosis in general practice is vital to allow the application of relatively simple and standard hospital treatment.

INTRODUCTION

The outcome of treatment of diabetic ketoacidosis has improved dramatically during this century. In the pre-insulin era, severe ketoacidosis was uniformly fatal. Despite current therapy, recent studies still indicate a mortality of 5 – 10% in good centres, but suggest mortality rates of 20 – 25% in average district hospitals.[1, 2, 3, 4] Diabetic ketoacidosis is the fourth major cause of death in diabetics and the most common in patients under the age of 20 years.[5] Lower mortality figures do not necessarily appear to be related to the centres with the largest number of hospital admissions, suggesting that clinical experience in hospital may not improve diagnosis in the community.[3]

The aim of this study was to ascertain the mortality from diabetic ketoacidosis over a 66-month period from January 1981 to June 1986 at Whiteabbey Hospital, and to compare this with published figures. Whiteabbey Hospital is a district general hospital with 67 general medical beds staffed by three general physicians, one of whom has an interest in diabetes mellitus. There is no intensive care unit. About 200 patients per month are admitted, the vast majority by direct admission from general practitioners who traditionally have a close relationship with the hospital. There is an accident and emergency unit which is closed from 11.00 pm to 9.00 am weekly and from 5.00 pm to 9.00 am at weekends. A diabetic clinic is held on 1½ sessions per week and approximately 1,500 diabetics attend, 300 of whom are insulin-requiring.

Whiteabbey Hospital, Doagh Road, Newtownabbey BT37 9RH.
D J Eedy, MB, MRCP, Medical Registrar.
A J McNeill, MB, MRCP, Senior House Officer.
W J Andrews, MD, MRCP, Consultant Physician.
Correspondence to Dr Andrews.

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PATIENTS AND METHODS

The hospital records and death certificates of all insulin-requiring and non-insulin-requiring diabetics admitted to Whiteabbey Hospital from January 1981 to June 1986 were examined. There were 156 admissions with poorly controlled diabetes. Patients were considered to be severely ketoacidotic only if ketonuria was noted on ward testing of urine and metabolic acidosis was found on blood gas analysis (standard bicarbonate less than 14 mmol/l). Clinical data on admission, venous plasma glucose, serum electrolyte status, insulin dosage and fluid and electrolyte therapy were gleaned from the medical records. Five patients admitted with hyperosmolar non-ketotic diabetic coma were not included in the study.

RESULTS

Fifteen patients (11 female, four male) were admitted in hyperglycaemic ketoacidosis, two female patients being admitted twice. Only three were previously undiagnosed. Mental state on admission ranged from fully conscious to deeply unconscious. The clinical data (Table) are broadly comparable in age, blood glucose and electrolyte parameters with other studies.[6, 7, 8] None of these patients died within six months of admission. Capillary blood glucose was measured hourly using a reflectance meter (Ames Ltd UK) and venous blood was taken for serum glucose, urea, sodium amd potassium measurement on admission, two, four and eight hours after admission, and then daily. Blood gas analysis for pH, the standard bicarbonate concentration and the calculated and base excess values were obtained on admission and at two, four and eight hours. Three patients on admission had a blood pH of less than 7.0, and in two of these it was below 6.8. Insulin was given by the 'low dose' regimen[9] via the intramuscular route in 14 of the cases. Three patients were given only subcutaneous insulin. Mean insulin dosage over 24 hours was 63 ± 8 units.

TABLE

Clinical data on admission in 17 cases of severe diabetic ketoacidosis

	Mean ± *SEM*
Age	30.1 ± 4.3 years
Serum glucose	29.1 ± 2.3 mmol/l
Blood pH	7.10 ± 0.04
Serum HCO_3	6.4 ± 1.0 mmol/l
Calculated base excess	−21.9 ± 1.7 mmol/l
Serum sodium	132 ± 1.5 mmol/l
Serum potassium	5.1 ± 0.3 mmol/l
Serum urea	8.9 ± 1.1 mmol/l

Intravenous fluids were administered for at least 24 hours via a peripheral vein. No central venous pressure monitoring was performed. A mean of 3.8 litres ± 0.3 litres 0.9% saline and 2.9 ± 0.3 litres 5% dextrose was administered in the first 24 hours. In addition, six patients received a mean of 176 ± 47 mmol of hypertonic (435 mmol/l) sodium bicarbonate. No patients developed signs or symptoms of fluid overload.

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All patients received intravenous potassium chloride supplementation, mean 119 ± 23 mmol, in the first 24 hours.

There was evidence of an underlying infection in six cases: one patient had pneumococcal pneumonia and one patient had two admissions following exacerbation of bronchiectasis. One patient had a myocardial infarction prior to admission. Three patients had omitted their insulin (one on two occasions) and one patient omitted his oral hypoglycaemic agent because of vomiting and inability to retain food: one of these four had supplemented their food intake with a proprietary glucose drink. One patient had omitted both insulin and food in an effort to lose weight. One patient developed ketoacidosis following pancreatitis.

DISCUSSION

Despite the success in our hospital treatment of diabetic ketoacidosis, we do not wish to under-emphasise the real risk of death in this condition,[5, 10] nor do we claim any special ability. Recent literature supports these suppositions,[1, 2, 3, 4] but there are undoubtedly a number of factors which have significantly contributed to our zero mortality rate over a five-year period.

Firstly, the hospital serves a surburban and rural population and has very close contacts with the family doctors of the area. Indeed, a direct admission policy to our medical wards for emergency cases has been operative since before 1981, partly because of this close contact and partly because of the part-time nature of our accident and emergency service. This system is of vital importance in reducing the time lag between diagnosis of diabetic ketoacidosis and initiation of hospital treatment, but effective operation depends on the enthusiasm and ability of the general practitioners who refer the patient.

Secondly, we have applied the basic low dose insulin regimen,[9] given intra muscularly for simplicity (the majority of our junior staff rotate six-monthly). Three patients were given insulin subcutaneously rather than intramuscularly with equally satisfactory clinical and biochemical outcomes. A guide sheet is present in each ward for inspection and reference by junior staff. In addition we have attempted to give intravenous fluids gradually with potassium replacement from the beginning, hoping to achieve a gradual and steady metabolic improvement. Empirically we have given small amounts of hypertonic sodium bicarbonate, (4.2%, 435 mmol/l) intravenously slowly for patients with pH 7.0 or less. Because of the small numbers we would not wish to make any pronouncements on the benefits or otherwise of this policy — one case who received bicarbonate when admitted unconscious with pH 6.78 remained unconscious for a further 48 hours, and at a much later time after recovery was found to have residual granulation at the site of the infusion.

Thirdly, we may have been fortunate in that the more gravely acidotic admissions were young diabetics (mean age 30 ± 4.6 years). The three newly diagnosed diabetics in our group are of interest, and maybe this relatively high proportion of a group of severely ketoacidotic diabetics would suggest that the diagnosis had not been made sufficiently early in the community. We have omitted five cases of hyperosmolar non-ketotic diabetic coma in order not to confuse the paper: one of these died within six months of admission.

The zero mortality for the treatment of diabetic ketoacidotics who reach our wards might suggest that some of our more elderly patients, who are more likely to have a high mortality, may not be reaching hospital at all or may be providing

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the general practitioners with greater diagnostic problems and thus going through the 'Belfast Emergency Bed Service'. In practice, the Emergency Bed Service copes with elderly patients with social problems or uncertain diagnoses, allowing admission to one of the main Belfast teaching hospitals through the 'on-call' take-in unit.

In conclusion we have been fortunate in achieving a zero mortality over five years for the hospital treatment of diabetic ketoacidosis: some peculiarities of our situation as outlined above may have contributed to these figures. Our results may indicate that earlier diagnosis of diabetic ketoacidosis, at least in young people, is taking place in the community. A simple regimen can be successfully followed even by relatively inexperienced staff provided that good nursing and laboratory facilities are available.

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Fatal ischaemic heart disease in Belfast: a comparison of two community surveys

W J McIlwaine, A T Chivers, M D I Donnelly, A E Evans, G MacKenzie

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SUMMARY

Data from two community surveys in Belfast were used to compare all deaths attributed to ischaemic heart disease during two one-year periods (1965/66 and 1981/82). There was an increase in mortality in men of all ages from 3.3 to 4.4 per 1,000 population (33%) and in women from 1.6 to 3.1 per 1,000 population (94%). Only in men aged less than 70 years was the mortality rate unchanged (2.2 per 1,000 population). The proportion of deaths in persons whose fatal attack began outside the hospital was virtually unchanged (65% in 1965/66 compared with 69% in 1981/82). Survival time was markedly decreased in the later survey, as were delay times in initiating medical care. The increase in mortality probably is due to an increase in the incidence of acute myocardial infarction. The introduction of mobile coronary care in Belfast in 1965 seems to have had equal effects in reducing mortality inside and outside hospital.

INTRODUCTION

McNeilly and Pemberton carried out a survey of all deaths due to ischaemic heart disease (IHD) in 1965/66 in Belfast.[1,2] A similar survey conducted in 1981/82 allows some comparisons to be made after an interval of 16 years,[3] during which mobile (pre-hospital) coronary care was established.[4,5] The accuracy of death certification and data relating to the fatal attack and possible medical intervention in relation to the 1981/82 study have been described elsewhere.[6,7] In the present communication, as well as ascertaining accurate community mortality rates, we consider place of death, delay in instituting medical care, survival time and any previous myocardial infarction as factors which may have been influenced by the introduction of mobile coronary care.

Regional Medical Cardiology Centre, Royal Victoria Hospital, Belfast BT12 6BA.
W J McIlwaine, MD, MRCP, Senior Registrar.
Department of Community Medicine, The Queen's University of Belfast.
A T Chivers, BSc, DipNumAnal, AMBCS, Senior Systems Analyst.
A E Evans, MD, MRCP, FFCM, MFCMI, DipSocMed, Senior Lecturer.
G MacKenzie, PhD, FIS, MSc, DIC, FRSS, Senior Lecturer in Statistics applied to Epidemiology.
Department of Community Health, Leicester University.
M D I Donnelly, MB, BCh, BAO, MFCM, DObstRCOG, DipCommMedEdin, Lecturer.
Correspondence to Dr W J McIlwaine, Consultant Physician, Ards Hospital, Newtownards BT23 4AS.

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MATERIALS AND METHODS

Population studied.

There are two coronary care units in Belfast, sited at the Belfast City Hospital and at the Royal Victoria Hospital. A mobile coronary care unit was introduced at the Royal Victoria Hospital on 1 January 1966. Only three deaths in 1965/66 occurred in persons transported by the mobile coronary care unit. The population of Belfast studied in 1965/66 was based on the 1961 Census, and that of the 1981/82 study on the 1981 Census. In 1981/82 the adjacent district of Castlereagh (population 60,757) was included in the study population. This is served by the Ulster Hospital which has a mobile coronary care unit[5] and medical care is similar to that in Belfast.

During the twenty-year interval between the two studies there was a major fall in the population of the city of Belfast, due to demographic factors. The study population in 1965/66 was 415,856 persons and in 1981/82 was 355,980 (14% lower). The population aged less than 70 years was 390,286 persons in 1965/66, and 321,647 persons in 1981/82 (18% lower).

Ascertainment of deaths.

The main source of ascertainment of deaths in both studies was death certification. In 1965/66 only deaths coded under the International Classification of Diseases (ICD), 7th revision, 1955, numbers 420.0 (arteriosclerotic heart disease) and 420.1 (heart disease specified as involving the coronary arteries) were included. A small number of deaths under ICD 420.2 (angina pectoris) were excluded and no deaths classified under other ICD numbers were included. Of the 1,017 deaths provisionally included, eight subsequently were excluded as a post-mortem examination had not confirmed ischaemic heart disease as the cause of death, and another 11 were excluded on clinical grounds which indicated other causes of death. A total of 19 deaths (1.9%) was excluded, leaving 998 for study.

In 1981/82, all deaths coded under ischaemic heart disease (ICD nos 410-414, 9th revision, 1979) were provisionally included. However, all other deaths thought possibly or probably to be due to ischaemic heart disease were provisionally included irrespective of ICD coding. These were identified as in the earlier study by a research worker (R H McN in 1965/66, M D I D in 1981/82) checking through each death certificate at the General Register Office on a weekly basis, usually about three weeks after the death was registered, and selecting those possibly due to IHD. A questionnaire was then filled in for each death. Some 143 deaths not coded under ICD nos 410-414 were included. This group has no equivalent in the earlier survey. There were 108 deaths (9%) coded under ischaemic heart disease which were excluded on clinical grounds by a panel of cardiologists, as there were strong competing causes of death. This is a higher proportion than in 1965/66 and assessment was more rigorous in the later survey.

Information sources and analysis.

The main sources of information — hospital notes, general practitioners, ambulance records, post-mortem records and home interviews with deceased persons' relatives — were similar, but disproportionately more interviews were carried out in 1981/82 (775) than in 1965/66 (approximately 200). The questionnaires were coded and stored on computer and the original coded punch cards from the 1965/66 study were entered into the computer since the original questionnaires for that study contained only coded information.

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RESULTS

Mortality from ischaemic heart disease.

Table I shows deaths from this cause in 1965/66 and 1981/82 by age and sex. There were 33% more deaths in 1981/82 compared with 1965/66. In men aged less than 70 years there were 16% fewer deaths (351 as opposed to 420), but in women there were 10% more (145 as opposed to 132). The proportion of deaths in those aged less than 70 years decreased from 55% (552 out of 998) to 37% (496 out of 1,323).

TABLE I

Deaths from ischaemic heart disease in 1965/66 and 1981/82 by age and sex

Age	Males		Females		Total	
	1965/66	1981/82	1965/66	1981/82	1965/66	1981/82
<70 yrs	420 (65%)	351 (48%)	132 (37%)	145 (25%)	552 (55%)	496 (37%)
⩾70 yrs	224 (35%)	384 (52%)	222 (63%)	443 (75%)	446 (45%)	827 (63%)
TOTAL	644	735	354	588	998	1323

The age/sex specific mortality rates per 1000 population are shown in Table II. The overall mortality in men rose from 3.3 in 1965/66 to 4.4 in 1981/82 (33%). In women the mortality rate rose from 1.6 to 3.1 (94%). For age less than 70 years the mortality rate was unchanged in men (2.2) but increased in women from 0.6 to 0.9 (34%). For age 70 years and above the mortality rate increased in men from 24.4 to 33.4 (37%) and in women from 13.5 to 19.4 (44%). There was a decline in mortality in men aged less than 45 years, no important change from 45–59 years, a small decline from 60–69 years and a marked increase in men above 70 years. In women there was a small decline in those aged less than 45 years, with an increase in all other age groups, especially those above 70 years.

TABLE II

Age/sex specific mortality rates per 1000 population from ischaemic heart disease, in 1965/66 and 1981/82

Age	Males		Females		Total	
	1965/66	1981/82	1965/66	1981/82	1965/66	1981/82
<35	0.1	0	0.1	0	0.1	0
35–39	0.6	0.4	0	0	0.3	0.2
40–44	1.8	0.7	0.5	0.2	1.1	0.4
45–49	2.7	2.6	0.3	0.4	1.4	1.4
50–54	4.2	4.3	0.7	1.3	2.3	2.7
55–59	6.6	6.9	1.6	2.2	3.9	4.3
60–64	11.3	10.7	2.7	3.5	6.4	6.7
65–69	18.9	14.3	5.8	5.7	11.1	9.4
TOTAL<70	2.2	2.2	0.65	0.87	1.4	1.5
70–74	19.4	26.1	9.3	10.3	13.0	16.3
75–79	27.7	31.8	17.1	18.1	20.9	22.7
80–84	31.8	41.9	16.6	27.0	21.9	31.2
85–89	25.6	71.9	20.0	35.2	21.8	44.1
90–94	40.8	87.6	7.7	58.1	16.7	63.4
95+	0	83.3	20	52.6	15.9	56.8
TOTAL⩾70	24.4	33.4	13.5	19.4	17.4	24.1
TOTAL	3.3	4.4	1.6	3.1	2.4	3.7

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Place of death.

In 1965/66, 59% of all deaths occurred outside hospital. This was very similar to the proportion among the 1,277 deaths occurring inside the study boundary in 1981/82 (57%). Fifty deaths occurred outside the study boundary and would not have been within range of the cardiac services under consideration. The proportion of persons whose onset of the fatal attack occurred inside hospital increased from 7% (37) to 12% (57) in those aged less than 70 years and from 13% (60) to 16% (158) in those aged 70 years and above. The proportion of deaths at all ages outside hospital in cases in which the onset of the attack also occurred outside hospital rose slightly from 65% (589 of 901 deaths) in 1965/66 to 69% (731 of 1,062 deaths) in 1981/82. In those aged less than 70 years the proportions were 66% (342 of 515) and 70% (292 of 416) respectively.

TABLE III

Median survival times and delay times in 1965/66 and 1981/82 by age

< 70 years		⩾ 70 years		All ages		Number of cases	
1965/66	*1981/82*	*1965/66*	*1981/82*	*1965/66*	*1981/82*	*1965/66*	*1981/82*
Survival time — minutes							
135	65	270	121	165	84	815	715
Onset to call for first medical aid — minutes							
60	10	60	8	60	8	581	457
Onset to call for ordinary ambulance — minutes							
235	7	342	21	278	11	234	252
Onset to call for mobile coronary care unit — minutes							
—	15	—	15	—	15	—	170
Onset to ward care or to mobile coronary care in 1981/82 — minutes							
360	90	642	195	425	138	259	207

Survival time.

In 1981/82 an upper limit of 28 days' survival time was defined,[8] so that 56 persons surviving for more than 28 days in 1965/66 have been excluded from our analysis (Table III). Cases with times known less accurately than ± 5% have been excluded in 1981/82 (145 deaths), along with those considered to have no specific time of onset to their fatal attack (152), and all unwitnessed deaths (265). For 1965/66, estimated survival times are included and only 127 deaths were excluded. Survival time was much shorter in 1981/82: median survival time decreased from 165 minutes in 1965/66 to 84 minutes in 1981/82. The proportion of very sudden deaths (within four minutes) increased from 5% (43 of 815) in 1965/66 to 19% (140 of 715) in 1981/82.

Delay times in initiating medical care.

Median delay times from the onset of the fatal attack to calling for first medical aid, the ordinary ambulance or mobile coronary care unit in 1981/82 and to arrival in the hospital ward (or arrival of MCCU to a live patient) are compared (Table III). In 1965/66 there were 127 persons with unknown survival times and 56 persons with survival times > 28 days who were excluded, and in 1981/82 data known less accurately than ± 5% were excluded. The delay times are much reduced in 1981/82, so that, in persons aged less than 70 years, 29% (44 of 153 persons) came under care in 1965/66 after 12 hours compared with only 12% (13 of 105 persons) in 1981/82. In those aged more than 70 years, these proportions were 48% (51 of 106) and only 18% (18 of 102) respectively.

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TABLE IV

Number of previous myocardial infarctions, number of persons and ratio of number of previous myocardial infarctions to the number of persons dying from ischaemic heart disease, in Belfast in 1965/66 and in 1981/82, in persons aged less than 70 years

	1965/66			1981/82		
Age group	*Previous MI*	*Persons*	*Ratio*	*Previous MI*	*Persons*	*Ratio*
<35 years	0	3	0	0	0	0
35–39	3	7	0.43	2	4	0.5
40–44	5	27	0.19	6	8	0.75
45–49	20	37	0.54	8	28	0.29
50–54	34	61	0.56	26	57	0.46
55–59	60	93	0.65	46	95	0.48
60–64	69	138	0.5	57	13	0.44
65–69	84	181	0.46	74	171	0.43
Total	218	416	0.5	219	494	0.44

Previous myocardial infarction.

In 1965/66 and 1981/82 the number of previous episodes of myocardial infarction was recorded for each person. Confirmation by electrocardiograph was available in 94% of these episodes in 1981/82; in 1965/66 the proportion was probably less. The ratio of previous infarctions per person by five-year age groups up to 70 years of age is shown in Table IV. This ratio is higher in 1965/66 in all age groups except those aged less than 45 years.

DISCUSSION

The 16-year period from 1965/66 to 1981/82 has seen significant changes in the epidemiology of fatal ischaemic heart disease in Belfast. The differences in methodology in the two survey periods probably does not affect the overall conclusions. The mean age at death increased by four years in both sexes. The periodic ICD revisions exert a small bias due to the re-allocation of certain causes of death. In men aged less than 70 years the mortality rate was unchanged, but mortality increased in older men by 37%. In women, mortality increased by 34% in those aged less than 70 years and 44% in those aged 70 years or over. As delay times from the onset of the fatal attack to providing medical care were substantially shortened, the increase in the community mortality rates is most likely due to a large rise in the incidence of acute myocardial infarction rather than to changes in the survival rates. The effects of mobile coronary care can only be studied indirectly. We may postulate that such care might prolong survival time while the survival rate after the acute attack remains unchanged, or it might solely reduce mortality outside hospital. However, Adgey et al (1971) showed a reduction of in-hospital mortality from acute myocardial infarction in patients seen early (within three hours of onset of symptoms).[9] Thompson et al (1979) also showed a reduction of in-hospital mortality with cardiopulmonary resuscitation performed outside hospital.[10] The shortening of survival time, together with the unchanged proportion of deaths occurring outside hospital may be due to equal reductions in fatality rates of acute myocardial infarction occurring inside

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or outside hospital. The data do not support the view that more persons are surviving a first episode of acute myocardial infarction, but the criteria used to establish previous episodes in the 1965/66 study could not be adequately assessed.

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Accuracy of blood glucose concentrations determined by four visually read reagent strips

M P MacManus, R D G Neely, P M Bell

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SUMMARY

Two new reagent strips have recently been introduced for blood glucose measurement by direct visual reading. Results obtained with these strips (Glucostix and Hypogard GA) were compared with those obtained using other commonly employed strips (BM-Test-Glycemie 1–44 and Visidex II) and a standard laboratory method. Blood glucose estimations were performed on samples of venous blood drawn from 125 patients attending the diabetic clinic using each of the four strips and the laboratory method. Results obtained with the strips correlated with the laboratory values as follows: BM-Test-Glycemie 1–44, r = 0.93; Glucostix r = 0.93; Hypogard GA r = 0.87 and Visidex II r = 0.92. The lower correlation with Hypogard GA reflected consistent underestimation of the laboratory value (slope of regression line = 0.63). Readings in error by 20% or more were: BM-Test-Glycemie 1–44, 14%; Glucostix, 15%; Hypogard GA, 31%, and Visidex II, 14%. With Hypogard GA strips, 57% of readings above 16 mmol/l were inaccurate. We conclude that Hypogard GA strips cannot be recommended for direct visual reading. Acceptable results may, however, be obtained using the other three strips.

Reagent strips allow reasonably accurate determinations of blood glucose concentrations when used with a reflectance meter.[1,2] Nevertheless many diabetic patients prefer to read the reagent strips visually. This method avoids the problems associated with meter calibration, is cheaper and also is more portable. Direct visual readings with BM-Test-Glycemie 1–44 (Boehringer Corporation) and Visidex II (Ames) have been shown to be acceptable in the hands of medical and technical personnel.[3,4] Recently two new reagent strips have been marketed, Hypogard GA (Hypogard UK Ltd) and Glucostix (Ames), and it is claimed that they are also suitable for direct visual reading. To test the validity of these claims, we have compared results obtained using the newer strips with readings from BM-Test-Glycemie 1–44 and Visidex II and with a standard laboratory method.

PATIENTS AND METHODS

Blood was removed by venepuncture from 125 patients routinely attending the diabetic clinic. A portion of each sample was sent to the laboratory for glucose determination by a glucose oxidase method (Glucoroder-E, Analytical Instruments Company). External quality control of this method takes place as

Royal Victoria Hospital, Belfast BT12 6BA, Northern Ireland.
M P MacManus, MB, MRCP, Senior House Officer in Internal Medicine.
R D G Neely, MB, MRCP, Registrar in Chemical Pathology.
P M Bell, MD, MRCP, Consultant Physician.
Correspondence to Dr P M Bell.

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part of the UK Prospective Diabetes Study. Over the period of the study mean bias of readings against external standards was 0.4% with a between-batch coefficient of variation of 2.2%.

A drop of blood from each sample was also placed, directly from the syringe, on to one of each of the four reagent strips. Strips were prepared according to the manufacturer's instructions. Blood was removed from two of the strips (Hypogard GA and BM-Test-Glycemie 1–44) by a wiping technique after 30 and 60 seconds respectively. Blood was removed from both of the other strips (Visidex II and Glucostix) after 30 seconds by a blotting technique.

Two doctors attended each session, one preparing and reading the 'wiped' strips and the other preparing and reading the 'blotted' strips. Blood was applied to each of the strips in random order and the strips were read in random order. The person who had read the 'blotted' strips at one session would read the 'wiped' strips at the next. Readings were acquired at five different sessions. At one session Visidex II strips were not used due to temporary unavailability at the clinic.

We attempted to estimate blood glucose values which lay between colour blocks to the nearest 0.5 mmol/l for values below 20 mmol/l. This gave the observers flexibility in their interpolations, although we recognised that visual reading of strips is not designed to give such precise readings particularly at higher glucose concentrations. Estimation to the nearest 0.5 mmol/l also helped us to overcome any difficulty caused by the different intervals between colour blocks used for visual reading of the various strips.

RESULTS

Blood glucose values obtained using reagent strips are plotted against corresponding laboratory values in the Figure. Readings with each of the strips

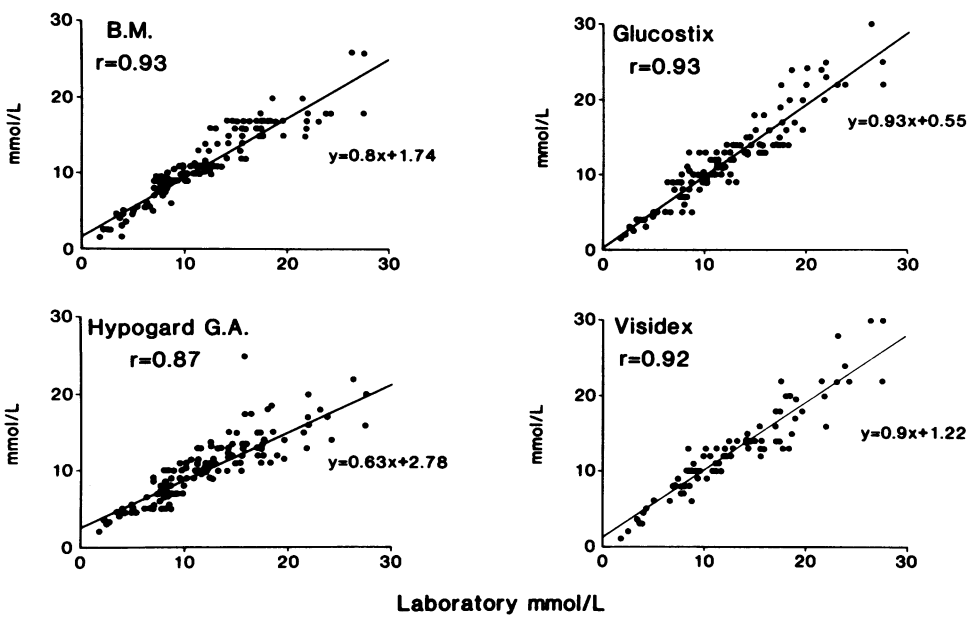

Figure. Blood glucose values obtained with reagent strips ('y' axes) plotted against corresponding laboratory values ('x' axes). Regression lines are also drawn.

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correlated reasonably closely with laboratory values (BM-Test-Glycemie 1–44 r = 0.93, Glucostix r = 0.93, Hypogard GA r = 0.87, Visidex II r = 0.92). With Hypogard GA strips the correlation coefficient was lower than with the other strips. In addition, readings with Hypogard GA strips were consistently lower than the corresponding laboratory values (regression line slope = 0.63).

We examined separately results in the low, middle and high ranges. In order to illustrate how consistently the strips performed over our different ranges, (arbitrarily chosen as < 8, 8–16 and > 16 mmol/l) readings which were inaccurate by 20% or more are shown in the Table. BM-Test-Glycemie 1–44 strips gave few incorrect results for values less than 16 mmol/l but were unreliable above this level. Glucostix and Visidex II were less reliable than BM-Test-Glycemie 1–44 in the low and middle ranges but were relatively accurate in the high range. With Hypogard GA strips, inaccurate results were relatively frequent in the low and middle ranges but very frequent (more than 50%) in the high range. Most discrepancies with BM-Test-Glycemie 1–44 and Hypogard GA in the high range were underestimates of the laboratory value, whereas with the other strips there were both over- and underestimates.

TABLE

Results in error by > 20%

	Blood glucose (mmol/l)							
Range	*< 8*		*8–16*		*> 16*		*Overall*	
BM Glycemie 1–44	4	(15)	5	(7)	8	(29)	17	(14)
Glucostix	6	(22)	10	(14)	3	(11)	19	(15)
Hypogard GA	7	(26)	16	(23)	16	(57)	39	(31)
Visidex II	4	(19)	6	(10)	5	(19)	15	(14)

Results are expressed as numbers in error by 20% in each blood glucose range and in parentheses as percentage results in error.

Only 106 results were available for Visidex II.

DISCUSSION

All of the reagent strips were simple to use and could provide a result within two minutes. Both observers felt that the 'wipe' technique (Hypogard and BM-Test-Glycemie 1–44) was easier and less messy.

The tendency for Hypogard GA strips to give inappropriately low readings does give rise to concern: in several cases errors in management might have resulted if treatment had been based on the reagent strip results alone. The manufacturers have suggested that the underestimation may be caused by excessive pressure, applied during wiping blood off the strips, leading to removal of reagent from the surface. If this is the case, it is likely that patients will make a similar mistake and we would suggest that these strips are not the best choice for visual reading.

There was little to choose between the other three strips. Over the 0–16 mmol/l range BM-Test-Glycemie 1–44 strips were perhaps superior, whereas above 16 mmol/l significant mistakes were frequent. Visidex II and Glucostix were a little less reliable in the lower ranges but proved quite accurate in the higher range.

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It should not be forgotten that these results were obtained under relatively ideal conditions. When testing their own blood glucose in everyday life, patients may be in a hurry or have difficulty obtaining an adequate sample of blood. Others may have visual impairment. In practice therefore a greater number of inaccurate readings can be expected.[5]

We thank Ames Division (Miles Laboratories Ltd), the Boehringer Corporation and Hypogard GA (UK) Ltd for supplying the reagent strips used in this study. We are grateful to the nursing staff of the Metabolic Outpatient Clinic who obtained blood samples from the patients, and to Mrs Marie Loughran for typing the manuscript.

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Thyroid screening in elderly hospital patients

E Byrne, D H Gilmore, T R O Beringer

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SUMMARY

A screening programme of thyroid disease in 214 elderly patients attending hospital was undertaken incorporating clinical and biochemical assessment. The prevalence of untreated hypothyroidism was 2.8%, treated hypothyroidism 2.8%, untreated hyperthyroidism 0.9%, sub-clinical hypothyroidism 4.7%, and non-thyroidal illness 3.3%. One patient with hypopituitarism was identified. Clinical judgement alone was poor. The costs of such a screening programme are discussed and the benefits highlighted.

INTRODUCTION

Thyroid disease in the elderly often presents atypically, and the traditional clinical criteria for diagnosis of both hypothyroidism and hyperthyroidism may be masked and therefore unhelpful.[1] As effective treatment is available, routine screening tests in the elderly of thyroid function have been advocated.[2,3] However, considerable debate continues as to the benefit of screening the healthy elderly living in the community.[4–7]

The aim of this study was to determine the prevalence of undiagnosed thyroid disease in all elderly patients presenting to the Geriatric Medical Unit at the Royal Victoria and Throne Hospitals, Belfast, and to assess the benefits of such a routine screening programme.

METHOD

New patients aged 65 years and over, referred to the Geriatric Unit between August 1986 and January 1987 for admission, outpatient and day hospital attendance, were assessed and screened for the presence of thyroid disease.

A standard proforma was completed for each patient at the first attendance, and included assessment of weight loss, energy, temperature preference, hair and skin changes, current medication and previous thyroid treatment. Clinical examination was carried out with particular reference to the presence or absence of signs of thyroid disease, including goitre, lid lag and retraction and delay of the tendon reflexes. Judgement was then made regarding the overall clinical impression of thyroid status and recorded as either hypothyroid, euthyroid or hyperthyroid.

Geriatric Medical Unit, Royal Victoria and Throne Hospitals, Belfast.
Eileen Byrne, MB, MRCP, Senior Registrar.
D H Gilmore, MB, MRCP, Consultant Physician.
T R O Beringer, MD, MRCP, MRCPI, Consultant Physician.
Correspondence to Dr T R O Beringer, Geriatric Medical Unit, Royal Victoria Hospital, Grosvenor Road, Belfast BT12 6BA.

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Blood was sampled for serum total thyroxine (total T_4) and thyroid-stimulating hormone (TSH) estimation. Serum total T_4 was measured by a solid-phase radioimmunoassay[8] and serum TSH measured by radioimmunoassay.[9] If initial results were abnormal, a second sample was sent for repeat total T_4 and TSH estimation. In addition, measurement of thyroid antibody titres, free T_4 and response to thyrotropin-releasing hormone (TRH) were performed as clinically indicated. The criteria used for a diagnosis of hypothyroidism were total T_4 repeatedly less than 50 nmol/l with an accompanying elevation of TSH above 5 mU/l. Hyperthyroidism was diagnosed if the total T_4 was greater than 150 nmol/l and accompanied by a diminished TSH response to an intravenous challenge with 200 μg of TRH.

RESULTS

Two hundred and fourteen subjects (160 females, 53 males) were studied, with a mean age of 80.6 years, mean total T_4 of 82.2 nmol/l and mean TSH of 3.1 mU/l. Normal thyroid function tests were found in 182 patients (85%), of whom 48 were male (mean age 76.2 years) with a mean total T_4 of 82.9 nmol/l and a mean TSH of 1.6 mU/l, and 134 female (mean age 82.1 years) with a mean total T_4 of 86.3 nmol/l and a mean TSH of 1.8 mU/l. Initial clinical assessment alone incorrectly judged three of this group as hypothyroid and five as hyperthyroid. Abnormal thyroid function tests were present in 32 patients (15%) (Table I).

TABLE I

Categories of patients with abnormal thyroid function results

	Number	*Mean age (years)*	*Male*	*Female*	*% of total*
Untreated hypothyroid	6	79	—	6	2.8%
Treated hypothyroid	6	88	1	5	2.8%
New hyperthyroid	2	72	1	1	0.9%
Normal total T_4 with raised TSH	10	79.5	1	9	4.7%
Low total T_4 with normal TSH	8	83.3	2	6	3.8%

Untreated hypothyroidism

Six new cases of hypothyroidism were identified (Table II) with a mean total T_4 of 32.9 nmol/l and a mean TSH of 32.8 mU/l. Initial clinical assessment of this group correctly judged one patient as hypothyroid and the remainder were assessed incorrectly as euthyroid. One patient died from cardiac failure, while the remainder improved considerably after treatment with resolution of many non-specific symptoms, accompanied by increased mobility, enabling a return to continuing independence at home.

Treated hypothyroidism

Six patients (5F, 1M) were identified with a previous history of hypothyroidism and thyroxine replacement therapy, with a mean total T_4 of 90.5 nmol/l and a mean TSH of 8.0 mU/l. Two of this group were receiving an inadequate dose of thyroxine and were correctly judged to be clinically hypothyroid.

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Hyperthyroidism

Two patients were found to have elevated total and free T_4 levels, flat TSH response to TRH; they were judged to be hyperthyroid and treated with radioactive iodine and carbimazole respectively. Neither case was identified on initial clinical assessment alone.

Normal total T_4 with raised TSH

Ten patients had normal levels of total T_4 with elevated TSH levels. On repeat measurement, the level of TSH returned to normal in three patients, while seven remained abnormal (Table III). Two of this group had been incorrectly judged clinically to have hyperthyroidism.

TABLE II

Untreated hypothyroid patients identified by the screening programme

	Age	Sex	Total T_4 nmol/l	TSH mU/l	Thyroid antibodies	Presenting illness
1.	90	F	46	10	—	Osteoarthritis
2.	75	F	34	20	—	Chest infection
3.	86	F	22	60	—	Cardiac failure
4.	82	F	35	26	—	Lack of energy
5.	75	F	6.3	25	—	Arthritis
6.	66	F	48	57	+	Myelofibrosis

TABLE III

Sub-clinical hypothyroidism: normal total T_4 with TSH greater than 5.0 mU/l on initial screening test

Age/ Sex		Total T_4 nmol/l (initial test)	TSH mU/l	Total T_4 nmol/l (second test)	TSH mU/l	Thyroid antibodies	Presenting illness
79	F	63	6.7	60	5.9	—	Epilepsy
80	F	59	5.4	72	5.4	—	Cardiac failure
78	F	84	5.9	60	5.7	—	Osteoarthritis, diabetes mellitus
71	F	83	5.3	70	3.0*	—	Gout
83	F	60	8.2	54	4.2*	—	Cerebrovascular disease
78	F	63	6.1	70	6.6	—	Partial thyroidectomy for thyrotoxicosis
78	F	61	26.5	62	16.2	+	Cerebrovascular disease
78	F	96	5.8	92	2.9*	—	Cerebrovascular disease
95	F	80	5.7				Terminal bronchopneumonia
75	F	68	8.9	59	7.0	+	Pernicious anaemia

*TSH returned to normal on repeat estimation.

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Low total T_4 with normal TSH (non-thyroidal illness)

Eight patients (six female and two male; mean age 83.3 years) with lowered levels of total T_4 and normal TSH levels were identified. The mean total T_4 was 37 nmol/l and TSH 2.1 mU/l. A diagnosis of hypopituitarism was confirmed in one subject. Five patients were seriously ill and subsequently died, and two were recovering after operative fixation of femoral neck fracture. Three of this group were suspected on clinical grounds to have hypothyroidism.

DISCUSSION

Untreated hypothyroidism was detected in 2.8% of the elderly hospital patients, a figure comparable to that of 2.3% recorded in hospital inpatients,[1] and higher than the prevalence rate of 0.94% reported from the elderly in the community.[5] The overall prevalence rate of untreated and treated hypothyroidism combined was 5.6%. Hyperthyroidism was less common with a prevalence rate of 0.9% in comparison with 0.47% reported from the elderly in the community.[5] The diagnosis of hypothyroidism on clinical grounds alone was poor, with detection of only one of the six new hypothyroid patients. That clinical diagnosis alone is difficult is further supported by the finding that the majority of this group had attended other hospital departments in the preceding months without detection of the hypothyroidism. This highlights the non-specific clinical presentation of hypothyroidism in the elderly [1] and is an important factor in support of an elective screening programme of the elderly attending hospital rather than reliance on clinical diagnosis alone.

One area of concern is that routine screening will reveal the presence of equivocal results, with resultant difficulty of interpretation and commitment to long-term follow-up. Seven patients had repeatedly normal total T_4 levels with moderate elevation of TSH but without clinical evidence of hypothyroidism. This group may be deemed to have sub-clinical hypothyroidism [10] and will require regular monitoring of thyroid function to detect progression to overt hypothyroidism at an early stage. If thyroid antibodies are also present, then the risk in females of progression to overt hypothyroidism is particularly increased and estimated at 5% per year.[11] A second group of eight subjects were identified with low total T_4 levels and normal TSH level. A diagnosis of hypopituitarism was confirmed in one patient and appropriate cortisone and thyroxine replacement therapy commenced, with resolution of confusion and immobility. Five of this second group were seriously ill and subsequently died. Two were recovering from major surgery, confirming a relationship with severe illness previously attributed to lowered protein and thyroxine binding globulin levels and inhibitors of binding.[12] Thus, the majority of elderly patients will fall into one of five distinct categories whose management is straightforward.

Screening of each elderly hospital patient will have an add-on cost to the laboratories of approximately £3, with the cost of total T_4 measurement approximately £1 and TSH £2 per sample. Costs may be reduced by screening using only TSH [13] or total T_4 estimation, but neither alone will accurately identify the 3% of the patients with sub-clinical hypothyroidism, a proportion of whom may benefit from thyroxine therapy.[14] The total cost of screening 214 patients was approximately £650, resulting in expenditure of around £100 to detect each case of previously undiagnosed hypothyroidism. Treatment costs with thyroxine replacement are small and we would deem the overall financial cost of such thyroid function screening to be cost-effective.

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There are considerable variations in the practice of geriatric medicine and selective procedures for admission throughout the UK, and thus reliance on prevalence figures from other reported studies may be misleading. This study has enabled us accurately to establish the prevalence of thyroid disease in the population of sick elderly patients presenting to this geriatric medical unit. It is our belief that the yield of both hypothyroidism and hyperthyroidism previously undetected is an important and worthwhile task and fully justifies screening of the sick elderly. Appropriate treatment has afforded considerable clinical benefit, symptomatic relief and improvement of the elderly patients' quality of life. Reliance on clinical diagnosis alone of thyroid disease in the elderly is inadequate, with many patients remaining undetected and denied effective treatment. Elective screening for thyroid disease in the elderly hospital patient is therefore an important, cost-effective, worthwhile and rewarding task.

We gratefully acknowledge the assistance of Mr Brian Sheridan with the laboratory estimations of T_4 and TSH, and the junior medical staff of the Geriatric Medical Unit who performed a large number of assessments.

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Case report

Congenital cutaneous candidiasis

T R J Tubman, M McC Reid

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Newborn babies commonly develop mucocutaneous candidiasis, which is usually acquired during the process of birth. Congenital cutaneous candidiasis presenting at birth is very uncommon and is due to intrauterine infection by the organism.

CASE HISTORY

A male infant was born by normal delivery at 39 weeks' gestation to a 29-year-old patient whose pregnancy had been complicated by a persistently heavy monilial vaginal discharge. Maternal history suggested that the membranes had ruptured two weeks prior to delivery, but there was no evidence of systemic maternal infection.

In the first hour of life the infant became tachypnoeic and was admitted to the special care nursery for investigation. On examination his weight of 3640g was appropriate for a gestational age of 39 weeks. He was tachypnoeic with an over-inflated chest. There was a widespread erythematous rash over the neck, trunk and limbs. White cell count was 20.3 $\times$ 10^9/l, 85% neutrophils; platelets 238,000/mm^3. Serum IgM was elevated at 0.44 g/l suggesting intrauterine infection (normal range $<$.02 g/l). Chest X-ray showed increased perihilar markings with mild patchy opacity in the right lung field. The clinical diagnosis was transient tachypnoea of the newborn but, as congenital pneumonia could not be excluded, he was commenced on intravenous benzylpenicillin and gentamicin. Over the next 24 hours his respiratory difficulty diminished. The rash became more macular, and changed over the next two days into papulovesicular lesions distributed over the trunk, face, palms of the hands and soles of the feet. There were no buccal lesions. At this stage, cultures of superficial skin swabs from ear, nares, umbilicus and erythematous skin and of gastric aspirate taken at birth grew *Candida albicans,* as did a bag specimen of urine. No bacteria were isolated. Blood cultures remained sterile.

On the second day he was commenced on oral nystatin suspension and nystatin ointment to the skin, and intravenous antibiotics were stopped. After the third day the infant remained active and was able to bottle-feed without supplemental oxygen. The rash was rather more slow to settle, beginning to desquamate on the fifth day and finally clearing with residual desquamation of the palmar skin around 14 – 21 days.

Neonatal Unit, Belfast City Hospital, Belfast, Northern Ireland, BT9 7AB.
T R J Tubman, BSc, MB, MRCP, Paediatric Registrar.
M McC Reid, MB, FRCP, Consultant Paediatrician.
Correspondence to Dr T R J Tubman, Royal Maternity Hospital, Belfast, Northern Ireland, BT12 6BB.

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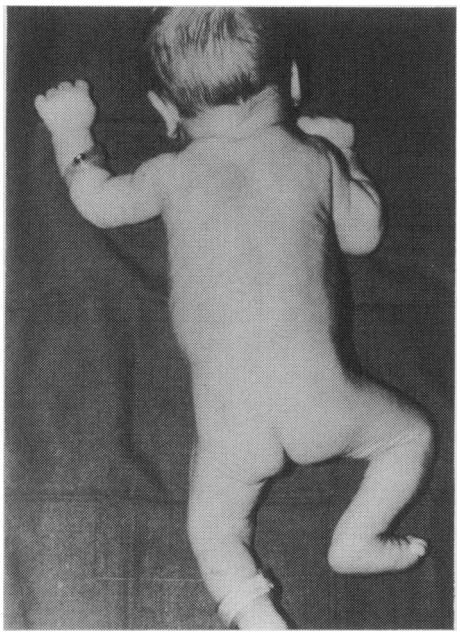

Fig 1. Congenital cutaneous candidiasis — diffuse distribution of rash. Age three days.

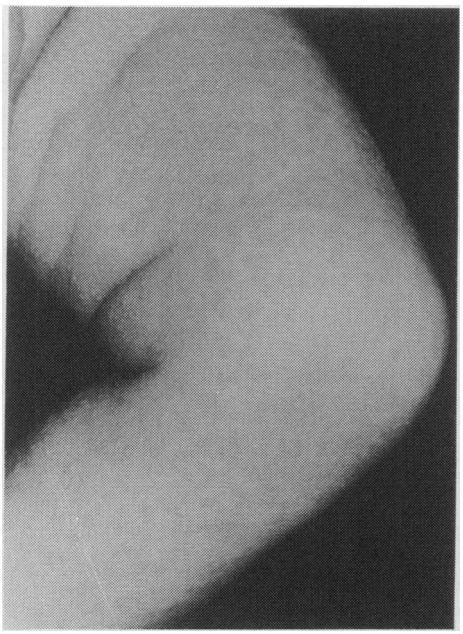

Fig 2. Left knee showing vesicular phase of the rash. Age three days.

DISCUSSION

Up to 25% of pregnant women harbour *Candida albicans* in their vaginal flora and not surprisingly mucocutaneous candidiasis is common in their babies. By contrast, congenital cutaneous candidiasis is very uncommon with only about 70 cases reported in the literature over the past 40 years. There are some important differences between these two varieties of neonatal candidiasis. Babies born to mothers with vaginal thrush acquire infection during descent through the birth canal but commonly do not show signs of infection until the end of the first week of life. This generally takes the form of oral or napkin candidiasis and usually requires only local therapy. In very occasional circumstances it may act as the focus for a more serious disseminated infection in sick premature infants.[1]

Congenital cutaneous candidiasis is always present either at or within 12 hours of birth, and results from previous chorioamnionitis. In many cases, ascending infection follows prolonged rupture of membranes, though the organism may also cross intact membranes. As opposed to 'late-onset' candidiasis, where the organism takes some time to gain a hold, and then only on moist sites, the congenital variety flourishes rapidly in the warm wet intrauterine environment, and affects all parts of the skin.[2] There is often accompanying evidence of white plaques on the cord or membranes if they are examined closely.[3]

The rash appears in a characteristic manner.[2, 3, 4, 5] At first it presents as an erythematous angry-looking macular rash over trunk and limbs. This progresses to form papules which vesiculate within days, being found all over the body including palms and soles. With adequate treatment the rash resolves by desquamation in about one week.

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Treatment involves oral and topical nystatin until the rash has cleared. Systemic antifungal therapy is warranted only in sick premature babies who have positive evidence of disseminated sepsis.

Despite its rather serious appearance, this condition is benign in full-term babies, with infection limited to the skin. Earlier reports of urinary involvement probably represent contamination of bag samples from the skin. Premature infants of less than 1500g with respiratory distress for not fare so well, and virtually all die rapidly from candidal pneumonia and overwhelming generalised fungaemia.[4] There is a risk of sick premature infants becoming colonised by candida from the hands of attendants working with infants who have congenital cutaneous candidiasis. They may then develop systemic candidiasis.

This infant presented with all the features of congenital cutaneous candidiasis as found in full-term babies, and his illness pursued a benign course as would be expected. His infection remained limited to the skin, the respiratory distress probably being due to transient tachypnoea of the newborn. Candida isolated from urine was undoubtedly a contaminant from heavily infected skin. He remained well at follow-up and has now been discharged from review.

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CORRECTION:

Ulster Medical Journal. Vol 56. Supplement. August 1987. p. S44.

Under heading JAMES DIXON BOYD.

First paragraph. Line 4. Delete Farm. — Delete Down and insert Antrim.

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Case report

Adult coeliac disease presenting with infertility

J P McCann, D P Nicholls, J A Verzin

Accepted 26 January 1988.

Coeliac disease in adults usually presents with gastrointestinal symptoms,[1] but has rarely been recognised as a cause of infertility.[2, 3] We report a case in which the institution of a gluten-free diet was followed by successful pregnancy.

CASE HISTORY

A 21-year-old female presented to the Mater Infirmorum Hospital in 1983. She had been married for two years and had been attempting to conceive for one year. She had taken a combined oral contraceptive for nine months previously. No abnormal clinical history was obtained, and in particular there was no history of menstrual irregularity. On examination, she showed normal secondary sexual development and there were no physical signs of systemic disease. Her sex hormone profile was normal — serum FSH was 4.4 U/l (normal 0.6 – 7.5), serum LH 11.5 U/l (0.8 – 16), oestradiol (luteal phase) 450 pmol/l (165 – 700), progesterone (luteal phase) 17.7 nmol/l (6 – 80). Serum testosterone was 0.85 nmol/l (0.5 – 2.8) and serum prolactin 310 mU/l (< 360).

Laparoscopy and dilatation and curettage were performed. The Fallopian tubes were patent and no ovarian abnormalities were noted. Endometrium obtained was in the secretory phase of the menstrual cycle. At this time she had a microcytic hypochromic anaemia (Hb 8.4 g/dl) which was treated by transfusion without further investigation. Following this, despite restoration of haemoglobin levels to normal, and also therapeutic trial of clomiphene and cyclofenil, conception failed to occur.

In November 1985 she presented to the Royal Victoria Hospital with a profound recurrent microcytic hypochromic anaemia (Hb 6.5 g/dl). She gave no history of overt blood loss. Her diet was satisfactory and she denied having steatorrhoea. Apart from pallor, no abnormality was found on clinical examination. Her height was 156cm and weight 48.1kg (93% of predicted). Blood films showed a dimorphic red cell pattern. Serum iron was reduced to 4.7 µmol/l (normal 15 – 30), with an iron binding capacity of 96.7 µmol/l (normal 45 – 72) and a serum ferritin of 15 µg/l (normal 40 – 70). Faecal occult bloods were negative.

Royal Victoria Hospital, Belfast, BT12 6BA.
J P McCann, MD, MRCP, Senior Registrar in Medicine.
D P Nicholls, MD, MRCP, Consultant Physician.
Mater Infirmorum Hospital, Belfast.
J A Verzin, MD, FRCOG, FICS, Consultant Gynaecologist.
Correspondence to Dr Nicholls.

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Serum vitamin B_{12} and folate levels were normal. Serum carotene was 0.4 μmol/l (normal 1.1 – 3.7) and she excreted only 9% of a 25 g oral D-Xylose load. There was a flat response to a lactose tolerance test. Her fasting breath hydrogen concentration was elevated at 76 ppm (normal less than 20) and no further elevation occurred in response to 25 g lactose. Small bowel barium studies showed dilatation of the jejunal mucosal fold pattern with flocculation, in keeping with a malabsorption state. Jejunal biopsy revealed the presence of subtotal villous atrophy and anti-gliadin antibodies were positive (titre 1:20). Family screening for the presence of anaemia or antigliadin antibodies was negative.

A diagnosis of coeliac disease was made and she was commenced on a gluten-free diet, haemoglobin levels being restored by transfusion. This led to a rapid improvement in her well-being, and within two months she had conceived. She was delivered of a healthy girl in September 1986. Her present haemoglobin is 13.8 g/dl on no supplements.

DISCUSSION

Adult coeliac disease usually presents with gastrointestinal symptoms.[1] In our patient, the lack of such symptoms obscured the diagnosis, which was suspected only after recurrent iron deficiency anaemia without excessive blood loss had been observed. Although infertility is a known complication of coeliac disease,[4] it is rarely a presenting feature.[2, 3] Furthermore, the presence of infertility does not correlate with the severity of the coeliac disease in either males or females.[5]

Conception within two months of commencing on a gluten-free diet, after three years of infertility, strongly suggests a relationship between the two. The infertility is not likely to have been due to anaemia alone as correction of this in the past had not been followed by conception, and likewise her body weight was normal. Similar restoration of fertility has been previously reported in both female[2] and male[6] patients. In male coeliac patients, abnormal sperm motility and morphology have been described[7] which may account for impaired fertility. In female patients the reason for infertility remains unknown, although it has been suggested that it may be due to zinc deficiency.[3] The zinc status of our patient was not determined.

We would suggest that coeliac disease be considered as a cause for unexplained infertility. Although the association is uncommon, treatment is simple and may lead to conception.

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Case report

Congenital absence of the left pericardium

W Dickey, R F McDowell, K R Logan

Accepted 7 July 1987.

Congenital complete absence of the left pericardium is a rare cause of clinical and radiological cardiovascular abnormalities. We describe a patient in whom this diagnosis was made coincidentally when she presented with another problem.

CASE HISTORY

A 20-year-old female sought advice for diarrhoea and vomiting which followed a holiday in Majorca. She had no other symptoms. Apart from investigation of a cardiac murmur when she was six, past medical history was unremarkable. On examination she appeared healthy, with no fever or dehydration. Her abdomen was normal. Pulse was 100/min, regular, and blood pressure 110/60. Her apex beat was not displaced and of normal character. On auscultation there was wide but variable splitting of the second sound with a loud pulmonary component, and a grade 3/6 mid-systolic murmur heard in the pulmonary area only. Her first heart sound was normal and no other murmurs or bruits were heard.

Electrocardiograph (Fig 1) showed right axis deviation and clockwise rotation. Chest X-ray (Fig 2) showed an abnormal cardiac outline, consistent with leftward rotation. The trachea was central, and lung fields and pulmonary vascularity

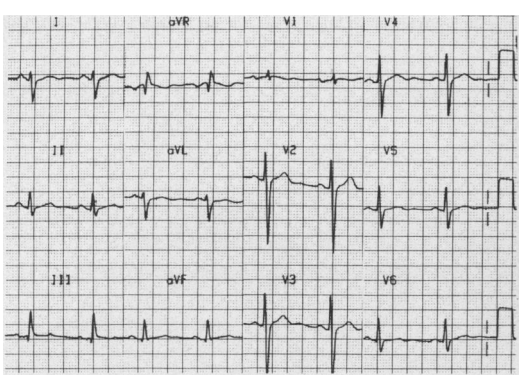

Fig 1. Electrocardiograph showing right axis deviation and clockwise rotation of the heart.

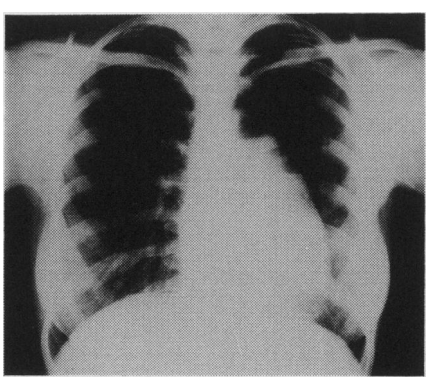

Fig 2. Chest X-ray consistent with leftward rotation of the heart.

Lagan Valley Hospital, Hillsborough Road, Lisburn, Co Antrim, BT28 1JP.
W Dickey, BSc, MRCP, Senior House Officer.
R F McDowell, MD, FFRRCS(Irel), DMRD, Consultant Radiologist.
K R Logan, MB, MRCP, Consultant Physician.
Correspondence to Dr Logan.

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were normal. There were no abnormalities on echocardiography or 48-hour ambulatory ECG monitoring.

Following referral at the age of six with a systolic murmur heard at the left sternal edge and similar chest X-ray appearance, right and left cardiac catheterisation had been performed. Pressures, oxygen saturations and ventricular contraction had all been normal: in particular there had been no evidence of pulmonary hypertension, pulmonary valve disease, or shunts. No diagnosis had been made.

It was concluded after literature review that her signs and ECG and chest X-ray appearances were consistent with complete absence of the left pericardium. Investigation of her presenting symptoms was negative and they settled without treatment. Six months after presentation she remains well.

DISCUSSION

Congenital pericardial defects are rare. The total number of reported cases is less than 200,[1] and one case was found in a series of 14,000 autopsies.[2] The clinical findings have been described by Nasser.[3]

Complete absence of the left pericardium is thought to result from premature atrophy of the left duct of Cuvier, causing impaired circulation to the pleuro-pericardial membrane from which it is derived.

ECG changes include right axis deviation, incomplete right bundle branch block, clockwise rotation and tall peaked P waves in the right chest leads.[4] Radiological appearances are characteristic. The heart is shifted to the left, causing apparent cardiomegaly, in the presence of a midline trachea. Prominence of the main pulmonary artery, and interposition of lung between diaphragm and heart and between aorta and pulmonary artery on anterior oblique views may also be seen. Echocardiography is often normal, but may show right ventricular dilatation and paradoxical movement of the interventricular septum.[5] Echocardiography is, however, essential to exclude other cardiac abnormalities which co-exist in one third of reported cases.[6]

The demonstration of a pneumopericardium, after the artificial induction of pneumothorax, had been considered the procedure of choice in the diagnosis of complete left pericardial absence. However, the X-ray appearances are sufficiently characteristic for this potentially hazardous procedure not to be necessary,[3] particularly since most cases are asymptomatic.

Rarely, chest pain and syncope may occur, attributed to lack of cardiac support and subsequent torsion and strain of the great vessels. Abnormal cardiac mobility is also thought to cause the characteristic systolic murmurs heard at the left sternal edge, and the echocardiographic findings.

While complete defects of the left pericardium appear not to have significant morbidity or mortality and require no treatment, sudden death may occur with partial defects.[6] Coronary arteries may be compressed at the margin of a partial defect, and herniation and incarceration of the cardiac apex may occur. With partial defects the chest X-ray is normal, except where cardiac incarceration produces a local bulge in the left cardiac contour.

We thank Dr J G Murtagh and Dr G C Patterson for permission to report the echocardiograph and cardiac catheterisation findings respectively.

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BOOK REVIEW

Davidson's Principles and practice of medicine. 15th ed. Edited by John Macleod, Christopher Edwards and Ian Bouchier. (pp 841. £17.50). Edinburgh: Churchill Livingstone, 1987.

I first read Davidson 30 years ago: at that time it was already in its 5th edition. A brief comparison shows that it then contained about 440,000 words in 1,100 pages: the current edition has compressed 625,000 words into 800 pages. I think I bought it as a student because it was cheap: but also because it seemed to be comprehensive and straightforward, and I have used it as a basis for my medical knowledge ever since.

So I miss some of the old pictures of the acute skin rashes such as scarlet fever: in fact infectious disease has been transferred to the back and genetic factors take the first chapters. But careful linguistic comparison will still uncover the old phrases which some of us know by heart — there are minor changes such as 'alarming reactions to intravenous iron are uncommon, but have occasionally been noted', which becomes 'alarming systemic anaphyllactic reactions can occur'.

Sir Stanley Davidson made 'no attempt to describe every rare disease or syndrome, but devoted most of the space available to those disorders most commonly encountered in practice'. I have grown up with successive editions, and have gradually come to appreciate the problems of the authors in the compression of knowledge. Having got to know many of them personally as real people rather than as names I can still recommend the book. It is the essential starting point for the study of internal medicine and for many doctors will remain their base reference work. The present editors have kept up-to-date and been prepared to prune the dead wood. There are many competitors in the market, and the publishers must take care with layout and illustration, although Davidson is still the best value for money.

I will continue to recommend it to my clinical students: they will need to read it and know it to pass final MB. Postgraduates will need to remember the facts, but also to be able to place them in a broader perspective. The older consultant will still happily read it, and to get to know the authors themselves is really to complete your medical education. Dr John Macleod and his team have successfully kept alive the primary objective 'to provide a rational and easily comprehensible basis for the practice of medicine'. The book is economical in price and compact in size, but still contains the essential truths for the practice of good medicine.

DRH

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Case report

Spontaneous oesophageal rupture in late pregnancy

S B Kelly, K J S Panesar

Accepted 20 February 1988.

Oesophageal rupture in pregnancy is extremely rare. Kennard [1] described the post-mortem findings of a ruptured oesophagus in a 37-year-old primigravida who suddenly collapsed and died shortly after a forceps delivery. The labour had been prolonged and the patient had earlier complained of dyspnoea but no chest pain. Henry [2] reported a case of spontaneous rupture of the oesophagus following severe vomiting in an 18-year-old primigravida at 10 weeks' gestation. She settled with conservative treatment, was discharged 12 days later and reached 32 weeks' gestation and presumably subsequent delivery without further problems.

CASE HISTORY

A 25-year-old primigravida was admitted at 35 weeks' gestation with a history of severe and excessive vomiting for one week and crampy abdominal pain for one day. Three days prior to admission, she suddenly developed retrosternal pain but there was no accompanying shortness of breath. She noticed swelling of the left side of her face and neck on the day of admission. On examination, a swelling was noted extending from below her left orbit to her neck. Crepitus was readily detectable over the swelling in keeping with subcutaneous emphysema. She was pale and dehydrated, pulse 110 per minute, blood pressure 130/90 mmHg. Her chest was clinically clear. The white cell count was raised, 22.9 $\times 10^9/1$. Chest X-ray showed mediastinal and subcutaneous emphysema but no hydrothorax or pneumothorax (Figure). A diagnosis of spontaneous oesophageal rupture was made. Oral fluid was prohibited and nasogastric suction was employed. Fluid and electolytes were given intravenously and she was treated intravenously with mezlocillin, gentamycin, metronidazole and cimetidine. Soon after admission, labour was induced because of the passage of meconium-stained liquor. At birth the baby's condition was poor, due to aspiration of meconium. The baby appeared dehydrated, with a blood urea of 15 mmol/l, which was equivalent to the mother's blood urea. A niopam and barium swallow carried out the following day in the mother failed to show a leak from the oesophagus. On the fifth day after admission, the nasogastric tube was removed, oral fluids were commenced and the antibiotics were stopped. Ten days after admission she was discharged.

Department of Surgery, Altnagelvin Hospital, Londonderry.
S B Kelly, FRCS, Surgical Registrar.
K J S Panesar, FRCS, FRCSE, Consultant Surgeon.
Correspondence to Mr K J S Panesar, Department of Surgery, Altnagelvin Hospital, Londonderry, Northern Ireland, BT47 1SB.

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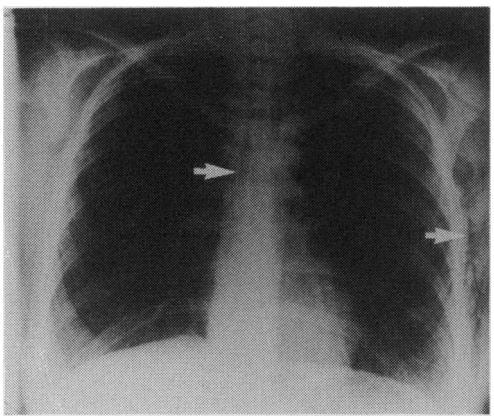

Fig. Chest X-ray demonstrating subcutaneous and mediastinal emphysema due to oesophageal rupture.

Rupture of the oesophagus can be the most rapidly fatal of all perforations of the alimentary tract. The most common cause is an episode of violent vomiting. The diagnosis is strongly suggested by the clinical triad of vomiting, chest pain and subcutaneous emphysema.[3] Pain is the most striking as well as the most common symptom although it may be minimal or absent as in this case. Anteroposterior X-ray of the neck, chest and upper abdomen taken with the patient erect is the most valuable examination, and will reveal the presence of air in the mediastinum, cervical tissues or pleural cavity before it is demonstrable clinically. In the present case the perforation must have occurred anteriorly and sealed itself off, accounting for the absence of a pleural effusion. A niopam and barium swallow failed to demonstrate a perforation in our patient, but gastrograffin or barium can slide past a sizeable oesophageal tear without extravasating unless the contrast is slowly injected down the nasogastric tube while it is being withdrawn.[4] Spontaneous oesophageal rupture usually requires emergency operation, although a few cases have been reported[2, 3] where patients have survived following conservative treatment as in this case.

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Pages from the past —

Extract from the memoirs of the late Dr R S Allison.

EPIDEMIC SMALLPOX AT SEA

My next experience was also a temporary one, as ship's surgeon on a vessel voyaging to the Far East in 1922. We had no refrigerators on board and no central heating. Cooling depended on fans attached to the bulkheads. On the outward voyage we carried no cargo or passengers and made a straight run from Liverpool to Port Said and the Red Sea in twelve days. Thereafter, our voyage took us down the Gulf of Sinai and the Red Sea until we called at a port on the Arabian coast called Jeddah, which was only some thirty or forty miles distant from Mecca. There we loaded the pilgrims who were returning from a pilgrimage to the Holy City. We lay off shore about a mile out because coral reefs abounded in the vicinity. At 10 o'clock the company's agent, a middle-aged Dutchman, took me ashore in his felucca to inspect the pilgrims and to make sure before they embarked that they were not suffering from any infectious diseases. On landing I was put in charge of a tall negro with a view to being shown around the town. The narrow streets, with open booths on either side taking the place of shops, were thronged with Arabs of the Hedjaz, tall bearded bedouins with rifles slung over their shoulders, negroes, stray dogs and beggars. Every now and then a string of camels or donkeys heavily laden with merchandise would push its way along the street, their progress hastened by the whips and the shrill cries of the native drivers. When we finally reached the Dutchman's house, lunch was provided, and after it we had a short rest before setting out to inspect the pilgrims. More than a thousand of them were awaiting in a big open warehouse or 'godown' for transport to Penang and Singapore. I could not fail to notice that many of the semi-naked pilgrims seemed to be affected with a skin disease, having large ulcerating sores on their skin. The Dutchman advised me not to be misled into thinking they might be suffering from smallpox. In reality, he said, they were suffering from endemic tropical sores and, as I could see by looking at them, they were obviously not ill with a disease like smallpox. In my innocence, and not having seen any examples of smallpox before, I readily agreed to this interpretation, and that evening saw 1,500 pilgrims embarked, and us steaming down the Red Sea en route for Penang in the Straits Settlements. However, I was not kept long in doubt that the Dutchman had pulled a fast one over me, for we had only been at sea for 72 hours when smallpox broke out on a large scale among our passengers, so that before we reached Penang there were some 300 cases. Fifteen had died and been buried at sea. Treatment was almost non-existent. The worst cases were put in the deck cabin at the stern, which served as a temporary hospital. Many of these were so covered with rash that it was virtually impossible to touch any part of the skin without encountering a pustule. This experience also gave me an object lesson into the value of preventive vaccination, for not one of our crew, all of whom had been vaccinated, became ill, although the decks and holds were full of the sick and dying. When we finally arrived at anchor off Penang and the Medical Officer of Health came on board, I was tempted to follow the captain's line and report the epidemic as 'chickenpox', but this brought down a storm on my head from the Medical Officer of Health who gave me an impromptu lecture on the distinction between smallpox and chickenpox. He pointed out, and I shall always remember his words, 'You see the

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rash in smallpox to best advantage when you inspect the patient's face and get him to hold his arms out crossed in front of him, for it is on the face and on the extremities that the rash is most conspicuous'. However, I was impressed to find that so few of the victims of smallpox had signs of constitutional disturbance, and it did not occur to me at the time that this simply illustrated the variability of the disease and the degree of immunity possessed by the sufferers.

One morning, before we reached Penang, I was hastily summoned from sleep by the boatswain to a confinement, but fortunately for the patient my rôle as an accoucheur on this occasion was purely that of an observer. Indeed, no forceps or help seemed to be needed. I found the patient lying on a mat spread on the deck. She was a young woman in the twenties and of typical Javanese blood. The baby's head was present at the vulva and it seemed an incredibly short space of time before the child was born. The only help she received was from another woman who stood by to tie the cord, the placenta being passed naturally within a few minutes afterwards. The patient was then turned on her face whilst two stalwart young women beat a sharp tattoo up and down her back with the ulnar edges of the palms of their hands. This remarkable treatment was most successful for within a few minutes the patient was able to sit up and eat some rice, grinning cheerfully as she began to nurse her baby at the breast. Later the same day I saw her going about the decks as usual.

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A HUNDRED YEARS AGO

The late Dr Hugh Calwell, among his many historical investigations, was responsible for organising the first index to the *Transactions of the Ulster Medical Society.* This record was published from 1884 to 1929, and was the predecessor of the *Ulster Medical Journal* which was first published in 1932.

As most of the diseases which were described by our predecessors are still with us one hundred years later, it will be of interest to scan the titles and authors of these early volumes. The full index to the *Transactions* is available in the Queen's University Medical Library. It is hoped to publish a detailed index of the *Transactions* and the *Journal* from 1884 to 1984 in due course.

The *Annual Reports of the Belfast Clinical and Pathological Society* antedate the *Transactions.* A note regarding these reports follows.

EXTRACT FROM THE *TRANSACTIONS OF THE ULSTER MEDICAL SOCIETY* (1887–88), pp 22–25.

Notes of some of the Gunshot, Revolver, and Buckshot Wounds which came under my notice during the Riots of 1886. By HUGH LEWERS, M.D., M.Ch.; Physician to the Ulster Hospital for Women and Children.

S. BROWN, aged twenty-three. Revolver wound in right arm. Bullet passed upwards, first through belly of flexors of forearm, and then obliquely through biceps, and was found lying at inner side of biceps, immediately over brachial. Made incision, and removed a ·450 revolver bullet, some pieces of clothing, and clots. At bottom of wound could see brachial pulsating. This bullet made altogether four wounds in arm — two entrance and two exit. No secondary haemorrhage, although sloughing was free. Did not heal for six weeks, as he engaged in other riots, drinking, and passed a month in gaol.

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Private Charles Fredrick Hughes, West Surrey Regiment, shot in Brickfields, by John Walker, July 13th. Lived about twenty minutes *Post mortem.* Bullet entered chest between 5th and 6th ribs, passed through lobe of lung, through left side of body of dorsal vertebra, and was embedded in muscles of back. Found on opening spinal canal that cord and meninges were uninjured, but anterior and posterior roots of nerves were cut away close to cord. Lung collapsed and pleura filled with blood. Death from shock and haemorrhage.

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July 13. — Watterson. Shot in Beverly-street. Saw him at 8 p.m.; found two small wounds on front of body, upper one to left side of sternum in nipple line; lower one at upper part of abdomen. Appeared like buckshot wounds. Probe would not pass owing to buttonholing, so could not tell if they had entered or dropped out. Patient was completely collapsed, and died in about ten minutes. *Post mortem.* Found that two pellets of buckshot had passed completely from chest to back; upper one between ribs through lung at root, and was embedded in muscles of back; lower pellet passed through abdominal wall, liver, and was broken into fragments by contact with body of lower dorsal vertebra. Internal haemorrhage was great, as pleural and peritoneal cavities were filled with blood.

This case shows the mistaken idea that buckshot is not dangerous to life. The range in this case was about forty yards. I have no doubt the pellets would have passed completely through the body had they not come into contact with bone. The *post mortem* search for buckshot is more difficult than one would imagine. In this case it was only after cutting the muscles into minute sections that the pellets were found.

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Two or three points struck me particularly when engaged in this sort of practice — the small quantity of blood lost, even when wounds were in neighbourhood of large vessels; the small amount of pain complained of during manipulation, whether from pluck or insensibility I cannot say; the fact that the exit wounds were invariably smaller than entrance wounds; the danger of the introduction of buckshot as an aid to keep the peace, the greater proportion of injuries in lower limbs, and especially buckshot wounds in front of thigh; the results obtained in cases of compound comminuted fractures by conservative surgery.

MISSING ANNUAL REPORTS OF THE BELFAST CLINICAL AND PATHOLOGICAL SOCIETY

The set of these reports held in the Queen's University Medical Library is incomplete and the Library is anxious to complete the set if possible. Missing are the Reports of 1855–56, 1856–57, 1857–58, and, while it is uncertain whether there were publications for 1860–61 and 1861–62, if there were, these also are missing. If any reader knows of the location of any of the missing volumes, the Librarian would be very glad to be informed. The Belfast Clinical and Pathological Society united with the Belfast Medical Society to form the Ulster Medical Society in 1862, so publication under their name must have ceased from that date.

J S LOGAN, Archivist, Royal Victoria Hospital.

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Book reviews

Sickle-cell anemia and thalassemia: a primer for health care professionals. By R G Huntsman. (pp 223. £5.00, paperback). St John's Newfoundland: Canadian Sickle-Cell Society, 1987. (Obtainable from The Canadian Sickle Cell Society, Sales Despatch, PO Box 13156, Station A, St. John's Newfoundland, AIB 4A4, Canada.

The haemoglobinopathies are perhaps the diseases in which the application of modern molecular biology techniques have been most helpful in unravelling the fundamental nature of the chemical defect, but sadly this knowledge has not as yet yielded any therapeutic gain. This small paperback sets out very clearly in two distinct sections the clinical and basic science features of this group of diseases. The clinical section emphasises the social problems faced by sufferers and the information on genetic counselling is good. The section on basic science is excellent, as is the glossary of terms encountered in dealing with all aspects of the disease.

Whilst haemoglobinopathies affect millions of the world's population, the problem is very rarely encountered in clinical practice here. This book will therefore be of very limited interest to those practising in Northern Ireland, but for any health care professional going to work in an area where the haemoglobinopathies are prevalent, this inexpensive book can be thoroughly recommended.

JMB

Road accident statistics. By T P Hutchinson. (pp 292. £28.00). Adelaide: Rumsby Scientific Publishing, 1987.

This book contains a comprehensive record of the information available about road accidents in many different parts of the world, including some developing countries. Data from police and insurance companies as well as medical statistics are used. Methods of data collection by the different groups are described and the major deficiencies in each are illustrated. The merits of different systems of classification of injuries and injury severity scoring are discussed. One chapter describes attempts to integrate police and hospital data to produce more information relating the accident cause to the nature of injuries sustained. Another gives statistical details on the effectiveness of seat belts and crash helmets in reducing mortality. We are also told that high blood alcohol in driver fatalities is worse in Illinois, Texas and Wisconsin in the USA, and Counties Dublin and Kildare in the Republic of Ireland than in other parts of the world.

Mr Hutchinson from the Department of Civil Engineering at the University of Adelaide has produced an excellent reference book which is recommended to anyone interested in road accident statistics.

CHD

Cardiopulmonary cerebral resuscitation: an introduction to resuscitation medicine. By Peter Safar and Nicholas G Bircher. 3rd ed. (pp 464, illustrated. £11.50). London: Saunders, 1988.

The third edition of this now classic book has doubled in size from the previous one. This makes it not only an excellent practical manual, but also a good book for consultation, with references included up to late 1987. The division of the book into three major parts is a useful exercise with the *Basic life support* chapter leading logically on to the *Advanced life support* and then the *Prolonged life support* sections. In practice, these stages can merge together very quickly. However, the divisions do delineate different types of teaching and practice for different groups involved in resuscitation.

The book is packed with useful information, and by all who are, or are likely to be, involved in resuscitation, should be read and considered well away from the hustle of the acute emergency. Its size is somewhat daunting in the acute situation, but the excellent method of highlighting the major messages in the text makes quick consultation easier than might otherwise have been expected. There are many excellent, instructive diagrams and tables, though one or two have rather small print or are unduly complex. The text dealing with central venous pressure measurement correctly advocates the use of the right internal jugular vein for central venous cannulation, but the complementary figure illustrates cannulation of the left internal jugular vein — a procedure with specific hazards, which are not mentioned.

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A book on resuscitation should be present in all theatres, high dependency and intensive care units and accident/emergency areas to provide basic reading and help in acute life-threatening situations. This book can be recommended for the purpose.

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