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The Ulster Medical Journal, Volume 57, No. 2, October 1988.

The Ulster Medical Journal

THE ULSTER MEDICAL JOURNAL

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The Ulster Medical Journal

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1988

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The Ulster Medical Journal, Volume 57, No. 2, October 1988.

THE ULSTER MEDICAL SOCIETY

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The Ulster Medical Journal, Volume 57, No. 2, pp. 101 – 120, October 1988.

The madhouses and mad doctors of Ulster

R J McClelland

Inaugural lecture, The Queen's University of Belfast, 21 October 1986

Accepted 29 October 1987.

'If we act only for ourselves, to neglect the study of history is not prudent: if we are entrusted with the care of others, it is not just.'
Samuel Johnson

Nineteen eighty-six was a most significant year in the life of the mental health services in Northern Ireland. The introduction of new and progressive legislation updates the landmark of reform — the 1961 Mental Health Act. The Royal Commission on Mental Health (1957) which laid the foundation to this Act articulated the philosophy of change — a change from institutional care to community care. This same year we had DHSS (NI) planning guidelines for mental health, which enshrined the principles of community care. Indeed the forces for change currently embrace professional interest, public opinion, legal provision and now Government strategy. Given such impetus for a shift from one major model of care, the mental hospital, it is appropriate to ask how such a form of provision arose in the first place. Where did these institutions and their providers come from? — Does their history have anything to teach us about the way ahead?

While the present reforms in psychiatric care occurring throughout these islands and indeed throughout much of Western society require major shifts in the balance of service provision, in styles of practice and in professional relationships, they are no more dramatic than the changes achieved by the first reformers in the early years of the nineteenth century.

To understand something of their achievements it is important to appreciate the context in which these developments took place — the nature of community in 18th and 19th century Ireland, the prevailing social attitudes which accepted the appalling conditions for the lunatic poor.

In the eighteenth and nineteenth centuries, superstitions abounded regarding the insane and their treatment. One of these gives Ulster its first link with asylum for the insane — at Glennagalt, the valley of lunatics, in Co. Kerry. It was once believed that all lunatics would ultimately, if left to themselves, find their way to this glen to be cured. The origins of the superstition go back to the legionary tale of Cathfionntra, or the Battle of Ventry in the Dingle Peninsula.[1] This tells of how Daire an Dornmhar, the monarch of the world, landed to conquer Erin. He was opposed in mortal combat by Finn MacCumhaill and his men. In the course of the battle Gall, son of the king of Ulster, came to the help of Finn MacCumhaill. After

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performing many outstanding deeds of valour, Gall fled in a state of derangement from the scene of slaughter never stopping until he plunged into the wild seclusion of Glennagalt. Gall was believed to be the first lunatic who went there, following which there have been many pilgrimages to this beautiful 'valley of lunatics' and its wells.

Turning from mythology to early history, before the Elizabethan period insane persons in Ireland were given considerable protection under the Brehon laws.[2] Based on the old Mosaic laws, they spelt out the social obligations to the mentally ill. The reality might well have been somewhat different from what the laws required. Some of the insane were certainly cast adrift, but from available evidence few were deliberately persecuted, as happened elsewhere in these islands.

Ireland's provision for its lunatics was not evolving in a vacuum and was greatly influenced by the social and political events on the larger island. By the end of the sixteenth century, Elizabeth and her armies had completed the conquest of Ireland and the remaining vestiges of the old Irish social order disappeared.[3] Throughout most of the seventeenth and eighteenth centuries, the requirements of an increasingly ordered society placed great emphasis on conformity and on the obligation on each individual to behave and conform. Vagrancy, drunkenness, witchcraft all tended to disturb the social and religious order and demanded restraint. To help maintain this process a public policy developed of incarcerating the non-conforming, the nuisance and the insane as well as the criminal.[4] Indeed, little distinction was drawn between them. Madness was seen as a matter of deliberate and perverse choice rather than the inescapable consequence of a sick mind.

Local communities recognised little corporate responsibility for the wellbeing of its citizens. As Beckett remarked on Irish social provision, 'It was the claims of the Kingdom of Ireland rather than the welfare of the bulk of the inhabitants that engrossed the attention of politicians'.[5] The country did not have the official benefits of a Poor Law legislation until late in the eighteenth century. In addition, the population was growing rapidly and the country's dependency on a poor agricultural economy was even greater than in England.[6] As a result, pauperism exploded and inevitably the more vulnerable, which included the insane, suffered most.

In 1771 Ireland was provided with an elementary Poor Law. Through a system of badging, the 'deserving poor' were allowed to beg. Grand juries in each county were also empowered to establish workhouses, or 'houses of industry', for vagrants, the destitute and the infirm. However, provision was made only in Dublin, Cork, Limerick and Waterford.[8]

EIGHTEENTH CENTURY PROVISION FOR THE INSANE

Specialised confinement of any sort for the insane was virtually absent. The one asylum was founded in 1745 by Jonathan Swift, Dean of St Patrick's Cathedral, Dublin. By 1800 it provided only 106 beds for the lunatic poor;[9] the great majority of lunatics were in the community. When their behaviour became unmanageable they were confined in the gaols or in the newly established houses of industry.

In the gaols, the jailers were unpaid and given free rein to extract money from their prisoners, in return for privileges. The moneyless, who invariably included the insane, lay on the flagstones.[10, 11] Conditions in the houses of industry were just as bad. They had become places of punishment rather than charitable institutions. Thomas Spring Rice, one of Ireland's leading reformers in the care of the insane, giving evidence to a Government Select Committee on the Limerick House of Industry, reported: 'Two and sometimes three patients occupied cells which were about six feet by ten feet. Those in a state of furious insanity were restrained by having their hands pressed under their knees and manacled in that position. Their ankles were secured with bolts.'[11, 12]

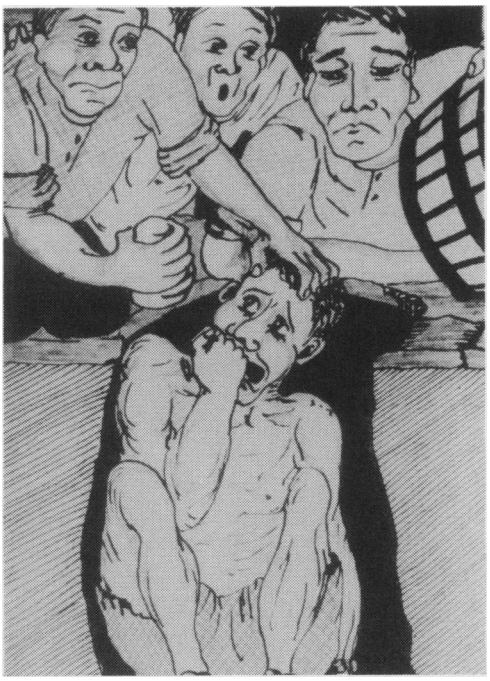

Fig 1.
Managing madness in the cabin of a peasant.

Other evidence to the Government Select Committee gives a graphic description of community care (Fig 1). 'There is nothing so shocking as madness in the cabin of the peasant.[11] When a strong man or woman gets the complaint, the only way they have to manage is by making a hole in the floor of the cabin, not high enough for the person to stand up in, with a crib over it to prevent his getting up.[11, 12]

THE BEGINNING OF A NEW ERA OF REFORM

It was from within this social framework that the first era of reform had its origins. As the eighteenth century draws to a close we return briefly to the larger canvas. The French Revolution, inspired by the demands for liberty and equality, encouraged and advanced social change in many countries. By the end of the eighteenth century, radical social developments were afoot which finally embraced the rights and needs of the disadvantaged including the insane.

Philippe Pinel, physician in charge of the Bicêtre asylum in Paris in 1793, liberated his patients from their chains and ordered that henceforth they should be treated with kindness and understanding.[13] While his action reflected the humanitarian spirit abroad in France at the time, it was also a manifestation of his views of mental illness as a natural phenomenon. New philosophies and developing sciences constituted a shift from a supernatural to a natural perception of world affairs, and with it a shift from acceptance of the *status quo*. Pinel's theories about insanity[14, 15] were greatly influenced by William Cullen, Professor of Medicine at Edinburgh, one of the most influential figures in medicine in the British Isles.[16]

For the first light of reform in these islands we look to the city of York, where, in 1790, a patient called Hannah Mills was admitted to the city asylum.[17] Her

relatives, who lived at some distance away, recommended her to the care of the Society of Friends. Members of the Society who attempted to visit her were refused admission on the grounds that she was 'not in a suitable state to be seen by strangers'. Hannah Mills subsequently died under circumstances which aroused strong suspicions of ill treatment and neglect. William Tuke, head of a Quaker family in York, resolved that a retreat should be established where there would be no concealment and where patients would be treated with 'all the kindness which their condition allowed'.[18] Tuke's resolve was strengthened by a subsequent visit to St Luke's hospital in London, where he found patients bedded on straw and in chains.[19] Pinel and Tuke had concurrently, and initially unknown to one another, pioneered methods of treatment based on common sense and enlightened humanitarianism.

One other factor influenced the process of lunacy reform in these islands — the occurrence of mental illness among the élite. On several occasions since becoming monarch in 1760 George III was seized by periods of insanity.[20] The first to become public was in 1788 and the episode lasted for several months. No-one in the writings of the day suggested he was being punished by heaven or possessed by the devil. Again many well known writers and poets were, if not psychotic, at least highly neurotic. Notable were William Cowper, Christopher Smart, Charles Lamb, Dr Johnson and William Blake. Well might Wordsworth write in 1802 in *Resolution and Independence*:

> 'We poets in our youth begin in gladness;
> But thereof comes in the end despondency and madness'.

LUNACY REFORM IN IRELAND

In the closing years of the eighteenth century, Irish legislature began to recognise the special problems of the insane. The first light of innovation in lunacy provision is to be found in prison reform. In the Irish Aquitted Prisoners' Act of 1763,[21] the health of prisoners was given consideration for the first time. This antedated British prison reform by more than a decade. Segregation was proposed beginning with separation of the insane. In 1786[22] new prison legislation permitted the establishment of separate lunatic buildings throughout Ireland and a system of inspection. However, like so many Acts concerned with social provision, it was merely permissive and special accommodation was again limited to Dublin, Cork, Waterford and Limerick. In fact, national asylum provision began very humbly with 10 cells at the Dublin House of Industry.

As Kirkpatrick remarks in his short history of lunacy provision in Ireland 'It was around the Houses of Industry that might be written the history of Irish medicine'.[23] They provided the only haven for the infirm and invalid poor. Conditions at the half-dozen houses of industry were extremely limited. At Limerick they were particularly appalling when John Carr visited in 1800 — walking towards the House of Industry 'the traveller will quit a noble city gay with novel opulence and luxury for a scene which will strike his mind with horror'.[24]

Provision hinged on the innovation and dedication of local reformers. In this respect, conditions at Cork were in striking contrast to those at Limerick, where, following the appointment of Dr William Hallaran in 1791 a separate asylum for the insane was established. Hallaran had his medical training at Edinburgh where he too came under the influence of Cullen.[25] He was the first Irish doctor to write at length on the problems of insanity.[26]

Just four years after Halloran's appointment as a physician to the House of Industry in Cork, Alexander Jackson was appointed attending physician to the Dublin House of Industry. Jackson was a native of Co. Tyrone and educated at Dungannon Public School.[27] Like Hallaran he had had his medical education at Edinburgh, again under the influence of Cullen. After a short period of medical practice in Lurgan, he settled in Dublin in 1795 and, soon after, began work at the hospital connected with the House of Industry. In the years ahead, Jackson would play a major rôle in the establishment of Ireland's public asylum system.

NINETEENTH CENTURY LUNACY PROVISION

In 1800 Ireland lost its parliament and became part of the Union. Our attention must now return to the political arena and the efforts of one of Ireland's key reformers, Sir John Newport, who sought to introduce new legislation for the care of the insane. Newport was a Whig and a keen supporter of Catholic emancipation. As MP for Waterford and Chairman of the local House of Industry, he was aware of the inadequacies and overcrowding at Cork and Dublin, due in no small way to the inadequacies or absence of provision in other parts of Ireland. As he noted, under the existing legislation 'it is entirely optional in the Grand Jury whether they will grant any and what sum of money for the erection of houses for insane persons'.[28] In 1804 he proposed to Parliament that a committee be set up 'to consider legislative provisions for the support of the aged and infirm poor of Ireland and making provision for the care of lunatics and idiots'. The report,[29] presented just two months later, recommended the establishment of four asylums, one in each of the provinces 'appropriated exclusively to lunatics and idiots'.

On 21 March 1805, Newport introduced an Irish Lunatics' Bill; but his attempt failed by just four votes. As Arthur Williamson's careful analysis[30] suggests, the Bill fell victim of a fierce inter-party debate concerning Lord Melville's financial management of the Admiralty.[31] Party feelings appeared to influence the voting pattern of the Whig-led proposals for Irish lunacy reform — a pattern not so uncommon in parliamentary handling of Irish affairs.

The next stimulus for reform in Ireland came in 1810 from the pen of an Ulster physician, Dr Thomas Hancock, writing in the *Belfast Monthly Magazine.*[32] Hancock had several links with Quaker reform. First he had attended the Quaker school at Ackworth in York founded by William Tuke. Tuke's son had been a contemporary at Ackworth, and was now active in lunacy reform. Both he and Hancock corresponded on these issues. Hancock made a powerful plea for the introduction of moral and humane methods of treatment stating that 'the dominion of fear will not produce a change like the domination of confidence and esteem'. That same year the Governors of the Dublin House of Industry presented a memorial to Parliament 'that the number of lunatics and idiots for some time transmitted from all parts of Ireland to this institution has so much increased as to render additional and appropriate buildings necessary'.[33] To this Jackson added a personal plea in a letter to the Lord Lieutenant.[12]

Parliament finally responded by authorising the establishment of Ireland's first public asylum in Dublin. It was to be named in honour of the Lord Lieutenant, Charles Lennox, fourth Duke of Richmond. Jackson visited a number of English

asylums, including the Retreat at York, and, on his return, submitted guidelines for running the asylum. These progressive and enlightened views included that every patient should be examined and the state of his mind ascertained before being admitted. He also stressed the need for a resident medical officer who would not follow any business other than the hospital.[27] Francis Johnston, from Co. Armagh, Architect to the Irish Board of Works, designed the new asylum which was to provide for two hundred and fifty patients.

The Richmond asylum was opened in 1815 and Jackson duly appointed as its visiting physician (Fig 2). From the beginning, the Governors, guided by Jackson, laid heavy stress on moral or psychological methods of treatment. The duties of its superintendent were based on those at the York Retreat. John Leslie Foster, MP for Armagh, giving evidence to a parliamentary committee two years later reported: 'there is not in the Richmond lunatic asylum to the best of my knowledge, a chain, a fetter or a handcuff'.[12]

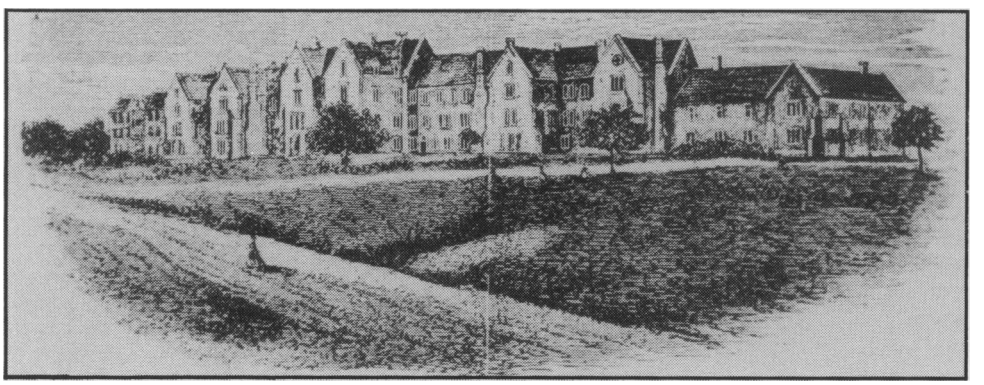

Fig 2.
Front elevation of the male department of the Richmond district lunatic asylum, erected in 1854.

It was originally hoped that the Richmond would provide sufficient accommodation for a large proportion of Ireland, but in November 1815, within six months of its opening, the Governors wrote to the Chief Secretary, concluding that 'the relief of lunacy could be neither fully nor conveniently met by lunatic establishments confined to the capital'.[12] They recommended enlargement of the lunatic asylum in Cork and the formation of a similar institution in Belfast to contain one hundred and fifty patients. The criterion for their choice of sites was that 'it would not be difficult to procure a sufficient number of intelligent and benevolent governors to serve without fee or reward'.

The major driving forces behind the provision of asylum in Ireland at this time were Sir John Newport, Thomas Spring Rice, MP for Limerick, and Robert Peel, who in 1812 had been appointed Chief Secretary for Ireland. We return briefly to the mainland for events which must have greatly influenced Peel and Newport. In 1815 the reformer George Rose, on hearing of a young woman found chained by both legs and arms in a private madhouse, moved 'that a committee be appointed to consider . . . the better regulation of madhouses in England'.[34] It is of significance that both Newport and Peel were members of this committee. The weakness of the permissive legislation introduced in 1808 for the provision of county asylums in England and Wales is revealed in the committee's findings.

Gross maltreatment was again uncovered at the York lunatic asylum with forging of records to hide deaths among patients. There was widespread use of chains and other forms of mechanical restraint. Similar conditions were discovered at Bethlehem Royal Hospital, or 'Bedlam', where the inspecting party found patients left naked or covered only with a blanket. Many had been chained to the walls of their cells for weeks and months at a time. Among them, the most notable was James Norris, in chains continuously for eleven years, immortalised through the work of the artist accompanying the visiting team. By the time the 1815 committee submitted its final report there was a wealth of documentation to support the reformers' contention that the lot of madmen in every sort of institution was one of appalling degradation and inhumane treatment.

It is of significance that the year following, Peel initiated an enquiry into lunatic provision in Ireland and, shortly after, in an address to the House of Commons, moved that 'a committee should be appointed to enquire into the expediency of making further provision for the relief of the lunatic poor of Ireland'.[34] The committee appointed consisted principally of Irish members and included Sir John Newport, John Leslie Foster, then a governor of the Richmond Asylum, and Thomas Spring Rice. After first considering accommodation at infirmaries, the committee recommended that distinct and separate lunatic asylums should be established. A major factor in this decision was the evidence given by Foster regarding the successful adoption of moral methods of treatment at the Richmond. It was considered impossible to establish such methods by untrained staff and where staff would not be devoted wholly to the care of the insane, 'The only mode of effectual relief would be found in the formation of district asylums . . . exclusively appropriated to the reception of the insane'. Separate provision for the insane therefore was not, as some have suggested, a mere symptom of social rejection; it was based chiefly on a therapeutic argument.

Within two weeks of the report being tabled, a Bill, drafted by Spring Rice, was presented to Parliament and became law on 11 July 1817 (57 Geo III C106, 1817). It differed in several important respects from Wynn's English Act of 1808. First, a comprehensive network of lunatic asylums was proposed. The country was to be divided into administrative areas in each of which an asylum would be established. Second, responsibility was placed centrally in the hands of the Lord Lieutenant and not with local magistrates as in England. Third, in an amending Act, additional power was given to the Lord Lieutenant to institute a system of inspection. The establishment of a centrally organised network ensured certain common principles and, modelled on the Richmond, Ireland's asylum network incorporated the 'moral' approach to treatment from its outset, characterised by humaneness and work therapy. The moral method of patient care was not introduced into the English district lunatic asylums until 1840.

THE DISTRICT ASYLUMS OF ULSTER

The report of a Government Committee of 1816[36] provides us with a glimpse of lunatic provision in Ulster 170 years ago, just before the establishment of the asylum network.

In Donegal, the Lifford gaol had an appendage for sixteen which was 'always full' and 'many patients recommended for each vacancy'. In the county and city of Londonderry, twelve cells were provided in a building, or rather a shed, in the

grounds of the county infirmary. In the county of Tyrone, four cells were appropriated for pauper lunatics in a new building added to the local gaol. The remaining counties had no provision whatever. The report lamented that 'the pauper lunatics who were committed to gaol are so miserably neglected and ill provided with every common necessity as makes it shocking to human nature to witness'.

According to William Todd, Secretary of the Asylum Commission, 'lunatics abound more in Ulster than any other part of Ireland'.[37] Such a perception of the high prevalence of lunacy almost certainly derives from two issues. First was the lack of any alternative institutional provision in Ulster at this time. Second was magnitude of the poverty-stricken population.[38] But perhaps the misperception of higher prevalence of lunacy was the reason why Ulster would obtain the first of the new asylums.

It is appropriate that Armagh, Ulster's oldest city, should be the first city to have a district asylum in Ireland. Early in 1819 negotiations took place between the Secretary of the Lunacy Commission, William Todd, and William Gregory, Archbishop of Armagh. Four acres of land were obtained from a Mr Thornton and a board of governors established.[39] The foundation stone was laid in May 1821, again based on plans furnished by Francis Johnston (Fig 3).

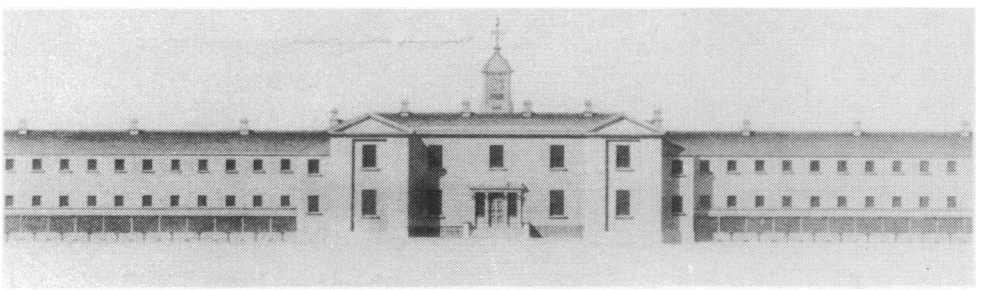

Fig 3. Front elevation of Armagh asylum.

The first meeting of the Board of Governors took place in December 1824,[40] and a quota system was established for admissions for each of the counties within the Armagh district, based on the census of 1822 (Tyrone 27, Donegal 26, Armagh 20, Fermanagh 13, Monaghan 18). The first patient was admitted on 14 July 1825.

The manager of the asylum was Thomas Jackson, formerly in charge of the lunatic department of the Dublin House of Industry. This second Jackson came to his post with considerable experience in the treatment of the insane poor and was deeply committed to moral methods of treatment.[41] His views of the value of employment as a therapeutic tool are noteworthy: 'The poor lunatic, when left to himself, without occupation or the busy and active scene of some pleasing employment, soon graduates into a state of incurability or idiocy and is left a burden to himself and to the community'. Such comments indicate the insight of these early managers into the problems of institutionalisation which would pervade the overcrowded asylums of the late nineteenth and early twentieth century. Jackson's management principles were to be replicated in the network of asylums now being established throughout the country.

Four years later the next two of Ulster's district asylums were established. Until

this time the only provision for lunatics in Londonderry consisted of the twelve cells at the local county and city infirmary erected in 1810. The population of the city at the time was almost 20,000. From Colby's 1832 ordnance survey of Ireland [42] we read 'The lunatic asylum is uniform with the asylum at Armagh and, like it, is a district asylum, being intended for the counties Londonderry, Donegal and Tyrone. It is a handsome building, situated on rising ground without the city on the north'.[11] (Fig 4).

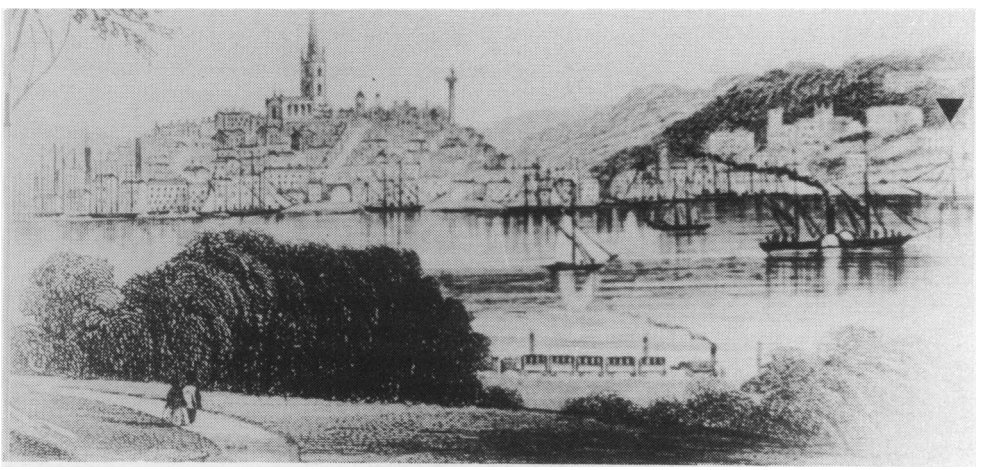

Fig 4. City of Londonderry and its asylum (far right). (By kind permission of Mr D Bigger).

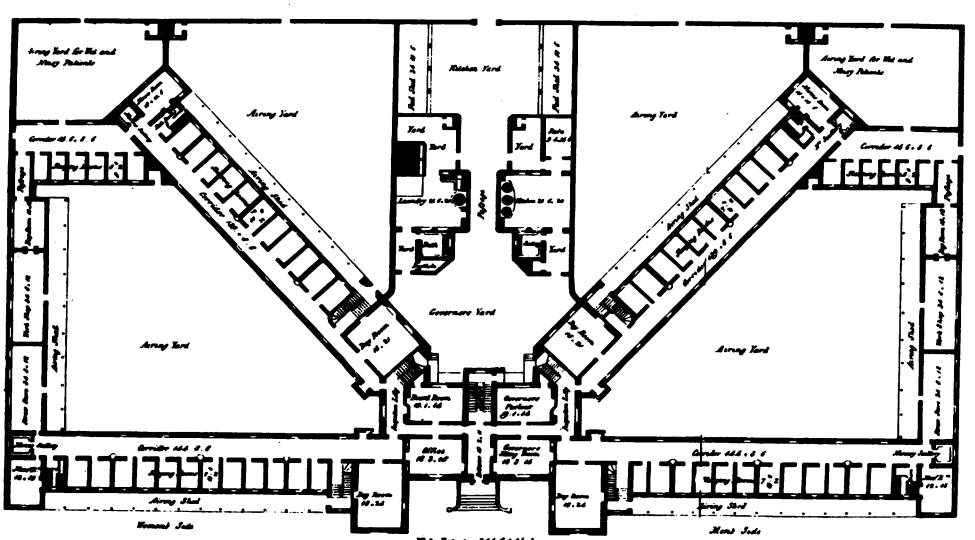

Fig 5. Plan of the Belfast and Londonderry lunatic asylums.

Again, the plan was that furnished by Francis Johnston. The panoptician design (Fig 5) gave the manager maximum surveillance of the entire hospital complex. The opening of the Derry asylum on 7 August 1824 soon relieved Armagh of patients from Tyrone and Donegal.[43]

We now turn to the establishment of the district asylum at Belfast. The history of social and medical care in the city is intimately bound up with the history of the Belfast Charitable Society. As the historian Owen observes 'few philanthropic societies have done more useful work or exercised more varied functions than the Society'.[44] It is therefore fitting that we turn to it for the earliest provision for the insane of the town. In 1802 two rooms were appropriated at the Charitable Society 'for the reception of such deranged persons as may belong to and have resided two years in this town'.[45] However, the general state of misery of the insane soon led the committee of the Charitable Society to 'consider the propriety of applying to the Lord Lieutenant to have a lunatic asylum erected in this town'. (Fig 6).

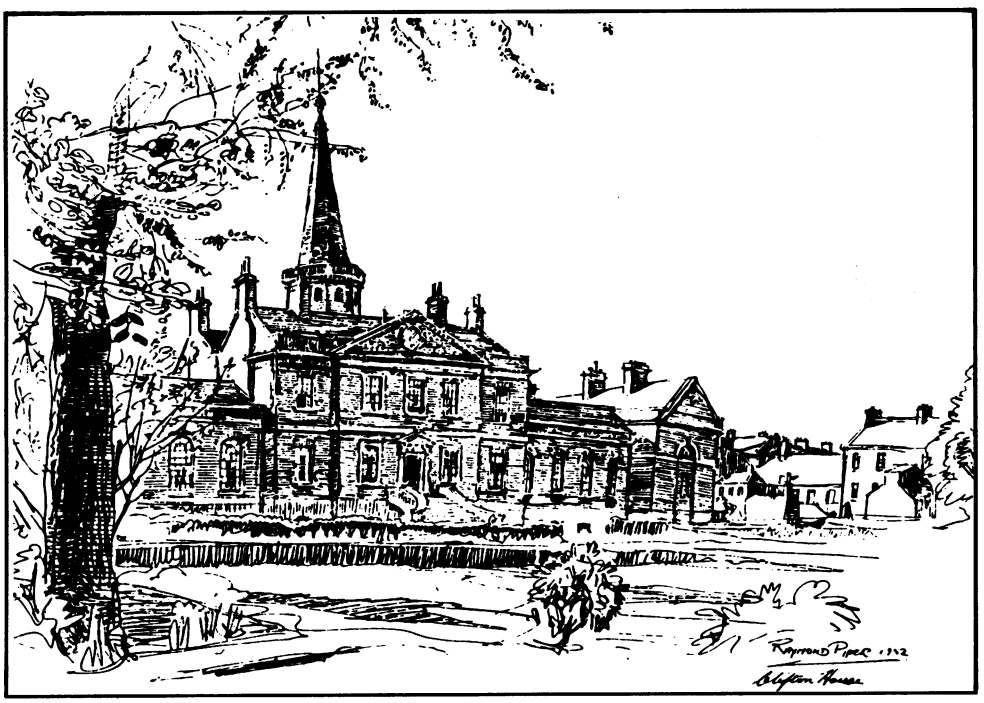

Fig 6. Belfast Charitable Society. (By kind permission of Dr RWM Strain).

The significance of such a proposal lies in the local initiative. While the new legislation of 1817 empowered the Lord Lieutenant to establish asylums, Belfast did not wait to have one proposed. Several abortive attempts were made to purchase land. Then in 1825, 'a joint deputation was formed from the committees of the District Hospital, the Charitable Society and the House of Industry in an effort to induce the Government to fix on Belfast as the most proper site for the new lunatic asylum'.[46] Land was finally obtained on the Falls Road. Totalling 33 acres, it was a larger site than any asylum either before or after. Indeed the grounds of the old asylum would eventually accommodate the entire Royal Victoria Hospital complex. (Fig 7).

A board of governors was formed and their first meeting was held on 20 May 1829.[47] It is most fitting that Dr James McDonnell, the father of Belfast medicine, was appointed as the hospital's first visiting physician. The first superintendent was Cummings who, like his colleagues at Armagh and Derry, came from the

Fig 7. Ordnance survey map (1923) of the Belfast lunatic asylum site showing its relationship to the present Royal Victoria Hospital, built in 1902.

Dublin House of Industry. The catchment area for the asylum included the City of Belfast, which at that time had about 30,000 inhabitants, the counties of Down and Antrim and the town of Carrick. On 7 July 1829 the first patients to be admitted were transferred from the Antrim gaol. (Fig 8).

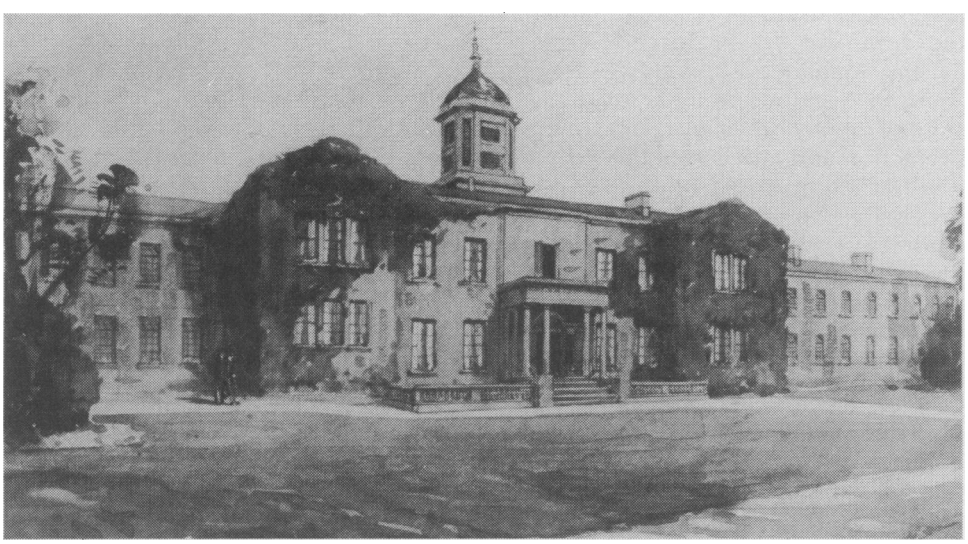

Fig 8. Belfast Lunatic Asylum. (By kind permission of the Ulster Museum).

By 1832, within 15 years of the Act of 1817, six more asylums were established in Ireland. The first major objective had been reached, the formation of a network of district lunatic asylums (Fig 9). In 1832 James MacDonald, physician to the Bloomingdale Asylum in New York, applauded the new district asylums in Ireland which formed 'a more complete system than the English'.[48] The eighth report of the Inspectors General of Prisons stated, 'The present asylums in Ireland are superior to anything of the kind in Europe, and the whole system of cure, chiefly consisting of employment, kindness, moral government and freedom from restraint is worthy of examination as a good example'. Sir Andrew Halliday, one of the key English lunacy reformers, in his assessment of the Irish asylums considered 'The system is so excellent and has been found to work so well that I am anxious it should be imitated in this country'.[49]

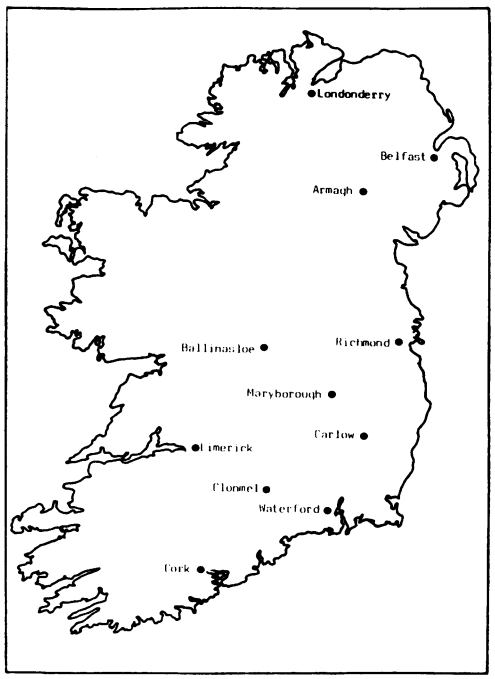

Fig 9.
The new network of Irish district lunatic asylums.

An interesting debate initiated by the French writer Foucault[50] and continued by Andrew Skull[51] suggests that the main force behind the establishment of asylums throughout the western world was the need to dispose of socially unacceptable individuals within an increasingly controlled and capitalist society. From this review of early Irish records, humanitarian concern would appear to have been the dominant driving force for the reformers. But what of the motives of the asylum managers? On this the writings of Thomas Jackson, manager of the Armagh Asylum, are most revealing. In response to an enquiry in 1827 into the state of lunacy provision, Jackson had this to say: the asylum was there 'to afford protection and ameliorate the sufferings of one of the most afflicted classes who, if left at large, would be the sport of the unfeeling rabble. Where cure may be impossible, to ameliorate their sad condition is not . . . It shall be my constant study to render the establishment commensurate with the benevolent views of the legislature'.[41] These were hardly the words of a custodian committed to protecting society from undesirables. Such was the spirit and driving force behind this new profession of carers in the field of mental health in Ireland.

The austere buildings may seem an anathema to the proponents of community care for the mentally ill today. Nevertheless, the relative comforts and asylum provided by these great institutions protected thousands of lunatic poor from the abject poverty and misery that the rest of Ulster's poor, now approaching one million, experienced at this time.

In Ulster as in the rest of Ireland, asylums provided relief for families in desperation.[7] One parameter of the relative success of Ulster's asylum provision

was the number of lunatics still residing in gaols. Even by the late 1830s, 30% of known lunatics in Ireland were still within the country's gaols. In Ulster, only 16% were resident and the majority of these were from the counties of Tyrone and Fermanagh, which at that time did not have a county asylum.[52]

THE BIRTH OF PSYCHIATRY IN IRELAND

Fig 10. Dr Robert Stuart, Ireland's first Resident Medical Officer. (By kind permission of Belfast Harbour Commissioners).

We now turn to a third event which would form another turning point in the history of Irish psychiatry. At the Belfast Asylum in 1835 the manager Cummings retired and in his place a Dr Robert Stuart was appointed. He became Ireland's first Resident Medical Officer, forerunner of the profession of psychiatrists.[53] (Fig 10). Dr Stuart was a graduate of Glasgow University where he obtained his MD in 1828, some five years after Dr John Connolly,[54] regarded by many as the father of British psychiatry. Both men came under the influence of the great clinical teacher Robert Cleghorn, Professor of Medicine and Chemistry at Glasgow University and physician to the Glasgow Asylum from its opening in 1814.[54] Cleghorn was considered an authority on running 'lunatic wards' in general hospitals and the Glasgow Asylum itself was inspired by the example of the Retreat at York and had a clear policy of minimising restraint. Stuart, following in this new tradition, pronounced himself a warm adherent of the non-restraint system of treatment. As the *Journal of Mental Science* noted, 'He was the father of the Irish asylum service and was looked up to with feelings of greatest respect and confidence by his colleagues in the profession, and by the general body of Irish superintendents'.[55]

During the forty years when Stuart was the chief officer of the Belfast Asylum, the institution attained a high reputation for the skill and humanity which guided its administration. Many eminent clinicians from the United States and Europe visited the Asylum.[56] Samuel Tuke from the York Retreat commented 'I have gone through this hospital for the insane with a high degree of satisfaction . . . There is an air of ease and comparative comfort in the general aspect of the patient, which has given me the most favourable impressions of the principles of management which are carried out by Dr Stuart'.[57] Richard Eades from Dublin, and formally a pupil in La Salpêtrière, commented 'having visited many similar institutions, both on the continent and in Great Britain, I was happy to find the Belfast Asylum second to none'. Unlike any other asylum in Ireland at the time it had a wing for long-stay patients and had its own infirmary. Dr Stuart was also

highly regarded by his medical colleagues in Belfast and as a member of the Ulster Medical Society was elected its third president.[58]

It is therefore with some surprise that we read a rather hostile and frosty editorial in the *Dublin Medical Press* of 1844, 'We are not at present prepared to advocate the appointment of a resident physician to an asylum for some three hundred patients, to have paramount control in the treatment of patients . . . We do not expect that benefit could arise from having the insane confided to the care of physicians, so doubtful of their own success in general practice, or so destitute of public support, that they would accept of such an appointment as that held by the managers of our asylums'.[59] It is perhaps even more surprising that such criticism should come from a member of Dr Stuart's own profession, Dr Jacob, the editor. Of considerable significance, however, is the fact that the brother of the editor, Dr John Jacob, was visiting physician to the Maryborough Asylum, Dublin.[60]

The true target of hostility becomes apparent when we read subsequent editorials in which criticism is also directed at Dr John Connolly who in 1839 pioneered at Hanwell Lunatic Asylum the non-restraint method in English asylums. Reporting on a second death at Hanwell, the editor of the *Dublin Medical Press* protests, 'We certainly have fallen upon the days of quackery, and, next to the quackery of knaves, the hobby horsical quackery of vain applause courting enthusiasts is the most inconvenient'.[61] The target for such criticism and abuse was not the individuals themselves but an emerging profession dedicated to the care of the insane — the Resident Medical Officer. Connolly supported these developments in Ireland and so had become a focus for criticism by those members of the profession who defended the supremacy of the visiting physicians.

At the centre of the debate was a struggle, a struggle for control. In 1843 legislation took control away from the asylum managers, two of whom were doctors — Stuart at Belfast and Flynn at Clonmel. The 1843 Act[62] placed managerial responsibility in the hands of the visiting physicians. A prime mover in this shift of control from the asylum managers to the visiting physicians was Francis White, the first medical inspector and a past president of the College of Surgeons in Ireland. Yet when White visited Belfast the year following the introduction of legislation he was persuaded to waive the rules in the light of what he described as the excellent superintendence of the entire physical and moral treatment of the patients at the Asylum, provided by Dr Stuart.[63, 64]

There is no evidence in any of the Belfast records to suggest a medical power struggle in either the appointment or full authority of the Resident Medical Manager.[65, 66] Indeed, the very opposite — as evidenced by the fact that in 1845 a testimonial was signed by all senior medical practitioners of Belfast recommending Stuart as a candidate for the office of Inspector of Lunatic Asylums. Such an atmosphere nurtured the growth of a new professional group. The present managerial system of our psychiatric hospitals can be likened to a gestation process. Its conception in Belfast was a very positive event but the gestation period was lengthy and stormy. For almost twenty years there was a 'tug of love' for control of the asylums.[67–70] Some have considered the struggle to be between the medical profession and lay managers. While this was indeed a factor, there was as much resistance to the establishment of resident medical officers as there was to the subordination of lay managers. Would asylums continue to be controlled by the long-established body of general physicians or should they be managed by a new speciality?

At the Commission of Inquiry held in 1856, no agreement could be reached on

the status of visiting physicians. The eminent physician Dr Dominic Corrigan argued 'that visiting physicians are a necessary appendage to lunatic asylums'. But the majority view of the Commission was 'that the resident physicians should have charge of the asylum, and be responsible for the treatment of the inmates as regards their insanity'.[69] In 1858 a Bill to amend the law relating to the lunatic poor in Ireland was introduced by Lord Naas, the Chief Secretary. Clause 39 provided for the appointment of a medical superintendent, 'who shall be a physician and surgeon, and shall be responsible for the treatment of the patients'.[74] The Bill failed on this very issue.

The final stage of parturition began at the Annual Meeting of the Association of Medical Officers of Asylums of Great Britain and Ireland held in Dublin on 22 August 1861. At this time all but one of the asylum managers in Ireland were now medical men. Dr Lawler, Resident Medical Officer at the Richmond, was President of the Association. Dr Stuart remarked that 'resident physicians have no feelings of hostility whatever to the visiting physicians; the resident physicians desire to have them still as colleagues, but in the character of consultants'.[71] Dr Flynn, Manager of the Clonmel Asylum, proposed that 'power should be given them to deal with the requirements of each case as his own judgement and experience may suggest, while the visiting physician is still retained as a consultant'. The motion was passed unanimously. A few weeks later a deputation waited on the Chief Secretary Sir Robert Peel.

The editor of the *Dublin Medical Press* protested strongly, 'An Act implementing such proposals could not for a moment be seriously entertained. The adoption of it would outrage our common sense'.[72] In spite of these protestations, on 16 January 1862 Peel carried revised rules for Irish asylums through the Irish Privy Council, cancelling those of 27 March 1843 and putting an end to divided responsibility. Rule 19 necessitated medical managers to be duly qualified as physician and surgeon. In spite of this, the struggle continued for another 30 years until the post of consulting physician was brought to an end in the early 1890s.

The medical profession has for centuries been one of the main providers for the relief of suffering of all kinds. Had such trust been based solely on the efficacy of our endeavours, then perhaps a very different state of affairs would have arisen. The parallels with the early care of the mentally ill are obvious. Daniel Tuke[73] during his presidential address to the Medico-Psychological Association in 1881 said, 'It may be difficult to suppress the hope, but we cannot entertain the expectation, that some future Sydenham will discover the antipsychoses which will as safely and speedily cut short an attack of mania or melancholia . . . Rather we must rest satisfied with the general advance in treatment in a scientific direction'. Given the advances in medical science, particularly in the fields of investigation and pharmacology, there can be no doubt of the advantages gained from the close links between those responsible for the care of the mentally ill and the rest of the medical profession.

Of equal importance have been the advances in our understanding of the roles of psychological and social mechanisms in precipitating, maintaining and compounding mental and physical ill health. Such issues rather than a prompt for the withdrawal of the medical profession from the care of the mentally ill surely point to the need for a balance in medical education, not only for those who choose the care of the mentally ill as their special interest, but for the profession as a whole.

THE GROWTH OF ASYLUM PROVISION

We turn to one final facet of provision for the mentally ill in the last century which would greatly influence the character of medical practice for more than a century. By the early 1840s the network of asylum provision, centrally organised and under the watchful eye of a medical inspectorate had been established for almost ten years. Throughout England and Wales there was no such network and the pattern of provision was extremely patchy. Wales at this time was without a single asylum, and over twenty counties in England were still without asylums, public or private. In 1843, Ireland's ten asylums provided inpatient facilities for two-and-a-half thousand patients — a residency rate of 30/100,000.[74]

Yet, in successive asylum reports of the 1840s[75, 76] we read of the 'urgent necessity for extension of accommodation. Accommodation is insufficient and a great evil arises from that deficiency'.[11] Within 15 years, asylum provision had more than doubled both in Ireland, and in England and Wales, yet the opinions of those concerned with planning and provision for the mentally ill remained unchanged.[77] Daniel Tuke, writing at the time, was apprehensive that the new round of difficulties being observed in Ireland would shortly be experienced in England.[11]

What had reversed the optimism regarding the original model provision consisting of several small 150-bed units, distributed throughout the country? The problem centred on the growing number of 'incurables' — the chronically mentally disabled. From the early statements on asylum provision it would appear that the reformers had intended to provide for all lunatic poor throughout Ireland. However, it is equally clear that the providers envisaged these new hospitals capable only of meeting the needs of the treatable and curable minority. As early as 1835, Jackson of the Armagh Asylum stated: 'We felt the serious inconvenience of being obliged to admit the chronic incurable cases and, I may say, have never been able finally to overcome the difficulty that the receiving of a large number from the gaols of such incurables caused on the first opening of the asylum . . . That these institutions are equal to meet all curable and violent cases I have no doubt'.[78] His estimate of need for acute care was 30/100,000, very close to current estimates of need.

Similar reports were emerging from the Belfast and Londonderry asylums.[79, 80] A clear discrepancy now existed between the objectives of the planners and those of the providers. The planners, however, were aware of the escalating costs which the grand juries of each county and district were obliged to provide. As a compromise, the newly-established workhouses were being encouraged to take the idiots and harmless lunatics. While this provided additional accommodation of a kind, the undesirability of such conditions was all too apparent to planners and providers alike. In the Lunacy Enquiry of 1843 Spring Rice considered the trend 'retrogressive'.[80] White, the Lunacy Inspector, described the workhouse accommodation as 'the very worst . . . none of these should be received anywhere but into district asylums'. Stuart, in the Belfast Asylum report of 1858, stated that 'fully two-thirds of patients in asylums are of the so-called incurable class — a class however requiring, for the most part, as much constant care and supervision as any other'.[84]

Why had there been such a growth in the number of people requiring chronic care, comprising two-thirds of residents of district asylums and the great majority of those in alternative accommodation? The early notion was of a real increase in

the prevalence of insanity. Later commentators have referred to the false optimism in the therapeutic effects of the moral methods. However, it is all too apparent from successive reports of enquiry over the first decades of the nineteenth century that there was no real appreciation of the size of the problem — that is, the problem in the community itself. Estimates of need were based wholly on the level of overcrowding in the asylums. Various references can be found throughout several lunacy enquiries to 'lunatics at large',[83] mentally ill persons living in the community. The only available estimates were those provided by Ireland's police force — hardly by today's standards a reliable source of prevalent estimates of psychiatric morbidity.

In 1850 Ireland was just emerging from the devastation of the Great Famine. Estimates of the insane were put at almost 10,000 or 150 per 100,000 population.[81] The number in the community was thought to be less than 5,000. Yet within 30 years the estimate of prevalence had apparently more than doubled to 350/100,000. The major determinant of the great expansion in asylum provision towards the end of the nineteenth century was the gradual recognition of the very large number of mentally ill people within the community itself, hidden from view like the base of an iceberg.

A second factor contributing to asylum expansion was the extreme general poverty. While in principle families were willing to take recovered relatives home, because of the poverty 'there was a great reluctance to do so'.[69, 84] Considerable pressure was placed on central government resulting in the building of more and more asylums to accommodate the chronically disabled lunatic poor who could not care for themselves in the community and who could not be readily cared for by relatives.

Throughout the nineteenth century, asylum population grew exponentially. By the early years of this century, provision in Ulster and throughout the rest of Ireland had risen by more than 20 times the original estimate of need[81, 85] (Fig 11). This unique explosion in institutional provision provides an explanation for the relatively high level of inpatient provision in Ulster today — still some 50 per cent higher than corresponding provision in England and Wales.

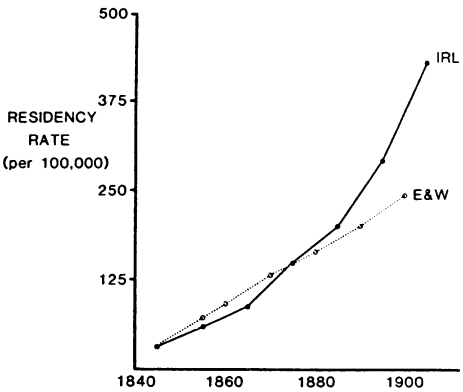

Fig 11. Growth of asylum provision.

EPILOGUE

Whatever criticism can be made of the resulting underfunded and understaffed institutional management, it has provided today's health planners with a firm estimate of the number of mentally disabled people in need of continuing care and support. Present institutional provision leaves us in no doubt of the costs of providing such care, a central ingredient for planning any change in the balance of provision back towards the community.

The 50 years beginning with the establishment of the Richmond Asylum and concluding with the creation of a network of district lunatic asylums staffed by a

professionally trained body can reasonably be regarded as the renaissance of the humane and enlightened care of the mentally ill in Ireland. In this process, the inspiration and dedication of individuals was critical. France had Esquirol and Pinel, England had William Tuke, Lord Shaftesbury and John Connolly. Ireland had John Newport, Thomas Spring Rice and a group of committed asylum managers and physicians among whom Ulstermen figured prominently: Thomas Hancock from Belfast, advocate of the non-restraint system; Alexander Jackson from Tyrone who established these methods of treatment in Ireland's first public asylum; Thomas Jackson at St Luke's, Armagh, who extended the principles to the district asylums; and Robert Stewart who 150 years ago began his career as Ireland's first psychiatrist, setting a fine example as a physician, a man of compassion, and an able manager of this city's embryonic mental health services.

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Health service performance indicators

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SUMMARY

The development of statistical indicators for monitoring performance throughout the National Health Service began in 1981. In recent years, computer software packages containing performance indicators have been amended to include Health and Social Services Boards in Northern Ireland. Although this information is available to anyone with an interest in service provision, the main purpose of the indicator packages is to provide a framework and discipline for ensuring proper accountability to a higher tier authority.

Efficiency and effectiveness are concepts central to performance appraisal. Most performance indicators are concerned with the input of resources or treatment activity rather than health outcome. Consequently the context within which the accountability review procedure operates is predetermined towards efficiency. The validity of the review process is limited by the accuracy of the indicators and the manner in which they are interpreted. The scale at which performance indicators function and the constraints associated with their use as an analytic tool are illustrated using data from a mental illness hospital in Northern Ireland.

INTRODUCTION

Accountability for spending public funds is fundamental to our democracy. In periods of economic recession, fiscal restraint is coupled with a concern for efficiency and a greater degree of parliamentary scrutiny. Recently, a great deal of attention has been directed at obtaining full value for money in the Health Service. Potentially the demand for health care is unlimited, but unfortunately resources are not. The constraint on new monies means that improvements in health care must be financed by savings made elsewhere in the Health Service. Consequently it is of paramount importance to define, measure and improve the performance of health service organisations.

This movement towards greater efficiency has coincided with the advent of micro-computers. The capacity of micro-computers to process large amounts of data, combined with their low cost, has provided statisticians with a useful instrument for interactive analysis. Management information can be readily presented in an easily accessible and visually attractive manner.

THE ACCOUNTABILITY REVIEW PROCEDURE

Today the high cost of developing new medicines and medical equipment, greater longevity among patients with serious illnesses, and high public expectations, have created a heavy demand for resources that has coincided with

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a fixed budget.[1] This has generated a need for good information to enable choices to be made between competing resource demands.

The accountability review procedure originated from the 1981 Report of the House of Commons Public Accounts Committee which called for more stringent monitoring of expenditure in the National Health Service.[2] Performance indicators are central to this procedure which is applied annually to Health and Social Services Boards in Northern Ireland. Review meetings are attended by a minister, senior civil servants, the Board chairman and his supporting officers.

Preparation for the review involves analysis of strategic plans and outturn reports to establish regional objectives and priorities for the provision of health care. The performance of services targeted for review is evaluated using statistical indicators. These highlight exceptionally poor or good performance by comparing the standard of service provision with that achieved by others. This allows an agenda outlining the main issues for discussion to be formulated. The purpose of the review is to agree upon specific policies and standards and to decide upon a programme for implementation and development in the year ahead.

EFFICIENCY AND EFFECTIVENESS

The present Government's commitment to obtaining value for money in health care provision, is expressed in a determination to obtain greater efficiency and effectiveness. These terms are defined as follows:

1. Efficiency is the ratio of output to resource input, and the aim is to improve output for the same or a lower cost.
2. Effectiveness refers to the extent to which the objectives of a policy programme or treatment are achieved and is concerned with medical outcome.

In theory, measurement of performance should be based on effectiveness in the first instance. Once it has been ascertained that a service is having the desired effect, for example eradicating smallpox by vaccination, then effort can be directed at improving the efficiency of the service. Unfortunately developing measures to determine whether health care objectives have been achieved is difficult because a clear relationship does not exist between resource input and outcome in the form of improved health or reduced need.[3] The health statistics available at present provide a great deal of information on inputs and activity, but virtually no data are available on outcomes. Consequently the accountability review process relies on indicators that focus on efficiency rather than effectiveness.

Efforts to curtail costs and boost productivity are made in the absence of knowledge about their impact upon the general health and well-being of the population. Simply increasing input or activity does not automatically guarantee better results, in fact the opposite may be true.

The seriousness of the situation has been succintly summarised by Culyer who recognises that: 'The lack of acceptable outcome measures to define need, and measure the extent of our success in meeting it, is chronic and a major impediment both to research and policy formulation in health services.'[4] A start must be made on measuring outcome.

PERFORMANCE INDICATORS

A performance indicator is a salient piece of statistical information that is sensitive to real variations in performance. Indicators can be used by anybody with an interest or involvement in service delivery, planning, the allocation of resources or the monitoring of performance. During the accountability review process performance indicators are used to show what has been achieved, to identify services which require improvement, and to help administrators and professionals decide if action is appropriate. Performance indicators are the starting point in the investigation, they enable the Department of Health and Social Services to question the Health and Social Services Boards. As such they must be relevant, accurate, consistent and readily available. Indicators do not measure performance; they simply point to performance which may require attention. Only a detailed systematic investigation can prove that resources are being used ineffectively or inefficiently.

Performance indicators are calculated from data that have been available for many years. They can be classified as indicators of input, activity/process, or outcome:

1. Input indicators:- invariably these refer to available resources in the form of staff, finance, equipment or buildings. Input indicators are commonly used when no others are available, but tell little about performance on their own. Examples are the ratio of part-time to full-time staff expressed in whole time equivalents or overtime costs as a percentage of total salary costs.
2. Activity/process indicators:- are normally defined in terms of patient treatment or care. They are readily available for hospital services although not for community services. An example is the ratio of nursing staff to average daily occupied beds.
3. Outcome indicators:- attempt to assess health improvements for patients or the result of medical interventions on the population. The number of vaccination payments to general medical practitioners per head of population is an example of an outcome indicator based on medical intervention.

Performance indicators were developed to allow clinicians and managers to compare their service with that achieved by others or over a period of time. Relative standards are set by ranking indicator values for individual regions, districts or hospitals against the national distribution. This helps to identify services with values lying in the extremities of the distribution. Conceptually and statistically this procedure for pinpointing exceptional performance is straightforward, it is also robust to data inaccuracies.

When statistical information is collated from a variety of sources it is inevitable that values for some data items will be inaccurate or incomplete, no matter how stringent the validation process. However, a gross error would be required to significantly alter the position of a district or hospital on a national distribution. A single indicator looks at performance in a narrow perspective. Like other statistics it may be directly or indirectly affected by factors outside its scope. Consequently groups of interrelated indicators need to be examined to identify fully all the facets of a problem. Examining indicators in groups rather than singly also helps circumvent the problem of inaccurate values.

PERFORMANCE INDICATOR SOFTWARE PACKAGES

Two computer software packages have been developed to display performance indicators in a visually attractive, informative and easily accessible manner:

1. The Department of Health and Social Security package contains approximately 425 indicators covering eight client or service groups including acute services, children, the elderly, mental illness, mental handicap, support services, estate management and manpower. Information on 14 Regional and 191 District Health Authorities in England is available from 1983 onwards. An abridged Northern Ireland version of the package containing 1985 data was issued for training purposes in 1987. Since then, complete Northern Ireland editions have been published for 1986 and 1987. Comparisons between health authorities are facilitated by box plots, histograms, outlier reports and tables of ranks and values.
2. The Inter-Authority Comparisons and Consultancy (Yates) package provides a range of indicators on patient flow, waiting lists, in-patient treatment, day case treatment and out-patients for 34 specialties. Histograms and profiles enable comparisons to be made across 16 Regional Health Authorities (including Northern Ireland and Wales) and 216 districts. Mental handicap and mental illness specialties have an additional subset of 20 indicators which allow performance to be compared between hospitals and over a period of time. 'District' information on Northern Ireland is available from 1983 to 1986. Inter-hospital indicators for mental handicap and mental illness hospitals in Northern Ireland cover the periods 1979–1986 and 1977–1986 respectively.

HOW TO APPLY AND INTERPRET PERFORMANCE INDICATORS

Literally hundreds of performance indicators are available for analysis using the Department of Health and Social Security and Yates software packages. Although an individual performance indicator cannot be used as a definitive measure of performance it is possible to monitor achievement using groups of crude indicators. These provide the starting point for an investigation. Performance indicators give an overview of the service under review and make problem areas easier to identify. In essence they generate questions about performance which can only be answered correctly by referring to local knowledge and more detailed data sources.

Yates and Vickerstaff[5] and Yates[6] have identified six crude indicators which are sensitive to performance variations in mental handicap and mental illness hospitals. Using data from the 1985 and 1986 Yates packages, the indicators can be applied to mental illness hospitals in Northern Ireland to illustrate the process of interpreting performance. The indicators are:

1. Size of hospital (measured by total number of in-patients):- some large hospitals can be more impersonal than smaller ones and this may make communication between patients and staff difficult. Large hospitals have traditionally served wide catchment areas and this can present problems for integrating patients into the community.
2. Percentage of patients over 65:- a large number of elderly patients increase nurse workload. If a substantial proportion of patients in this age group cannot be rehabilitated this may place high demands on staff morale.

3. Patients per consultant:- if support staff provision is inadequate a consultant may experience difficulty in supervising his patients as this ratio rises.
4. Patients per nurse:- as the number of patients attended by a nurse increases, the level of nursing care may be reduced.
5. Patients per therapist (includes occupational therapists, physiotherapists, speech therapists, remedial gymnasts, PE instructors, industrial work therapists and handicrafts staff):- a high ratio suggests that the rehabilitative services provided are insufficient.
6. Length of stay:- exceptionally long average stays for curable diseases may indicate that some patients are becoming 'institutionalised' and that success in rehabilitation is low. This may be combined with little short-term care provision.

The patients per nurse ratio is a suitable indicator for illustrating the performance analysis process. The impact of changes in nurse staffing on the overall level of treatment and attention in a unit are readily understood and appreciated. Patient/nurse ratio values for all mental illness hospitals in England, Wales and Northern Ireland in 1985 can be displayed in a histogram (Fig 1). From this, the position of hospitals in Northern Ireland can be examined against the national distribution. Two hospitals (Holywell and Purdysburn) have a value greater than or equal to 1·4 and lie to the right of the distribution.* It is of interest to see how these hospitals perform on the other five key indicators listed above.

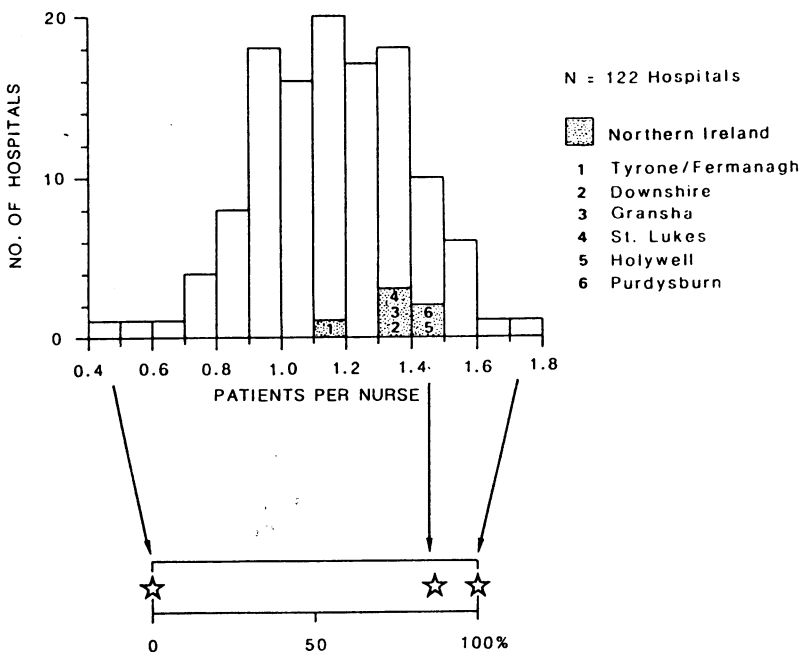
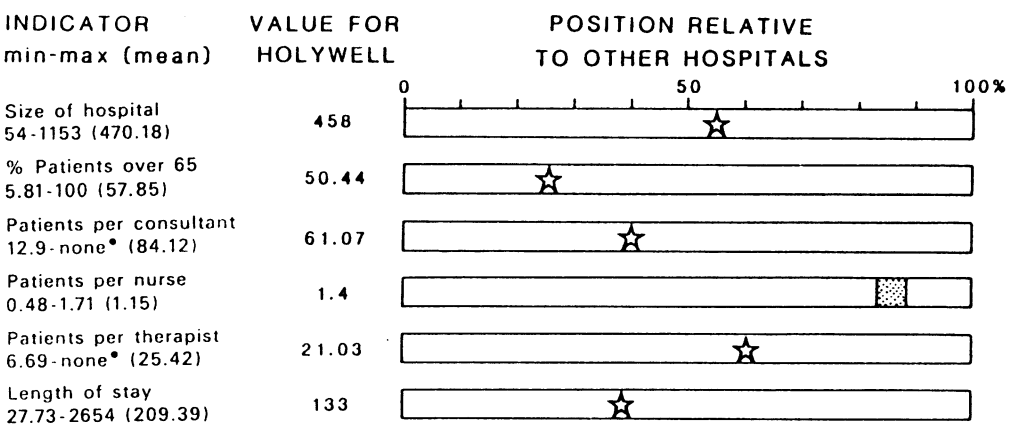


Fig 1. Histogram of patients per nurse ratio for 122 mental illness hospitals, 1985. The local distribution is shown above in comparison to the national distribution. This is converted to a percentage bar in the lower section.

* The 1985 data set is used for illustration purposes only. In 1986 the patients-per-nurse ratio for Holywell and Purdysburn was 1·25 and 1·28 respectively. This removes both hospitals from the critical area at the right hand side of the national distribution.

Unfortunately a histogram only portrays one indicator at a time. To enable simultaneous comparison of several indicators for one hospital each separate histogram is converted into a percentile bar by ranking all valid values in ascending order. A percentage rank is then calculated by taking the rank value of each hospital as a percentage of the total number of hospitals with valid values. This fixes the position of a hospital relative to all other hospitals, and compresses the distribution by eliminating the influence of values lying in the tails of the distribution.

A number of percentile bars can be displayed at one time in a diagram known as a 'profile'. Fig 2 is a multi-indicator profile for Holywell Hospital. At a glance it can be seen that this unit performs well on all the key indicators except nurse staffing. A shaded square on the percentile bar draws attention to performance which should be investigated. This symbol denotes that the value for the indicator lies in an extreme portion of the distribution of all values, arbitrarily this is fixed at 15% of the distribution. With staffing ratios it is only necessary to emphasise bad staffing and the 15% critical area is located on the right hand side of the distribution.



It is important to indicate that some hospitals in the distribution do not have consultants or therapists. Mathematically it is impossible to divide by zero. Rather than remove these hospitals from the analysis the value 'none' is used to denote infinity. They are assigned an exceptionally large fictitious value which forces them to the extreme right hand side of the distribution.

Fig 2. Multi-indicator profile for Holywell Hospital, 1985.

Before accepting the patient/nurse ratio value an administrator or clinician in this hospital should initially question the accuracy of the indicator, for example:

1. A crude ratio does not show the number of trained nursing staff or the mix of nursing skills.
2. Dependency levels vary among patients and if staffing has been adjusted to accommodate this the indicator will give a false impression.
3. Non-ward nursing workload cannot be distinguished from total nurse workload using this ratio.

Assuming that the limitations associated with the indicator have been considered and that they still do not explain the poor staffing level, then a second set of questions are generated, for example:

1. A high ratio may result from too many patients rather than under-staffing. Are rehabilitative services adequate?
2. Does the lower staffing reflect a large number of low dependency patients in this hospital?
3. Or is staff recruitment difficult because there is a large proportion of high dependency patients?

At this point it is also of interest to know whether poor nurse staffing occurred in one year only. Fig 3 is a multi-year profile which shows the position of Holywell relative to all other hospitals in England, Wales and Northern Ireland over a ten year interval from 1977 to 1986. In absolute terms the patient/nurse ratio has improved from 2·2 to 1·25 during this period. However, almost all mental illness hospitals in Britain have improved staffing over the last decade. Compared with other hospitals nationally, a relative improvement in the performance of Holywell occurred between 1980 and 1983 and again in 1986. How was this achieved?

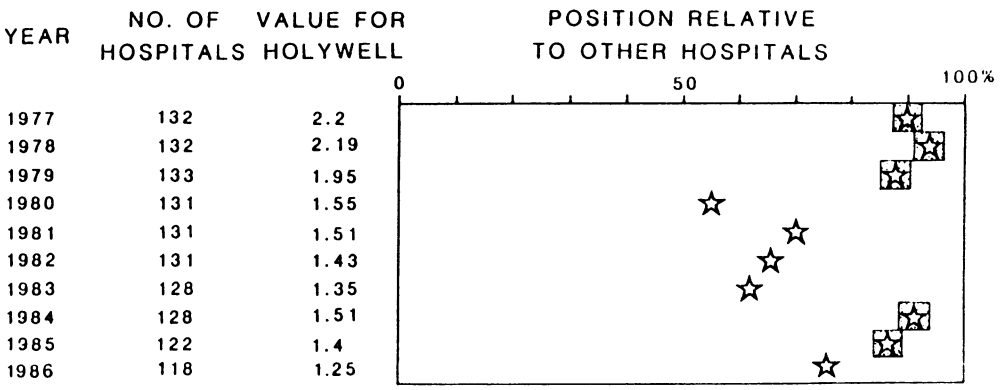


Fig 3. Patients per nurse multi-year profile for Holywell Hospital, 1986.

The problem of nurse staffing at Holywell Hospital has been identified but not answered by the analysis of performance indicators. Indicators simply facilitate systematic investigation. Spatial and temporal analysis of value distributions must be interpreted in a local context by administrators and professionals with an awareness of the policies, priorities and problems operating on the ground. As the first tier in the investigative process, performance indicators function at a level of generalisation which may fail to identify certain specific types of problem. However, exceptionally poor performance will virtually always be represented at this scale and can be detected.

CONCLUSION

Ultimately the continuing development and application of performance indicators should enable health authorities to set their own standards. This will entail a movement away from merely observing the position of a hospital, district or region on a national distribution, towards measuring achievement in reaching specified objectives. Implicitly this requires accurate indicators of health outcome. However, very often the effects of health care provision cannot be distinguished from social change or economic factors. Therefore in the future it may be necessary to restrict performance monitoring to the Health Service itself, rather

than to attempt to measure the result of interventions on the health of the population.

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The Northern Ireland Cancer Registry

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SUMMARY

The Northern Ireland Cancer Registry was evaluated using a 5% sample of all cancers diagnosed histologically in 1983 as the standard for comparison. The overall registration rate was low. Two years following histological diagnosis only 63% of the cancers were registered and 19% of these were notified solely by the Registrar General's office. In a subgroup of patients who were known to have died by the time of the study, only 49% of the cancers were registered while the patient was alive. A further 30% of cases were registered only after death and 21% of cases went unregistered. There was no significant variation in registrations by area, by hospital or by age group. There was a considerable variation in registration rate by disease group. A low level of awareness among hospital doctors about the Northern Ireland Cancer Registry was postulated as a reason for the low levels of registrations received. This was investigated through a postal questionnaire. A response rate of 51% was achieved after two postings. Both the response rate and level of knowledge varied by grade and specialty of the doctor. Only 43% of responders knew of the existence of the cancer registry and only 2% registered patients more often than once a year. Possible methods for improving the system are discussed.

INTRODUCTION

Cancer is an important disease. In developed countries it is second only to heart disease as the most commonly registered cause of death. In Northern Ireland, approximately 3,000 individuals die annually from cancer and this accounts for one-sixth of all deaths. The local Hospital Activity Analysis records cancer as the reason for almost 8% of hospital admissions. The total cost of cancer to the Health Service and society is enormous but unquantified. In our daily lives we are brought into increasing contact with carcinogens and so an accurate data base is essential to identify, monitor and ultimately reduce exposure to these hazards. The evaluation of interventions such as screening and health education requires accurate data. Adequate information is also required by those responsible for planning health services.

Mortality data reflect cancer prevalence less and less well as cancers are diagnosed earlier and treated more effectively than in the past. Cancer registration schemes, by collecting information on all cancers diagnosed in an area, provide a method of monitoring cancer incidence and prevalence within a population. Cancer has been registered in Northern Ireland since 1959. The

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registry is located in a civil service department at Castle Buildings, Stormont and receives registrations from hospital doctors who complete cards for their cancer patients, and from the Registrar General's office which provides a list of registered cancer deaths.

Information on approximately 80,000 cases has been gathered by the registry, but until now neither the completeness of registration nor the accuracy of the data have been evaluated. It is staffed by 1·5 whole time equivalent clerical officers with minimal input from senior civil servants. It does not have its own computer. It costs about £25,000 per annum and produces only one document, a bulletin which does not analyse or interpret the data gathered.

METHODS

In 1985 an evaluation of the registry was performed using cancers diagnosed histologically during 1983 as the standard for comparison. This was a valid standard, as it represented the most complete and accurate source of cancer cases and there was no linkage between the records of the histology laboratories and those of the cancer registry.

A 5% random sample, stratified by laboratory, was selected (n = 358) from all cancers diagnosed in 1983. Identification details and information about the cancer were extracted by the author from laboratory records and hospital notes. The cancer registry files were then manually searched to ascertain whether the patient had been registered by 31 July 1985 and, if so, whether this occurred during life or solely from the Registrar General's returns after death. The accuracy of the recorded diagnosis was also assessed.

Since, in N. Ireland, doctors are the main source of registrations it was decided to investigate the level of awareness about the registry among hospital doctors. This was investigated by a postal questionnaire sent to each hospital doctor of registrar grade and above, who might diagnose or treat cancer patients. The questionnaire was designed to obtain information on the respondent's grade, specialty, knowledge of the cancer registry, the method by which such knowledge was gained, the frequency of registration by the respondent and whether the registry could be of use to them in their work or research. A space was left for comments. The questionnaire was posted along with an information sheet about the registry, a registration card and a prepaid return envelope. All non-responders were remailed after ten weeks had elapsed. Analysis of the questionnaires was performed using the SPSS computer package on a minicomputer. The exercise was evaluated by comparing the number of registrations received by the registry before and after the questionnaires were distributed. The levels of registration for 1986 were also compared to previous years.

RESULTS

The sample of 358 cancer cases contained 52% males and 48% females. Overall 225, (63%) of patients were registered, 44, (12%) of the sample were registered solely from the Registrar General's returns, accounting for 19% of total registrations. Inaccuracies in the recorded site of tumour was noted in less than 1% (2) of registered cases.

Analysis by area of residence showed that 7·4% of the sample lived in the Western Health and Social Services Board, 23·6% in the Northern Board, 18·5% in the Southern Board and 50·4% in the Eastern Board. The Western Board was significantly under-represented in the sample ($p < 0·01$), The age distribution showed most cases in the older age groups, in keeping with the

epidemiology of cancer. In each age group the proportion of cases registered with the cancer register was similar.

The data were analysed by hospital of diagnosis. Only three hospitals contributed 50 or more patients to the sample: the Belfast City Hospital had 69% of its cancer patients registered with 3% of these registered solely from death certification. The respective percentages for the Royal Victoria Hospital were 61% and 7%, while for the Ulster Hospital, Dundonald, they were 56% and 6% (Table I).

TABLE I

Measure of registration by source hospital (hospital of initial attendance)

Hospital	*Absolute frequency in sample*	*Percentage of sample registered with cancer registry prior to death*	*Percentage of sample registered with cancer registry only by Registrar General's returns*	*Percentage of sample not registered*
Royal Victoria	72	54%	7%	39%
Belfast City	74	66%	3%	31%
Ulster	50	50%	6%	44%
Craigavon Area	24	54%	8%	38%
Forster Green	10	70%	10%	20%
Waveney	12	33%	25%	42%
Mid Ulster	11	55%	9%	36%
Whiteabbey	14	64%	0%	36%
Altnagelvin	11	55%	9%	36%
Others	80	47%	15%	38%

There was considerable variation in registration by site of disease. Over half (54%) of skin malignancies, 82% of cancers of the cervix (more severe than CIN III), nearly half (46%) of the myelomas and half (50%) of the leukaemias went unregistered. Of the disease groups with sufficient cases to allow reliable comparisons, lymphomas, lung and breast malignancies had the highest percentage registered before death (Table II).

The completeness of registration did not vary significantly between the four health boards: the Eastern Board had the highest registration (66%), while the Western Board had the lowest (58%). The contribution of the Registrar General's returns to the total registrations was assessed. This was the only source of registration in 44 cases, (19%) of all registered cases. Of the total sample of 358 cases, 128 (36%) were known to be dead, 24 (6·7%) were known to be alive and in 206, the status was unknown. Four-fifths (79%) of those known to be dead were registered. Only 49% were registered during life and 30% were registered only after death via the Registrar General's office. Of the 24 persons known to be alive, 54% were registered by the cancer registry (Table III). Therefore, there was a failure of registration in 21% of the subgroup of patients who were known to be dead and 37% of all cancer patients.

TABLE II

Distribution of registration by disease groups

Diagnosis Malignancies	*Absolute frequency in sample*	*Percentage of sample registered with cancer registry prior to death*	*Percentage of sample registered with cancer registry only by Registrar General's returns*	*Percentage of sample not registered*
Lung (ICD 162)	20	75%	5%	20%
Gastrointestinal tract (ICD 140–159)	80	50%	24%	26%
Skin (ICD 172–173)	67	46%	0%	54%
Breast (ICD 174)	42	76%	2%	22%
Lymphomas including Hodgkins (ICD 200–202)	14	86%	0%	14%
Cervix (ICD 180)	17	18%	0%	82%
Lip, oral cavity, vocal cord, larynx and nasopharynx (ICD 140–149 and 161)	18	67%	0%	33%
Bladder (ICD 188)	25	64%	8%	28%
Prostate (ICD 185)	18	50%	11%	39%
Leukaemia and myelofibroais (ICD 204–208)	18	33%	17%	50%
Ovary and vagina (ICD 179, 181–184)	12	50%	8%	42%
Myeloma (ICD 203)	11	55%	0%	46%
Others (thyroid, testis, kidney, brain secondary malignancies)	16	50%	6%	44%

TABLE III

Performance of cancer registry for live and deceased persons

	Alive on 1 January 1985	*Deceased by 1 January 1985*	*Total sample (2 years following histological diagnosis)*
Registered during life	13 (54%)	62 (49%)	182 (51%)
Registered only from death certificates	—	39 (30%)	44 (12%)
Not registered	11 (46%)	27 (21%)	132 (37%)
Total	24	128	358

A cross-check was made between the sample and death certificates for 1983 which mentioned cancer, to identify those cases which were wrongly classified as alive by the cancer registry. Of the 128 persons known to be dead, 10 (8%) were incorrectly classified as alive by the registry; three of these errors were the result of doctors failing to complete death certificates accurately so that cancer was not recorded as a cause of death.

The total number of questionnaires posted to hospital doctors was 803. The initial response rate was 38%, which increased to 51% on remailing. The response rate varied between specialties as follows: general medicine 59%, obstetrics and gynaecology 48%, surgery 45%, psychiatry 54%, accident and emergency 53% and anaesthetics 40%. These differences were statistically significant ($x^2 = 14{\cdot}98$, 3df, $0{\cdot}05 > p > 0{\cdot}01$).

The response rates also varied significantly by grade of staff:- associate specialists 32%, registrars 41%, senior registrars 58%, consultants 53% ($x^2 = 22{\cdot}3$, 3df, $p < 0{\cdot}001$).

The level of knowledge about the cancer registry among hospital doctors was low. Only 175 responders (43%) were aware of the registry, of these, 84% stated they had learned about it from a letter or circular, 10% from a colleague and less than 1% from a lecture. Knowledge of the cancer registry varied with the timing of response to the questionnaire: of the 304 initial responders 36% knew of the registry while the figure for the 101 subsequent responders was 64%. Knowledge of the registry also varied significantly by grade of staff. Of those who responded, only 14% of registrars, 10% of senior registrars and 24% of associate specialists knew of it, whereas 60% of consultants were aware of its presence ($x^2 = 82{\cdot}8$, 3df, $p < 0{\cdot}001$). (Fig 1).

There were also significant differences in the levels of knowledge among the various specialties. The highest level was in those working in obstetrics and

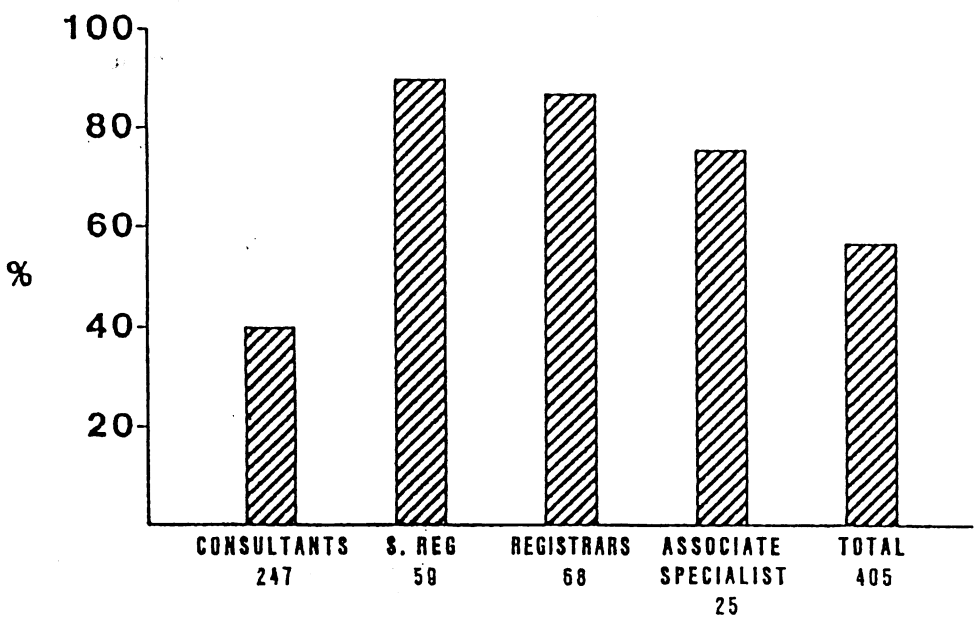

Fig 1. Percentage of responders who were unaware of the cancer registry (by grade of staff)

gynaecology (69%). Only 30% of psychiatrists, 41% of surgeons and 40% of those working in medical specialties were aware of the cancer registry ($x^2 = 22{\cdot}3$, 3df, $p < 0{\cdot}001$). (Fig 2).

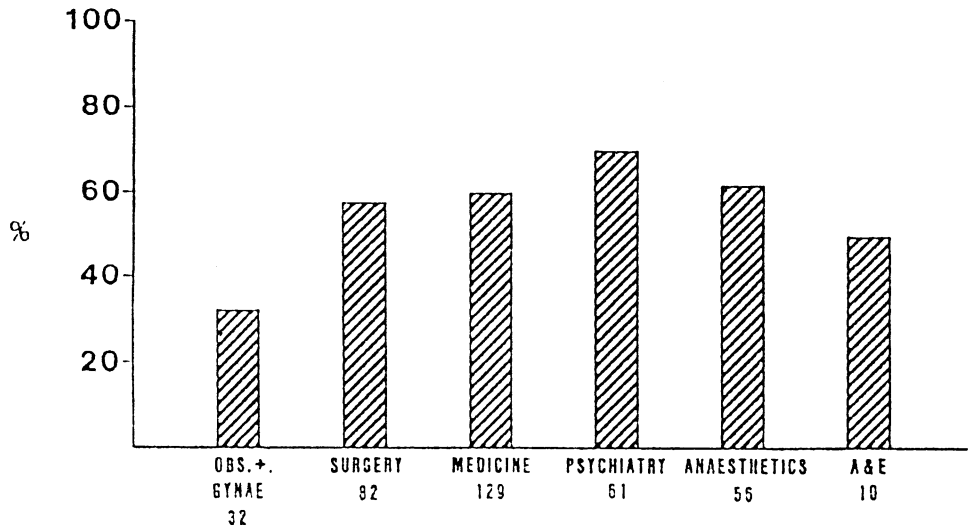

Fig 2. Percentage of responders who were unaware of the cancer registry by specialty

Responders were asked how often they registered cancer patients. Over two-thirds (69%) replied that they had never registered a cancer patient, 11% registered cases occasionally, while only 2% registered cases more often than once a year. Over half (51%) of responders thought the registry could be useful in their work or research, while 32% did not.

Responders were asked who they thought should be responsible for submitting registrations. The majority (53%) thought consultants should register cancer patients, 21% thought clerical staff should perform the task, while 28% thought it should be the responsibility of junior medical staff (in a few cases all three options were chosen and so the total is greater than 100%).

The majority of responders added at least one comment to the questionnaire. Many were surprised to discover that the registry existed and that it was dependent on their co-operation (Table IV). In addition to the popular comments, a few felt the registry should be closed. Many felt it should be reorganised and in particular, that it should be computerised to include information from histology laboratories. A few complained about the potential extra work that cancer registration would involve. Following the distribution of the questionnaire the number of registrations increased transiently, but this rise was not sustained.

DISCUSSION

The routine recording of basic information on cancer patients is intended to provide information on the frequency of different types of cancer and on survival following diagnosis and treatment. It also may alert researchers to environmental hazards. However, the value of a cancer registry is highly dependent on the accuracy and completeness of the recorded data. If a population-based registry is

TABLE IV

Responders' comments

1. Education: increased and widespread education of medical staff about the cancer registry is required.
2. Registration card design: should request more information.
3. Registration card availability: problems in obtaining cards at ward level.
4. Responsibility: many different opinions were expressed about who should actually register patients. A fee per registration was advocated by a few.
5. Feedback: an annual report containing data interpretation in addition to tables of registration was suggested.
6. Organisation: it is difficult to obtain access to the data contained in the registry.
7. Cost: the value for money was questioned.
8. Relevance: some questioned the value of a cancer registry to clinical medicine.

to be of real epidemiological value it should aim to register 90% of all malignant disease occurring within the area it serves.[1] Incompleteness of ascertainment of new cases leads to an underestimation of the incidence rate and may result in false assumptions about trends.[2]

The level of completeness of registrations (63%) found in this study is low when judged by these criteria, and by comparison with other cancer registries which have evaluated their performance and published results. In north-west England, registration completeness was found to be 94% (ranging from cervix, 81% to ovary, 98·5%).[3] Using morbidity data in Scotland, Haddow[4] showed that completeness of registration ranged from 40% to 90% between areas. The relative contribution of total registrations derived solely from death certificates in Northern Ireland was calculated as 19%, which is higher than the figure of 7·7% calculated by Nwene[3] for north-west England and reflects the generally low levels of registrations by doctors.

It was reassuring to find there was no variation in registration by geographical area. Such variations, if present, could lead to false impressions about disease occurrence. Unlike Benn et al[5] this study did not find a significant variation in the completeness of registration for different age groups. The response rate to the questionnaire was low (51%) and may reflect the level of interest among some specialists about cancer registration. This would account for the variation in response rate by specialty. In view of the fact that hospital doctors represent the main source of registration, it is hardly surprising that the registry performs poorly. There are over 7,000 cancers diagnosed histologically in Northern Ireland each year, yet the registry receives details on under 5,000 cases annually.

The means by which responders became aware of the cancer registry will be of interest to those planning to further the education of doctors in this area. Almost all of those who were aware of the Northern Ireland Cancer Registry had learned about it by letter (84%). This method of education about the cancer registry could be pursued but not in isolation; doctors need to know the benefits of cancer registration before they register cases. The fact that very few junior staff but over half of consultants knew of the registry suggests that there has been little or no education about its existence for some years.

There was a strange discrepancy between the small number of doctors who registered cases and the large number (over half) who felt the registry could be of use to them in their work or research. There is not only a general lack of awareness about the registry but also an ignorance among doctors concerning their role in registering cancer patients. This was borne out by the variations in responses to the question about who should register such cases. Each group of staff appeared to be under the impression that some other group was registering the cancer cases. There was not a sustained increase in cancer registrations over the months following this exercise, perhaps because of issues other than levels of knowledge, for example availability of registration cards or poor perception of the value of cancer registration.

The poor registration percentages in general, and for some disease groups in particular, casts doubt on the value of the registry as currently organised. A cancer registry is a valuable tool for researchers in general, and epidemiologists in particular, and so efforts should be made to increase the level and accuracy of registrations. Ultimately this may involve the automatic registration of patients by histology laboratories, but in the meantime the system could be vastly improved if all doctors who diagnose or treat cancer patients register the details. Apart from its use as a research tool, the registry is essential for monitoring cancer incidence. A convincing demonstration of the need for adequate surveillance has been 'the sustained and genuine public concern over the discharge of radioactive waste from Sellafield into the Irish Sea'.[6] The Chernobyl disaster further strengthens the case for improving cancer registration. This work has quantified the shortcomings in the Northern Ireland Cancer Registry and this awareness has already spurred those concerned about the problem to propose an improved system.

For the success of an improved cancer registry, it should use multiple sources of information including hospital doctors, general practitioners, diagnostic facilities including pathology, neuropathology, haematology and immunological laboratories, radiology departments, terminal care hospitals and the Registrar General's office. It should have sufficient resources, including adequate staff numbers and mix to allow analysis and interpretation of data received and engagement in research projects. We look forward to the realisation of such a system in Northern Ireland.

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Wegener's granulomatosis and autoantibodies to neutrophil antigens

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SUMMARY

We report five cases of Wegener's granulomatosis all of whom had clinical and histological evidence of disease activity at presentation and in whom autoantibodies to neutrophil antigens were detected. This test may prove useful for the diagnosis of this serious condition and help to monitor disease activity during treatment.

INTRODUCTION

Wegener's granulomatosis is a necrotising granulomatous arteritis[1] which preferentially involves the kidney and upper and lower respiratory tracts.[2,3] Its aetiology is unknown and its pathogenesis not entirely characterised.[4] Despite the lack of understanding of the immune mechanisms involved in the disease, it can be successfully treated with immunosuppressive agents.[2,5] Diagnosis is usually based on clinical and histological data.[2,5,6] There are a variety of clinical manifestations which include proteinuria, microscopic haematuria, impaired renal function, fever without evidence of infection, ocular inflammation, otitis, sinusitis, tracheitis, haemoptysis, pulmonary infiltrates and evidence of granulomatous vasculitis usually on nasal, lung or renal biopsy.

A number of non-specific immunological abnormalities have been reported including circulating immune complexes,[7,8] rheumatoid factor and IgG and IgA autoantibodies. These have not proved helpful in diagnosis or monitoring disease activity and are present in relatively few patients. Laboratory monitoring of disease activity has relied on non-specific markers such as the erythrocyte sedimentation rate and C-reactive protein.[9]

Van der Woude et al reported the presence of IgG autoantibodies to neutrophil antigens in patients with active Wegener's granulomatosis.[10] More recently Lockwood et al have described a radioimmunoassay method to quantitate the level of these autoantibodies and have shown that they are detectable in certain types of vasculitis including Wegener's granulomatosis.[16]

PATIENTS AND METHODS

Five patients (three female, two male) were studied. Serum samples were tested for the presence of antineutrophil antibodies by the indirect immunofluorescent

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and radioimmunoassay methods described by Lockwood et al.[16] In all patients the diagnosis of Wegener's granulomatosis was confirmed by histological examination of tissue biopsy and the pre-treatment serum samples showed the presence of antineutrophil antibodies (Table).

TABLE

Case	*Age*	*Sex*	*Neutrophil autoantibody*	*Renal impairment*	*Positive biopsy*
1	53	F	+ve	No	Nasal
2	28	M	+ve	No	Lung Nasal
3	41	M	+ve	Yes	Nasal Renal
4	33	F	+ve	Yes	Nasal Skin
5	62	F	+ve	Yes	Skin (Postmortem)

CASE 1

A 53-year-old woman presented with epistaxis, nasal congestion, deafness and a painful swollen left calf. On examination, her temperature was 39°C. She had rhinitis and otitis media. There were signs of consolidation at the apex of the left lung. Venography confirmed the clinical impression of an extensive left leg venous thrombosis. A chest X-ray demonstrated cavitating lesions in both lung fields. A nasal biopsy was reported as showing necrotising arteritis. Urinalysis and microscopy were negative. Treatment with 80mg prednisolone and 2mg/kg cyclophosphamide daily induced clinical remission. Pre-treatment serum samples showed the presence of antineutrophil antibodies both by indirect immunofluorescence and radioimmunoassay. Unfortunately, she died three months after diagnosis from a cerebrovascular accident.

CASE 2

A 28-year-old man presented with earache, deafness, nasal congestion, epistaxis, cough and pleuritic chest pain. On examination, his temperature was 39·2°C. He had episcleritis, rhinitis and a serous effusion in the right middle ear. Signs of consolidation were present in the left lung apex. A chest X-ray revealed the presence of circular opacities in both lung fields. Biopsy of the nasal septum and percutaneous needle biopsy of lung confirmed the diagnosis. Serum samples showed the presence of antibodies to neutrophil antigens. Treatment with 60mg prednisolone and 2mg/kg cyclophosphamide induced clinical remission. Repeat serum samples showed no evidence of antineutrophil antibodies either by indirect immunofluorescence or the radioimmunoassay.

CASE 3

A 41-year-old man presented with facial pain, recurrent epistaxis and jaundice. On examination, his temperature was 38·5°C. Apart from jaundice, he had no other stigmata of liver disease. Biochemical investigations included bilirubin

102 umol/l, alkaline phosphatase 45 U/l, gamma GT 771 U/l, ALT 1295 U/l and AST 365 U/l. Viral hepatitis, leptospiral and toxiplasma screening was negative. Initial urinalysis was negative except for bilirubinuria. Facial X-rays showed opacification of both maxillary antra which were subsequently drained. He developed right hypochondrial pain two weeks after admission and ultrasound scan and percutaneous cholangiogram showed a dilated gallbladder and dilated common hepatic duct. He developed pleuritic chest pain, cough and haemophysis and on examination there was a left-sided pleural rub. Investigations revealed microscopic haematuria, pulmonary infiltrates on chest X-ray and multiple perfusion defects on isotope lung scan. He developed rapidly progressive origuric renal failure. Renal biopsy demonstrated crescentic glomerulonephritis. He was treated with 60 mg prednisolone and 2 mg/kg cyclophosphamide daily but there was no recovery of renal function despite remission of other clinical features. He was subsequently maintained on twice weekly haemodialysis until receiving a cadaveric renal allograft in November 1985. Repeat assay for antineutrophil antibodies are consistently negative.

CASE 4

A 33-year-old woman presented with general malaise and weight loss for three months. She developed arthralgia, skin rash, cough, haemoptysis and sinusitus in the two weeks prior to admission. On examination her temperature was 37·8°C. She had tender nodular erythematous swellings on the right ankle and left wrist and a florid haemorrhagic rash. Investigations revealed rapidly progressive renal failure with proteinuria and haematuria on urinalysis and haemogranular casts and microscopic haematuria on microscopy. A chest X-ray showed pulmonary infiltrates. Nasal biopsy was consistent with the diagnosis. Treatment with 60 mg prednisolone and 2 mg/kg cyclophosphamide induced clinical remission with recovery of renal function. Significant proteinuria 2–5 g/24 hours persists at follow up.

CASE 5

A 62-year-old woman presented with a three week history of a flu-like illness, cough and sputum. On examination, her temperature was 38·4°C. She was anaemic and in congestive cardiac failure. Consolidation was present at the left lung base. Investigations revealed rapidly progressive renal failure. A skin biopsy was reported as vasculitis. Renal arteriography was suggestive of multiple micro-aneurysms. Peritoneal dialysis was instituted but was complicated by peritonitis. She died from cardiac arrest during haemodialysis. Post mortem demonstrated a myocardial infarct secondary to haemorrhage into an atheromatous plaque occluding the right coronary artery. The diagnosis was confirmed by the finding of necrotising granulomatous arteries in lungs and spleen. The renal lesion was crescentic glomerulonephritis.

DISCUSSION

Wegener's granulomatosis is a serious and life-threatening condition in which a rapid and accurate diagnosis may improve the clinical outcome.[11] Untreated, the one year survival in patients with renal disease is 20 per cent.[2, 5] It can be effectively treated with immunosuppressive drugs, usually a combination of steroids and cyclophosphamide.[12, 13, 14] Azathioprine and plasma exchange may also have a role in treatment.[15]

Autoantibodies against neutrophils and monocytes in Wegener's granulomatosis have been described by Van der Woude et al[10] and were reported as being specific for this disease. Lockwood et al have confirmed the presence of autoantibodies to neutrophil antigens in this condition, but have shown that they may also be detected in other forms of vasculitis.[16] We report five patients with clinical and histopathological evidence of Wegener's granulomatosis in whom neutrophil autoantibodies were present at time of diagnosis and in whom the antibody test became negative when clinical remission was achieved. Testing for neutrophil autoantibodies may soon become part of the screening investigations in vasculitic disorders and testing for these immunoglobulins may be of benefit when biopsy material is inconclusive or unobtainable. Rapid serological diagnosis would enable earlier treatment to be initiated in an attempt to reduce overall morbidity and mortality of this disease. This assay would also appear to be a more reliable and sensitive method for monitoring disease activity than the non-specific markers such as ESR or C-reactive protein.

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Acoustic neuroma surgery in Northern Ireland 1976 – 1986

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SUMMARY

Forty acoustic neuromas have been removed surgically between 1976 and 1986. The condition was unilateral in 32 and bilateral in four. There were 31 large, four medium and five small tumours. Excision was complete in 16 and incomplete in 24. Of the incomplete removals 14 were subtotal leaving microscopic remnants, eight were partial capsular and two were intracapsular. Follow-up ranged from two months to ten years (median 3·5 years).

There was one early death in an 83-year-old. The overall incidence of post-operative complete facial paralysis was 20% but reached 55% for large tumours when excision was complete. Twenty-eight patients had hearing before operation and in eleven patients some preservation of hearing was possible (39%). In these, the excision was complete in three, subtotal in four, partial capsular in three and intracapsular in one.

Of the unilateral tumours, there have been three recurrences requiring repeat surgery. All were initially incompletely excised. Two were of an invasive nature causing considerable erosion of the petrous temporal bone making complete excision impossible. For the bilateral tumours a deliberate incomplete excision was first performed on one side to ensure preservation of hearing. Further excision on this side was then left until such time as hearing was lost. Complications included CSF otorhinorrhoea (5%), persistent but temporary nausea and vomiting (10%), meningitis (5%), facial numbness (5%) and hoarseness and dysphagia (3%).

INTRODUCTION

The most common tumour arising in the eighth cranial nerve is usually termed an acoustic neuroma. Because of tradition we shall continue to use this term but it is important to state at the outset that it is doubly inaccurate — the tumour most commonly arises from the vestibular nerve, and is a schwannoma.

The first successful operation on an acoustic neuroma was performed in 1894 by Sir Charles Ballance.[1] In those days the mortality rate for total tumour removal was very high. At the 1913 International Congress of Medicine, the mortality rates of three eminent European surgeons — Horsley of London, Eiselsberg of Vienna and Krause of Berlin — ranged from 67% – 84%. These results prompted

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Cushing[2] to perform partial removal, with a reduction in operative mortality to 20%. However, although the mortality was reduced, one fourth of the surviving patients died within five years as a result of recurrence or revision surgery. In 1925 Dandy[3] using the suboccipital approach reported an operative mortality rate of 40% for total tumour resection. The survivors invariably had permanent complete postoperative facial paralysis.

In 1964, House[4] introduced a microsurgical translabyrinthine technique for the removal of acoustic neuromas with a considerable reduction in both mortality and morbidity. He reported an operative mortality of 5·4% and in the survivors a 94% preservation of facial function. He also suggested the development of a team approach to the removal of acoustic tumours, with co-operation between otologists and neurosurgeons. Rand and Kurze[5] in 1965 were the first to treat acoustic tumours by microsurgery using the suboccipital route.

Until the last two decades most patients had little or no residual auditory function by the time of diagnosis. Because of improved methods of investigation and diagnosis this now is frequently not the case and a further challenge is to preserve residual cochlear function while accomplishing the main goals of surgical excision and preservation of facial function.

PATIENTS AND METHODS

Between 1976 and 1986, 48 acoustic neuromas were treated surgically in the Royal Victoria Hospital, Belfast. This report analyses the results obtained in 40 of these tumours under the care of an individual neurosurgeon and two otologists. All of the procedures were carried out through a suboccipital craniectomy as described by Smyth et al.[6]

There were 32 patients with unilateral and 4 with bilateral tumours. Two of the patients with bilateral tumours were brothers; their mother had a unilateral tumour, and a sister had bilateral tumours but these latter two do not form part of this report. All four of this family had von Recklinghausen's neurofibromatosis. The tumour was on the right side in 19, and the left side in 21 cases. There were 22 males and 14 females. Age ranged from 11–83 years (median 44 years).

Twenty-nine patients presented with unilateral deafness (Table I). The first symptom was tinnitus in three, vertigo in two and progressive bilateral deafness in

TABLE I

Symptoms and signs of acoustic neuroma on presentation in 40 cases

Symptoms	*No*	*Signs*	*No*
Deafness	39	Ataxia — Gait	17
Tinnitus	26	Arm	6
Unsteadiness of gait	16	Reduced corneal reflex	14
Facial numbness/paraesthesia	8	Nystagmus	7
Headache	5	Facial weakness	5
Vertigo	4	Papilloedema	3
Facial pain	3	Vocal cord paresis	1
Hemifacial spasm	1		
Vomiting	1		
Hoarseness	1		
Dysphagia	1		

two. Where vertigo was the first symptom, one patient had had a totally deaf ear on the side of the tumour following mastoid surgery 20 years previously and the other had a tumour which arose medial to the internal auditory meatus and reached but did not enter the meatus.

There are three stages of clinical progression. In Stage I only the eighth nerve is involved, with deafness and tinnitus being the only symptoms. In Stage II other neurological manifestations are present, usually cerebellar ataxia and trigeminal nerve involvement. Patients in Stage III have raised intracranial pressure and papilloedema. There were 31 large ($>2{\cdot}5$cm diameter), four medium ($1{\cdot}0-2{\cdot}5$cm) and five small ($<1{\cdot}0$cm) tumours. Small and medium tumours all presented in Stage I, apart from one patient with bilateral tumours, as did 40% of the large tumours (Table II). The duration of symptoms ranged from a few months to 25 years but there was no correlation between size of tumour and duration of symptoms.

TABLE II

Tumour size and clinical stage at presentation, and completeness of surgical excision

Tumour size	Tumour Stage			Total	Excision	
	I	*II*	*III*		*Complete*	*Incomplete*
Small	4	1	—	5	4	1
Medium	3	1	—	4	3	1
Large	12	16	3	31	9	22

Tumour excision was complete in 16 and incomplete in 24 cases. Of the incomplete removals 14 were subtotal, eight were partial capsular and two were intracapsular. Subtotal removal ranged from a microscopic plaque of tumour capsule left attached to the facial nerve or brainstem to a cuff of tumour remaining extending from the porus acousticus to the brainstem surrounding the facial and vestibulocochlear nerves. The reasons for incomplete excision included the patient's age or debility, fluctuating vital signs during surgery, attempts to preserve facial or cochlear function, or adherence of the tumour to the brainstem. In the four patients with bilateral tumours, a deliberate decision was taken before operation to perform an incomplete excision in all the operations on the first side and in two of the four second side operations, thereby attempting to ensure some preservation of hearing for as long as possible. Varying degrees of cerebellar hemisphere excision were required in 13 tumours, 12 large and one medium sized, to improve exposure and reduce retraction. The duration of follow-up ranged from two months to ten years (median 3·5 years).

RESULTS

Mortality

There was one death during hospitalization, on the 12th postoperative day. This occurred in an 83-year-old woman who had a large tumour which necessitated surgery and was incompletely removed. Her initial postoperative course was satisfactory but she later developed a basal pneumonia and died.

Facial nerve function one year postoperatively

In the nine small and medium sized tumours facial nerve function was preserved fully in eight (89%) and partially in one (11%). In the 31 large tumours facial function was preserved fully in 19 (61%), partially in four (13%) and there was a permanent and complete facial paralysis in eight (26%). (Table III).

TABLE III

The influence of tumour size and completeness of surgical excision on facial nerve function postoperatively. The result was considered good when function was normal or near normal, and fair when weakness and asymmetry were marked

		Facial nerve function			
Size	*Surgical excision*	*Good*	*Fair*	*None*	*Total*
Large	Complete	2	2	5	9
	Incomplete	17	2	3	22
Medium	Complete	2	1	0	3
	Incomplete	1	0	0	1
Small	Complete	4	0	0	4
	Incomplete	1	0	0	1
Total		27	5	8	40

Large tumours were completely excised in nine patients, of whom five (56%) had a complete facial paralysis; in three of these the nerve was known to have been divided or damaged during the operation. Excision was incomplete in large tumours in 22 patients of whom only three (14%) had a complete facial paralysis. Of these 22 patients there were 13 in whom excision was subtotal and of these only one (8%) had a complete facial paralysis. The overall incidence of complete facial paralysis was 20%.

Cochlear nerve function following surgery

Twenty-eight patients had varying degrees of hearing before operation. Of these, 17 (61%) had no recordable hearing postoperatively; the cochlear nerve was seen to be divided in seven. Some hearing conservation was therefore achieved in 11 patients (39%). (Table IV). Of these, excision was complete in three, subtotal in four, partial capsular in three and intracapsular in one. Of the completely excised tumours two were large and one was medium sized. In two of these three the hearing preservation has been maintained at two and five years. However, the remaining patient developed meningitis three weeks postoperatively and there was a gradual decline in his hearing threshold so that at seven years there was no recordable hearing. Of the 13 large tumours that have been subtotally excised, prolonged preservation of cochlear function has been possible in three.

If a speech discrimination of 50% is considered to be essential for useful hearing only three out of the 40 ears still have useful hearing and of these only one had a complete excision.

TABLE IV

Cochlear nerve function following surgery: average pure tone hearing is recorded in decibels at 500–4000 Hz and speech discrimination is recorded as a percentage of normal

		Hearing				
		Preoperative		Early Postoperative		
Size	*Surgical excision*	*Pure tone hearing*	*Speech discrimination*	*Pure tone hearing*	*Speech discrimination*	*Follow-up*
Small	Subtotal	65	76%	50	88%	Gradual decline (repeat surgery at 3 years)
Medium	Complete	30	84%	65	72%	Meningitis (gradual decline to nil)
Medium	Partial capsule	50	80%	45	80%	Gradual decline to nil (repeat surgery at 3 years)
Large	Complete	20	52%	45	84%	2 years
Large	Complete	30	92%	60	24%	5 years
Large	Subtotal	55	92%	70	24%	4 years
Large	Subtotal	65	72%	60	no record	1 year
Large	Subtotal	85	nil	95	nil	4 years
Large	Partial capsule	30	96%	35	96%	1 year
Large	Partial capsule	45	80%	60	88%	4 years
Large	Intracapsular	65	72%	65	76%	Gradual decline to nil

Other complications

Balance was impaired in 16 patients before operation. Only two remained significantly handicapped by balance problems beyond one year postoperatively. Cerebrospinal fluid otorhinorrhoea occurred in two patients both of whom required a second operation. Persistent nausea and vomiting occurred post-operatively in two patients lasting from several days to four weeks; all eventually settled. Other complications consisted of two cases of meningitis, two of facial pain, two of facial numbness and one of hoarseness and dysphagia.

To date there have been six recurrences requiring repeat surgery. Three were patients with bilateral tumours where the initial surgery was deliberate incomplete excision to preserve hearing, in the knowledge that further surgery would be required; repeat surgery was performed three to nine years later. The other three cases were patients with large tumours. One was initially incompletely excised and required repeat surgery after three years. The other two were unusually invasive with extensive erosion of the petrous bone, making complete excision impossible even at repeat surgery.

DISCUSSION

The risk of death following excision of an acoustic neuroma remains low. The incidence for this series was 2·5%. The size of the tumour tends to determine the difficulty of the operation and the postoperative morbidity and mortality. One of the four patients with bilateral tumours subsequently developed neurofibromata elsewhere in the central nervous system and died from that cause.

Facial nerve preservation is excellent after removal of small and medium sized tumours. In an attempt to reduce the morbidity associated with removal of large tumours, subtotal excision was performed in 42%. Where the tumour was large, the incidence of complete facial paralysis was 56% with complete excision and 8% with subtotal excision.

Although the cochlear nerve can be isolated and spared during tumour removal, the blood supply of the tumour and the cochlear nerve is intertwined making preservation of hearing more difficult than preservation of facial function. Of those patients in whom there was residual hearing before operation, some hearing conservation has been possible in 39%. Paradoxically, for the five small tumours hearing preservation has been possible in only one (20%), whereas of the 19 large tumours with preoperative recordable hearing, preservation of hearing was possible in eight (42%). Although Sterkers[7] noted that postoperative facial function was always normal when hearing was preserved, we did not find this to be the case. Of the three cases where excision was complete and cochlear function was preserved, two had a permanent and complete facial paralysis.

One of the advantages of the translabyrinthine technique claimed by King and Morrison[8] is that it avoids damage to the cerebellum which they believe is responsible for ataxia after the posterior fossa approach. However, in this series only two patients who did not have preoperative ataxia were unsteady after the operation and both recovered within a few months. Thirteen patients had excision of the lateral third of the cerebellar hemisphere; this did not appear to cause postoperative ataxia.

Cerebrospinal fluid otorhinorrhoea appears to be a relatively common post-operative complication in other series. King and Morrison[8] experienced a 14% incidence following translabyrinthine removal and Harner and Ebersold[9] a 12%

following the suboccipital approach; we noted an incidence of only 5%. One case occurred as a result of incomplete waxing of exposed mastoid air cells and the other because of the presence of a posteromedial air cell tract in the posterior wall of the internal auditory meatus.[10]

To date the incidence of clinical recurrence requiring repeat surgery is 15%. These are largely limited to the deliberately incomplete operations on bilateral tumours and the invasive tumours where there is considerable erosion of the petrous temporal bone. Cross[11] has suggested that subtotal removal might cause regression of the tumour, perhaps from interruption of its blood supply. Silverstein et al[12] have advocated subtotal excision chiefly for elderly patients with large tumours thereby enabling them to live the remainder of their lives without distressing neurological symptoms. In this series, of the 14 patients who had an initial subtotal excision, two (14%) have required a further operation because of clinical recurrence, the duration of follow-up being from 1·2–8 years.

The management of patients with bilateral tumours presents formidable difficulties because of the risk of total deafness, loss of labyrinthine function and bilateral facial paralysis. Of the four patients in this series with bilateral tumours, three were diagnosed as being bilateral at first presentation. In these patients, all of whom had residual bilateral hearing, our approach was to perform a deliberate incomplete excision on one side, concentrating on decompression of the internal auditory meatus, where compression ischaemia of the cochlear nerve probably takes place. Most of the tumour was removed leaving a bridge of tumour covering the seventh and eighth cranial nerves between the internal auditory meatus and brainstem. Any further excision was then left until such time as hearing was lost. With successful preservation of hearing on the first side, a complete excision was then performed on the opposite side in one patient and an incomplete excision in two. Following the initial incomplete operation on the first ear with preservation of the hearing, all experienced a gradual and progressive decline in the hearing over a period of three to five years, reaching a stage where there was no residual hearing on that side. Repeat surgery has since been performed in two. So far we have managed to preserve some hearing in one ear in two out of the four patients with bilateral tumours. Facial function was preserved fully following the initial operation on both sides in all patients, but repeat surgery in one has resulted in a complete unilateral facial paralysis.

The mortality rate for acoustic neuroma removal using the suboccipital approach remains low. For small and medium sized tumours preservation of facial function is excellent; for large tumours when completely excised there is still a high incidence of complete facial paralysis. The likelihood of hearing preservation remains low when excision is complete. Subtotal excision has reduced the morbidity associated with surgery and the incidence of clinical recurrence has been low.

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Short- and long-term academic predictors of medical student performance

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SUMMARY

The relationship between entry qualifications and performance throughout the medical course was studied in individual cohorts of students admitted in the years 1977 to 1981. A modest but significant relationship with overall initial qualifications was found throughout the course. Students without biology showed a marked disadvantage in the first year but the effect did not persist. There were quite marked variations in all these effects between cohorts; studying only a single cohort could lead to inappropriate conclusions.

INTRODUCTION

While it is accepted that prior academic achievement should have a role in the selection of medical students,[1] the importance of this role and the use of other possible predictors, such as interviews, have been sources of doubt and controversy.[2–6] It has been suggested that biology is a particularly good predictor[3] but there may be an increasing tendency to accept candidates without this subject at A-level.[7] McManus and Richards[8] have suggested that entrants without A-level biology are particularly at risk and that further investigation is needed. We have quantified the predictive value of performance at the Advanced level of the General Certificate of Education for performance in examinations throughout the whole medical course for three cohorts of students. Correlations have been made with overall performance, and in addition students without Advanced level biology have been compared with those who had taken the subject.

METHODS

The students studied were admitted to the Belfast medical school in the period 1977–1981 inclusive. In selected years all students were included who had entered on the basis of the Advanced level of the Northern Ireland General Certificate of Education (the vast majority), entering directly the first medical year to study anatomy, biochemistry and physiology without taking a premedical year. In 1977 and 1978 substantial numbers took the premedical year, which was being phased out, thereafter virtually none took it. Progress was followed

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throughout the five year course, professional exams being taken after four terms (anatomy, biochemistry, physiology), nine terms (microbiology, pathology, therapeutics) and fifteen terms (medicine plus surgery (combined), midwifery and gynaecology). The tables, which compare performance throughout the course, are based on students who completed the course, omitting those (around 10%) who left before completion.

During the period of study, entry requirements were a minimum of three Advanced level subjects, of which chemistry was obligatory; the other two subjects were almost invariably selected from biology, mathematics and physics.

Statistical associations between entry qualifications and subsequent performance were investigated by calculating correlation coefficients and by comparing groups using t-tests. Two main associations were explored. Firstly the effect of overall entry qualifications; an "A" grade in a subject was allocated five points, a "B" four points and so on. These were summed to give a "total entry score". Most candidates presented three or four subjects; a candidate with three 'A' grades would get 15 points, with four 'A' grades 20 points. This total entry score was correlated with performance in each subject of the professional examinations and the performance of students with three (or more) "A" gradings was compared with that of the rest of the class. Secondly, the effect of the presence or absence of Advanced level biology was studied by comparing the performance of those without biology with that of the majority who presented with biology.

RESULTS

The mean total entry score for students with and without biology is shown on a yearly basis in the Figure. The number of direct entrants to the first medical year rose between 1977 and 1979 as the premedical year was phased out. In 1977 and 1978 the best students were selected for direct entry so their total entry scores were relatively high; values fell from 17·5: 17·4 (with: without biology) in 1977 to 15·4: 15·3 in 1979, after which they levelled off. Those without biology comprised some 30% of the total during the period, and were indistinguishable from the rest in terms of total entry score.

Table I shows correlation coefficients between total entry score and marks obtained in professional examinations. Two main trends can be seen. Firstly the size and significance of the

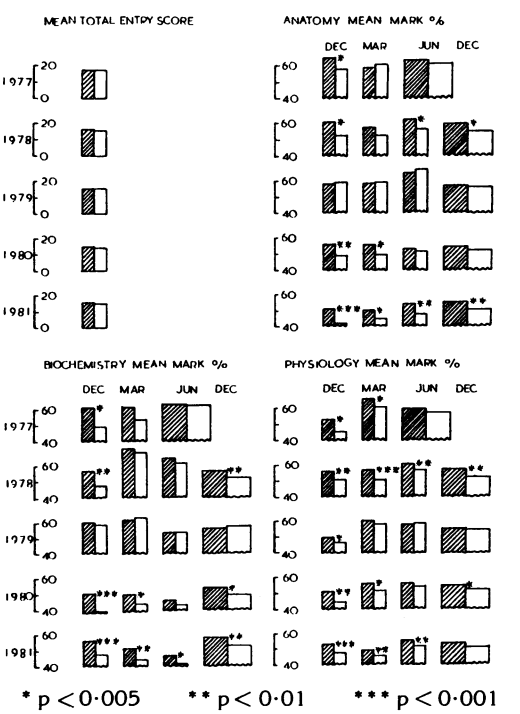

Figure. Students with biology (hatched columns) compared with those without (open columns) with respect to total entry score [see text] and performance in class tests (narrow columns) and professional examination (broad columns). The professional examination was moved from June to December in 1978.

correlations varied widely from year to year in all subjects. Secondly the correlation coefficients were rather higher with the subjects of the first professional examination than with subjects taken later. The highest coefficient was 0·370, in physiology in 1981. However, positive and in some cases significant correlations persisted throughout the course.

TABLE I

Correlation coefficients between total entry scores and marks obtained in professional examinations

Year of entry (number of students)	1977 (52)	1979 (143)	1981 (140)
Anatomy	−0·060	0·196*	0·320***
Biochemistry	0·201	0·284***	0·336***
Physiology	0·237	0·304***	0·370***
Microbiology	0·320*	0·033	0·142
Pathology	0·008	0·151	0·135
Therapeutics	0·318*	0·099	0·080
Medicine & Surgery	0·131	0·087	0·153
Midwifery & Gynaecology	0·114	0·210*	0·145

* $p < 0·05$ ** $p < 0·01$ *** $p < 0·001$

Some explanation for the lesser correlations may lie in the nature of the subjects. Table II shows that between subjects, correlation coefficients decline throughout the medical course. At the first professional examination six of the nine correlation coefficients were above 0·7; at the next examination only two values were above 0·7 and at the final examination no coefficient was above 0·5 (all coefficients at the first examination were above 0·5).

TABLE II

Correlation coefficients between subjects within professional examinations

Year of entry (number of students)	1977 (52)	1979 (143)	1981 (140)
Anatomy/Biochemistry	0·577***	0·611***	0·798***
Anatomy/Physiology	0·661***	0·768***	0·766***
Biochemistry/Physiology	0·800***	0·729***	0·766***
Microbiology/Pathology	0·529***	0·666***	0·678***
Microbiology/Therapeutics	0·499***	0·742***	0·545***
Pathology/Therapeutics	0·553***	0·710***	0·614***
Medicine & Surgery/ Midwifery & Gynaecology	0·229	0·450***	0·428***

*** $p < 0·001$

Dividing each year of students into those with three or more "A" gradings and those with less than 3 "A" gradings (Table III) showed that the former group averaged higher marks in every subject. Fluctuations from year to year were rather less. Significant differences persisted throughout the course, but the differences again were less at later examinations.

TABLE III

Comparison of students with three or more "A" gradings on entry (3 A's) with the rest, showing mean marks obtained in professional examinations

Year of entry	1977		1979		1981	
	3 A's	rest	3 A's	rest	3 A's	rest
(number of students)	36	16	40	106	36	104
Anatomy	65·1	58·6	61·9	55·4***	59·3	52·6***
Biochemistry	65·0	58·8*	62·4	54·4***	64·5	54·3***
Physiology	60·8	57·3*	58·6	54·3***	57·5	51·2***
Microbiology	65·4	61·4*	56·3	55·9	57·3	54·6*
Pathology	56·2	54·5	56·4	53·4*	53·9	51·8*
Therapeutics	58·8	55·3*	56·0	54·3	58·6	56·7
Medicine & Surgery	117·9	115·1	116·8	113·6*	116·1	113·1
Midwifery & Gynaecology	53·5	52·8	54·5	51·6**	54·3	53·4

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

The effects of the presence or absence of biology was most marked in the first year of study. At the first class test, differences were often of the order of 10 percentage marks in favour of those with biology. Differences were smaller and less frequently significant at the professional examination. Again the pattern varied from year to year, differences between the groups being much less for 1979 entrants. Table IV shows the comparison for all professional examinations. Two significant differences (in favour of those with biology) were present in the first professional examination, but after this only one significant difference was found (in favour of those without).

TABLE IV

Comparison of students with and without biology, showing mean marks obtained in professional examinations

Year of entry	1977		1979		1981	
	with biology	without biology	with biology	without biology	with biology	without biology
numbers	41	11	104	41	103	37
Anatomy	63·6	60·9	57·0	56·4	55·5	50·8**
Biochemistry	63·1	62·7	56·4	57·3	57·9	52·8**
Physiology	60·1	57·7	55·6	55·2	53·3	51·4
Microbiology	64·5	62·4	56·1	55·8	55·5	55·2
Pathology	56·1	53·9	54·5	54·4	52·3	52·4
Therapeutics	57·9	57·6	54·6	55·1	57·2	57·5
Medicine & Surgery	117·4	115·6	114·1	115·6	113·7	114·3
Midwifery & Gynaecology	53·2	52·3	51·8	53·7*	53·8	53·1

* $p < 0.05$ ** $p < 0.01$

DISCUSSION

This study agrees with others[3, 4, 7–11] which have found a relationship between entrance qualifications and subsequent performance. A previous study of three cohorts (1968, 1970, 1972) of Belfast students[11] showed correlations of a similar order to the present study. As in the previous study there was quite marked variation from year to year. For example, the 1968 cohort showed no significant correlations between entry qualifications and the professional physiology examination, while the 1970 cohort showed the highest correlation registered, 0·61. Again midwifery and gynaecology showed a significant correlation, 0·26, for the 1970 cohort but not for the 1968 and 1972 cohorts. The other studies quoted in the introduction did not examine these correlations on a year by year basis, so it is not possible to confirm whether marked year to year fluctuations occurred. A hint of similar fluctuations is given in another study from Belfast[4] which reported on a single year and studied males and females. This found that entry scores correlated similarly for males and females with performance in anatomy, biochemistry and physiology, whereas in six other subjects the 72 males showed a significant correlation and the 46 females did not.

Since most correlation coefficients in all studies quoted were around 0·3 to 0·4 it must be assumed that other factors accounted for 80–90% of the variance in these students, who by entrance selection constitute a group of fairly uniform academic standard. It is not surprising that these other factors should from time to time swamp the influence of entrance qualifications.

It has been shown in this study that the influence of overall entrance qualifications tends to persist throughout the medical course, whereas the influence of the subject mix in the entrance qualifications is more ephemeral. Combining the previous Belfast study[11] with the present results, typical findings were that overall entrance qualifications correlated with professional examination results for anatomy in five of the six cohorts, for therapeutics in four of the cohorts, for medicine and surgery in four and for midwifery and gynaecology in two. The inter-subject correlations were also lower in later parts of the course.

A study in Cambridge[12] has shown significant but modest correlations of performance between preclinical and clinical performance and between undergraduate performance and subsequent career success, so the "chain of correlation" would seem to extend from admission qualifications to eventual career.

By contrast the influence of the presence or absence of Advanced level biology in the entrance qualifications was marked in the first year or so, but not after the professional examination in anatomy, biochemistry and physiology; thereafter only two subjects showed a significant effect, one in favour of those with biology, the other in favour of those without it. Again the year to year variation was seen, with the 1979 cohort in particular failing to show advantage for those with biology.

Bearing in mind the fairly narrow academic spectrum,[7] the finding of a modest but persistent effect of initial qualifications supports the role of entrance qualifications in selection. The finding of a marked initial effect of the absence of biology suggests that students are well advised to take this subject, but the ephemeral nature of the effect also supports the admission of otherwise well-qualified students who are prepared to overcome their initial disadvantage. The powerful initial effect of the presence or absence of the biology qualification is a tribute to the teaching and assessing of this subject at school level.

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Mobile coronary care and mortality from ischaemic heart disease in a predominantly rural community

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SUMMARY

The mobile coronary care unit based at Coleraine Hospital was called to 155 patients in the community during a six-monthly period, 74 of whom had sustained a myocardial infarction. Over the same period, 25 of 49 patients admitted via the ordinary ambulance with suspected ischaemic heart disease had sustained a myocardial infarction and received medical care significantly later than those seen by the mobile unit. A further 12 patients out of 39 with suspected ischaemic heart disease admitted by other means (the accident and emergency department or other hospital units) brought the total number of patients admitted to hospital with myocardial infarction during the study period to 111.

Overall mortality from myocardial infarction was 19·8% and was significantly higher in those ⩾70 years of age. Nine patients with myocardial infarction seen by the mobile coronary care unit required early defibrillation (four outside hospital) and eight of these survived to be discharged. No patients admitted by other means required emergency defibrillation. Although no significant difference in mortality was demonstrated between those seen before or after three hours from the onset of symptoms or between patients admitted by the mobile unit or by the ordinary ambulance, a subgroup of patients below 70 years of age and seen by the mobile unit less than three hours after the onset of symptoms had the lowest mortality of 6·7%. Estimated overall mortality from ischaemic heart disease in this community over the study period was in excess of those deaths accounted for in this survey, implying a high mortality in those not admitted to hospital.

INTRODUCTION

An "intensive care" ambulance first came into operation at Coleraine Hospital in June 1977. It was staffed by an ambulance driver and a trained intensive care nurse until June 1978 when medical staffing levels permitted a senior house officer to join the team. At present the ambulance covers a mixed urban-rural area of 400 square kilometres in north Antrim and northeast Londonderry, and serves a population of 86,500 people (42,600 males, 43,900 females). In the

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tenth year of its operation the present prospective survey was conducted over a six-month period to assess the use of the ambulance by this community and the effect of mobile coronary care on hospital survival following admission with ischaemic heart disease.

PATIENTS AND METHODS

All admissions between 1 October 1986 and 31 March 1987 for ischaemic heart disease (including myocardial infarction) were followed for the duration of their hospital stay. They were classified in three groups. 1. All patients seen in the community by the mobile coronary care unit. 2. All patients admitted directly to a general medical ward via an ordinary ambulance or from the accident and emergency or other hospital departments, with suspected ischaemic heart disease or myocardial infarction. 3. All in-patients subsequently proven to have sustained an unsuspected myocardial infarction irrespective of the source of admission or reason for referral.

A diagnosis of myocardial infarction was accepted when at least two of the following criteria were present: typical history of chest pain; characteristic and evolving electrocardiographic features; significant rises in cardiac enzymes in serial blood samples. For each patient the mode of admission was determined, the time from onset of symptoms to hospital conditions or arrival of the mobile unit, complications ensuing before and after two hours from the provision of hospital or mobile unit care, the length of hospital stay, outcome and the final diagnosis.

The mobile care unit is continuously on stand-by, and requests are transferred from ambulance control to the hospital telephone switchboard whilst the ambulance is being mobilised. The duty senior house officer and staff nurse are alerted and join the ambulance at the accident and emergency entrance. Calls are not usually vetted by medical staff. All patients transported by the mobile unit are admitted to the intensive care ward. Admissions by ordinary ambulance are arranged between the general practitioner and the house physician on duty, and these patients are brought to one of the general medical wards, where seven beds are equipped with a monitoring facility. Admissions from the accident and emergency unit are arranged with the duty senior house officer and can be directed either to intensive care or a general ward.

The time of receiving a call for the mobile unit and its arrival at the destination is recorded to the nearest minute and logged at ambulance control.

Data were analysed using Student's t- and chi-square tests. Statistical significance was accepted when $p < 0{\cdot}05$.

RESULTS

There were 243 patients in this study group over the six-month study period, of whom 155 were seen by the mobile coronary care unit in the community, 49 were admitted by ordinary ambulance, 33 via the accident and emergency department, and six from other sources (non-medical hospital wards and out-patient clinics).

Of the 155 patients seen by the mobile unit, 74 (47·7%) had sustained a myocardial infarct (56 male, 18 female, mean age 66·2 yrs). An additional 30 patients were considered to have acute coronary insufficiency and 10 had arrhythmias without infarction, three of whom had ventricular tachycardia at presentation (Table I). Only one patient refused admission to hospital.

TABLE I

Final diagnoses of 155 patients seen in the community by the mobile coronary care unit over a six-month period

Final diagnosis	*Number*
Myocardial infarction	74
Acute coronary insufficiency	30
Left ventricular failure:	
chronic cardiac ischaemia	6
aortic valve disease	2
cardiomyopathy	2
Dead on arrival	6
Resistant ventricular fibrillation	2
Arrhythmias:	
atrial fibrillation	5
ventricular tachycardia	3
supraventricular tachycardia	2
Syncope	4
No diagnosis at discharge	4
Cholecystitis	2
Musculoskeletal pain	2
Hiatus hernia	2
Asthma	2
Pulmonary embolus, intracerebral haemorrhage, duodenal ulcer hypertension, anaemia, anxiety neurosis, drunk	1 each

In 17 cases (11%) the mobile unit was requested by a lay person and in one case by an ambulance crew. In all other cases the patient was first seen and assessed by a general practitioner. The median time taken from call to arrival of the ambulance was 12·0 min (mean 16·2 ± 8·8 min). The median time from onset of symptoms to arrival of the ambulance was 2·0 hr (mean 3·0 ± 3·2 hr) for all patients and 2·0 hr (mean 3·1 ± 3·2 hr) for the 71 patients with myocardial infarction for whom time of onset of symptoms was available. Of these 71 patients, 20 (28%) were seen within one hour and 50 (70%) within three hours.

Of the 74 patients with myocardial infarction admitted by the mobile unit, nine required emergency defibrillation (ventricular tachycardia or fibrillation) within two hours of being seen in the community, mean 2·1 hr following onset of symptoms, mean age 64·3 yr. One was successfully defibrillated at home and three in transit. Eight of these nine patients survived to leave hospital. Three patients presenting with ventricular tachycardia without infarction were defibrillated at a mean of 1·3 hr following the onset of symptoms, and all survived to discharge. Two patients had refractory ventricular fibrillation when seen in the community, and a further six were dead on arrival of the mobile unit.

Of 49 patients admitted by ordinary ambulance, 25 (51%) had suffered a myocardial infarction (14 male, 11 female, mean age 70·4 yr). An additional 12 patients had acute coronary insufficiency and one had ventricular tachycardia without infarction. No patient with myocardial infarction admitted by the ordinary ambulance required subsequent emergency defibrillation. The median time from

onset of symptoms to arrival in hospital was 4·0 hr (mean 8·9 ± 8·4 hr) for all patients and 4·2 hr (mean 7·3 ± 6·5 hr) for those with myocardial infarction. This was a significantly longer time than that taken by myocardial infarction patients transported by the mobile coronary care unit ($p < 0·005$). Although patients with myocardial infarction transported by ordinary ambulance were slightly older than those transported by the mobile unit, this was not statistically significant.

Out of the further 39 patients admitted from the accident and emergency and other hospital departments, 12 had proven myocardial infarction and 15 had acute coronary insufficiency. The median time from onset of symptoms to presentation at hospital was 2·5 hr (mean 4·5 ± 4·9 hr) for all patients in this category and 2·0 hr (mean 4·5 ± 4·3 hr) for those with myocardial infarction.

A total of 111 patients (45·5% of the study group) had a definite myocardial infarct (78 male, 33 female, mean age 66·6 ± 12·3 yr). Sixty-seven percent were admitted by the mobile unit. Overall in-hospital mortality was 22 (19·8%). Mortality amongst mobile unit admissions with myocardial infarction was 20% (15/74 patients) and although lower than the 28% of ordinary ambulance admissions (7/25 patients), this difference was not statistically significant ($p > 0·25$).

For patients aged 70 years or more, mortality was significantly higher, 32% (16/50) compared to 9·8% (6/61) in those aged less than 70 yr ($p < 0·01$). When those admitted via the mobile coronary care unit were analysed separately, statistical significance was lost (Table II).

TABLE II

Distribution of 111 patients with myocardial infarction by age, and mode of admission. ($p < 0·01$)*

	Mobile coronary care unit		*Ordinary ambulance*		*Other admissions*		*Total*	
Age yr	No	Deaths (%)	No	Deaths (%)	No	Deaths (%)	No	Deaths (%)
<70	42	5 (11·9%)	10	1 (10%)	9	0 (0%)	61	6 (9·8%)*
⩾70	32	10 (31·3%)	15	6 (40%)	3	0 (0%)	50	16 (32%)*
All	74	15 (20·3%)	25	7 (28%)	12	0 (0%)	111	22 (19·8%)

Mortality amongst females with myocardial infarction was significantly higher than males ($p < 0·01$), 36% of females (12/33) died in hospital compared with 13% of males (10/78). Again statistical significance was lost if only admissions via the mobile coronary care unit were analysed. This higher female mortality doubtless reflects the greater mean age of the female patients (74 yr compared to 64 yr).

In 105 of 111 cases of proven myocardial infarction, the time from onset of symptoms to the provision of hospital care or arrival of the mobile unit was established to the nearest 15 minutes, allowing comparison of mortality between patients seen before and after three hours. Considering all 105 patients, 10/61 (16·4%) of those seen at three hours or less died, which is not significantly different from 7/44 (15·9%) of those seen after three hours. For patients seen by

the mobile unit considered separately, 9/50 (18%) of those seen at three hours or less died, not significantly less than 4/21 (19%) seen after three hours of the onset of symptoms (Table III). A subgroup of patients aged less than 70 yr seen by the mobile unit by three hours or less had a mortality of 6·7% (2/30 patients). Only two out of 10 patients aged less than 70 yr admitted by ordinary ambulance reached medical care within three hours, so no meaningful comparison is possible. For ordinary ambulance patients analysed separately, 1/6 (16·7%) died when admitted three hours or less after onset of symptoms, not significantly less than 3/16 (18·8%) admitted more than three hours after onset.

TABLE III

Distribution of 105 patients with myocardial infarction by time from onset of symptoms to medical attention, and mode of admission

	Mobile coronary care unit		*Ordinary ambulance*		*Other admissions*		*Total*	
Time hr	No	Deaths (%)	No	Deaths (%)	No	Deaths (%)	No	Deaths (%)
⩽3	50	9 (18%)	6	1 (16·7%)	5	0 (0%)	61	10 (16·4%)
>3	21	4 (19·1%)	16	3 (18·8%)	7	0 (0%)	44	7 (15·9%)
All	71	13 (18·3%)	22	4 (18·2%)	12	0 (0%)	105	17 (16%)

All five patients aged less than 70 and 9/10 patients over 70 who died from myocardial infarction following admission by the mobile unit, had called a general practitioner (93%). The mean length of hospital stay amongst survivors admitted by the mobile unit was 13·7 ± 5·6 days, not significantly shorter than 15·8 ± 4·8 days for all other myocardial infarction patients. There were no deaths amongst any patients with acute coronary insufficiency.

DISCUSSION

Since the introduction of mobile coronary care in Belfast in 1966,[1] several other units have been established in this province and worldwide.[2, 3, 4] Improved survival was clearly demonstrated for patients with myocardial infarction seen by a mobile unit within three hours of onset of symptoms.[5] Seventy percent of our patients receiving coronary care outside hospital did so within this time limit, the median being two hours. Our figures for "total delay" for both mobile coronary care and ordinary ambulances are comparable to other units serving a mixed urban-rural population in Northern Ireland.[3, 6]

The high mortality of our patients aged over 70 is an expected finding. The mortality of 11·9% for those aged less than 70 seen by the mobile unit is of the same order as that previously reported.[3] Although the reduction in hospital mortality in our mobile care group failed to reach statistical significance, we feel that this reflects the relatively higher numbers of elderly patients (with higher mortality) present in both groups, and the smaller number of patients admitted by ordinary ambulance overall. The reduction in mortality seen in patients admitted earlier than three hours from onset of symptoms was small, but again may be an effect of the relatively higher numbers of elderly in this study, since the mortality in the subgroup aged less than 70 and seen earlier than three hours was reassuringly low at 6·7%.

Using the mortality rates for ischaemic heart disease derived by McIlwaine et al[7] from a study in Belfast (4·4/1000 for males, 3·1/1000 for females), an estimated 160 deaths from ischaemic heart disease might have occurred in our community during the study. We accounted for 22 following hospital admission and at least another 2 outside hospital, leaving a possible 135 deaths in the community from ischaemic heart disease unaccounted for. Would it be possible to further reduce this apparently high mortality outside hospital? A high percentage of these deaths are caused by primary rhythm disturbance[7] and may be preventable with early defibrillation. One strategy which has been adopted for reducing early mortality in an urban-rural community operating mobile coronary care has been to provide defibrillators to all general practitioners in the region.[8] This might be reasonable in our community since 88% of our patients with myocardial infarction had called a general practitioner as first line of aid, and only 7% of our mobile unit admissions who subsequently died of ischaemic heart disease received mobile coronary care as the first line of aid. McIlwaine et al[7] reported that 26% of patients aged less than 70, dying in Belfast from ischaemic heart disease, summoned mobile coronary care as first line of aid. Other strategies which must be considered include the earlier administration of thrombolytic therapy,[9] and an attempt to reduce delay time between onset of symptoms and the provision of coronary care. Most delay is accounted for by the patient,[3, 10] and delays attributed to the general practitioner mediating between the cardiac patient and the arrival of coronary care range from 15 minutes in an urban environment[7] to 45 minutes in an urban-rural environment.[10] Reducing these delays involves both patient education and the facilitation of direct access of the public to the mobile coronary care unit, with medical staff vetting public calls. Although this would increase the workload of the coronary care unit, the facility would appear to be underused by the public at present.

We thank Dr D G Sinnamon and Dr P Gilmore for permission to study their patients.

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Massive pulmonary embolism; the place for embolectomy

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SUMMARY

Untreated massive pulmonary embolism is associated with a high mortality. Pulmonary embolectomy has been largely superceded by thrombolytic therapy, but there are cases in which pulmonary embolectomy remains the treatment of choice. We present three case reports and discuss the merits of the various treatments available for massive pulmonary embolism. The primary treatment of massive pulmonary embolism should be thrombolytic therapy, but for patients who are at risk of haemorrhage following surgery, who are in cardiogenic shock despite medical treatment, or fail to improve following cardiac arrest, then pulmonary embolectomy remains the treatment of choice.

INTRODUCTION

The role of pulmonary embolectomy in the treatment of massive pulmonary embolus has been in doubt since the introduction of thrombolytic therapy. Indeed a recent review article failed to mention pulmonary embolectomy as a treatment option,[1] relying on anticoagulation or thrombolytic therapy for the treatment of all pulmonary emboli. However, thrombolytic therapy is not always appropriate, and an alternative treatment is desirable for patients who fail to respond to medical treatment. We report three patients in whom thrombolytic therapy was inappropriate and discuss the option of embolectomy.

PATIENTS

1. A 64-year-old man with acute urinary retention underwent open prostatectomy under spinal anaesthesia in another hospital. Postoperative haemorrhage resulted in clot retention necessitating re-exploration two days later, again under spinal anaesthesia. Within twelve hours he became acutely dyspnoeic and hypoxic with tachycardia. An initial differential diagnosis of either adult respiratory distress syndrome or massive pulmonary embolism was made. He was urgently transferred to the respiratory intensive care unit at The Royal Victoria Hospital for further treatment. On admission he gave no history of chest pain, but was centrally cyanosed with a respiratory rate of 28 per minute. He had bilateral varicose veins with thrombophlebitis of the left long saphenous vein from the lower leg to the groin. The pO_2 in arterial blood was 58mmHg (7·6kPa), pCO_2 was 31mmHg (4·1kPa) and pH 7·3. Electrocardiograph showed no

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specific abnormality. Chest X-ray showed oligaemia of the left lung consistent with pulmonary embolism (Fig 1). Pulmonary angiography confirmed a large pulmonary embolus obstructing nearly all flow in the left pulmonary artery (Fig 2). In view of the recent history of surgery with bleeding complications, thrombolytic therapy was felt to be contra-indicated, and pulmonary embolectomy was performed using cardio-pulmonary bypass. A 10cm clot was removed from the left pulmonary artery. He made an uneventful recovery and was discharged back to the referring hospital on the 7th postoperative day, taking oral anticoagulants. He remained well at out-patient follow-up two months later.

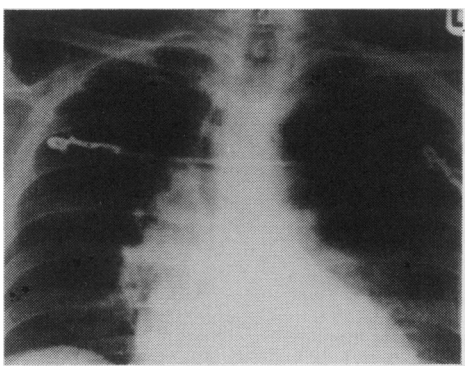

Fig 1. Patient 1: Plain chest radiograph showing oligaemia of the left lung.

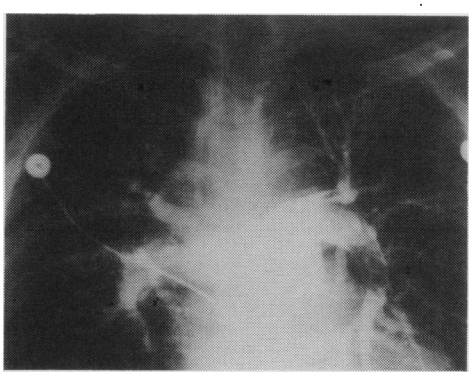

Fig 2. Patient 1: Pulmonary angiogram showing large embolus in the left main pulmonary artery.

2. A 68-year-old man was admitted with a history of vomiting and diarrhoea. Investigation revealed a tumour in the ascending colon. Nine days after admission he underwent right hemicolectomy for an obstructing well differentiated adeno-carcinoma of the ascending colon (Duke's stage C). He received subcutaneous heparin 500 units twice daily before and after operation. At 48 hours he became dyspnoeic and cyanosed. Electrocardiograph and chest X-ray were interpreted as normal and he was considered to have developed septic complications of his large bowel surgery. He was started on a dopamine infusion because of poor peripheral perfusion and a blood pressure of 80/60mmHg. His condition continued to deteriorate and a cardiac surgical opinion was sought 11 hours after the initial episode. At this time he was cyanosed with a markedly raised venous pressure and a low cardiac output. Heart rate was 120 per min with a gallop rhythm. Respiratory rate was 28 per minute. Repeat chest X-ray showed oligaemia of the right lung and of the upper zone of the left lung. The pO_2 in arterial blood was 57mmHg (7·5kPa), pCO_2 26mmHg (3·4kPa) and pH 7·37. A diagnosis of pulmonary embolism was made, and confirmed by pulmonary angiography. Both lungs were severely affected, with sparing only of the lingular segments of the left upper lobe (Fig 3). His condition was continuing to deteriorate despite

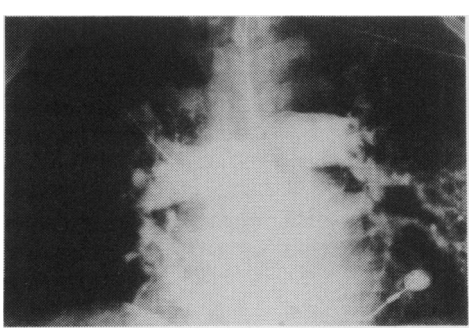

Fig 3. Patient 2: Pulmonary angiogram showing bilateral multiple pulmonary emboli with sparing of the artery to the lingular segment of the left upper lobe.

inotropic support. At operation, fifteen hours after the initial event, cardio-pulmonary bypass facilitated removal of a large amount of clot from both pulmonary arteries. He made an uneventful recovery, remained on oral anti-coagulants for three months, and remained well four months postoperatively.

3. A 64-year-old woman was admitted to the Mater Infirmorum Hospital with a two-year history of bright red rectal bleeding associated with diarrhoea, lethargy and weight loss. She was found to have a carcinoma of the ano-rectal juntion. Abdomino-perineal resection was performed nine days after admission, secondary closure of the perineal wound was performed after three days. Five days later, her condition deteriorated suddenly. She complained of nausea, and was found to have a tachycardia with hypotension and a raised jugular venous pressure. Electrocardiograph showed no acute changes but a diagnosis of pulmonary embolism was made on clinical grounds. She was transferred to the intensive care unit and treated by heparin anticoagulation and oxygen by face mask. Arterial blood showed a pO_2 of 48 mmHg (6·3 kPa), pCO_2 of 26 mmHg (3·4 kPa), pH of 7·2 and "base excess" of −11·2. Ninety minutes following the initial episode she became more hypotensive and suffered a cardiorespiratory arrest. The regional cardiothoracic unit was being consulted by telephone, and a decision was made to attempt embolectomy. Cardiopulmonary resuscitation was continued during her transfer to theatre whilst the operating surgeon travelled between the two hospitals. Pulmonary embolectomy was performed during a two-minute period of inflow occlusion after thirty minutes of attempted resuscitation. A large clot was removed from the right main pulmonary artery, and satisfactory cardiac output was restored with the aid of inotropic support. During this episode she suffered a major cerebral insult and required tracheostomy and ventilation for twenty-four days. The electroencephalogram at ten days was grossly abnormal and the report suggested a poor prognosis for cerebral recovery. However, she regained consciousness and returned to the ward twenty-six days after embolectomy. Normal cerebration and speech slowly returned. She was discharged home 110 days following embolectomy, with a residual right hemiparesis, but able to walk with the aid of a frame. Her hemiplegia has continued to improve.

DISCUSSION

Pulmonary embolism remains a significant cause of hospital morbidity and mortality, especially following major surgery. Untreated massive pulmonary embolism with significant hypotension is associated with a greater than 50% mortality rate, and 70% of the fatalities occur within two hours.[2] Treatment should therefore be started as soon as the condition is diagnosed. The major problem in making the diagnosis is that the symptoms are often non-specific[3] and patients who survive the initial embolus may be diagnosed as having a wide variety of disease. The most accurate diagnostic test is pulmonary angiography, but as it has its own morbidity and mortality in the severely compromised patient, it is generally only used to confirm a diagnosis made on the basis of clinical examination, chest radiography, electrocardiography and blood gas measurement. The lung perfusion scan is a useful test to exclude pulmonary embolism, but the false positive rate may be over 50% in patients suspected of having pulmonary emboli. Early pulmonary angiography is more than 90% accurate.[3]

The operation of pulmonary embolectomy for massive pulmonary embolism was first described by Trendelenberg in 1908,[4] performed via a left thoracotomy.

However, he never succeeded in saving a life despite more than 12 attempts and it was left to one of his assistants, Kirschner, to perform the first successful embolectomy in 1924.[5] The technique of inflow occlusion, clamping both the inferior and superior venae cavae prior to opening the pulmonary artery, removing the clot, and then placing a side clamp on the pulmonary artery so that the vena caval clamps were removed in under two and a half minutes, was described by Ivor Lewis in 1960.[6] In the same year, Allison[7] described the technique of embolectomy under hypothermia. In 1961 the first embolectomy using cardio-pulmonary bypass was performed by Sharp.[8]

The scope for treatment of pulmonary embolism was greatly improved by the development of thrombolytic therapy after the 1960s.[9] Miller, Hall and Paneth[10] from the Brompton Hospital reported a comparison between embolectomy using cardiopulmonary bypass, thrombolytic therapy, and anticoagulation with heparin, in massive pulmonary embolism. Their conclusion was that in most patients there was no advantage of embolectomy over thrombolytic therapy, but both were better than treatment with heparin alone. However, they recommended embol-ectomy in patients who were within 48 hours of surgery, because of the risk of haemorrhage with thrombolytic therapy. They also recommended surgery for those who were shocked, and showed no sign of improvement, or continued to deteriorate after one hour of medical treatment.

Many of the above patients were selected, already having survived transfer from a referring hospital where the facilities for cardiopulmonary bypass were not available. Clarke and Abrams[11] in Birmingham have offered an embolectomy service, travelling to the hospital where the patient was in extremis, taking with them the few extra instruments required in a sterile pack and operating under inflow occlusion, rather than forcing the patient to undergo a hazardous journey to a hospital with bypass facilities. They have reported a commendable success rate over a twenty-five year period. All but one of their patients had a systolic blood pressure < 100mmHg. Three patients had embolectomy on cardio-pulmonary bypass and survived. Fifty-five had embolectomy under normothermic inflow occlusion: of 36 who had not undergone a period of circulatory arrest, 35 survived the operation, 7 died during the postoperative period, and 4 deaths were not related to pulmonary embolism. In the 19 patients who had a period of cardiac arrest the mortality was 16, two not being related to the operation or pulmonary embolus. To minimise the time between the embolus occuring and surgery and because the facility was often not available, they did not usually perform angiography, and this led to a further six patients being operated on in the absence of embolism. They claim that any surgeon with thoracic training should be able to perform this operation and in an emergency when alternative treatment is inappropriate or is failing, it should be considered.

Mattox et al[12] reported an impressive series of 40 embolectomies on 39 patients in extremis over a twenty year period, of whom 17 (43%) survived to leave hospital. Two of the deaths were in patients who had tumour emboli and three more were in patients who had cor pulmonale from previous emboli. Twenty-three patients had angiography performed prior to embolectomy, twenty-two of these were on partial bypass during angiography, which had been instituted under local anaesthetic via femoral arterial and venous cannulation.

Greenfield[13] from Richmond has reported the use of a suction catheter for the trans-venous removal of emboli at the time of angiography, but it may be unsuitable in the severely compromised patient. The use of femoro-femoral

partial bypass to support patients undergoing pulmonary angiography may make this technique more widely applicable in the future, but its role is not yet fully established.

Following embolectomy or thrombolysis, anticoagulation with heparin, continued until oral therapy is established, is recommended for at least three months to decrease the risk of recurrent emboli.[1]

Interruption of the inferior vena cava should probably only be considered in cases of recurrent embolism. Although Mattox reported routinely performing ligation of the inferior vena cava at the time of embolectomy, both Clarke (two patients) and Miller (no patients in the surgical group, one patient in the streptokinase group and two patients in the heparin group) reported low incidence of recurrent emboli. Clarke reported using this technique initially, but abandoned it because the decreased venous return produced a profound fall of cardiac output in several patients and also three patients complained of long term oedema of both legs.

Two of the three patients we report here were unsuitable for treatment with thrombolytic therapy because of recent surgery, and anticoagulation alone was not considered likely to offer satisfactory haemodynamic improvement. The third patient's collapse made thrombolytic therapy inappropriate. Although the first of the three patients might have survived without embolectomy, the second patient was close to death by the time of his operation, and the third patient was not responding to cardiopulmonary resuscitation prior to embolectomy.

Although pulmonary embolectomy has been largely superceded by thrombolytic therapy, and the new thrombolytics undergoing investigation may prove clinically more effective and safer, there remain three indications for surgery in massive pulmonary embolism. These are in the patient with a history of recent surgery, in the patient who is not responding to or deteriorating with thrombolytic therapy, and in the patient who is profoundly shocked needing cardiopulmonary resuscitation. The absence of facilities for cardiopulmonary bypass does not preclude surgery. In the future the availability of easily portable cardiopulmonary bypass equipment may help further to reduce the mortality of this condition.

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Meningitis in an Irish community

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SUMMARY

A series of 26 cases of meningitis occurring in one year in a defined area is presented. The clinical features, and complications are reviewed. Neisseria meningitidis *occurred twice as commonly as* Haemophilus influenzae, *suggesting that the pattern of infection differs from that reported in England and Wales. An incidence of 4·6/100,000 for* N. meningitidis *is reported exceeding rates of infection in previous UK "epidemics".*

INTRODUCTION

Acute pyogenic meningitis continues to be responsible for morbidity and mortality in the community despite the availability of effective antibiotic therapy. Debate concerning the timing of lumbar puncture has made acute meningitis a controversial subject once again.[1] The disease itself is also changing rapidly, with an alteration in the relative contributions of the different pathogens. In the past *Neisseria meningitidis* was the most common pathogen isolated,[2] but in the period between 1984–5 *Haemophilus influenzae* became the predominant organism isolated in cases of meningitis in the United Kingdom.[3] The incidence of *N. meningitidis* rises to a peak every ten to twelve years. This phenomenon occurs in countries of the developed world, where peak incidence reported may be as high as 95/100,000 (Faroes 1981).[4] This contrasts with the dramatic epidemics which occur in the "meningitis belt" of Africa where incidence may exceed 1000/100,000.[5] Recent medical and media attention has been focused on an upsurge in meningococcal meningitis.[6] Although this represents an increase in the incidence of sporadic cases of meningitis the term "epidemic" is inappropriate.

There are no previous surveys of acute non-viral meningitis and meningococcal septicaemia in an Irish community. We therefore present the clinical features, laboratory results, and outcome from a series of patients resident in the Southern Health and Social Services Board Area, Northern Ireland.

METHODS AND PATIENTS

Twenty-six cases of acute pyogenic meningitis arising in the area of the Southern Health and Social Services Board between August 1984 and August 1985 were studied. The area has a population of 286,600 and is served by three main hospitals, Craigavon Area Hospital, South Tyrone Hospital, and Daisy Hill

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Hospital. All cases presenting to these hospitals are included. In addition to this, the medical records departments of the regional referral centres in Northern Ireland were consulted to find cases arising in the area but presenting elsewhere. Two additional cases were found by this means. The case records of the patients identified were obtained and a series of standard items abstracted and recorded on computer database. The diagnostic criteria for inclusion in the study were (*a.*) Culture of bacteria or fungi in the CSF, (*b.*) Culture of bacteria or fungi in the blood with $>0{\cdot}4 \times 10^9/l$ white blood cells in the CSF, or (*c.*) Culture of *N. meningitidis* in the blood.

RESULTS

Twenty-six patients (10 females and 16 males) presented with pyogenic meningitis, of whom 22 were children under the age of 12. There was one case of neonatal meningitis (*Escherichia coli*). This gives a crude incidence of 9·1/100,000. More than half of the cases presented from January to March. In only one occasion, a sibling, was there any evidence of direct contact between cases. This child who presented with a purpuric rash was the brother of a boy with *N. meningitidis* septicaemia. Although there was no conformation by bacterial culture his case was included with a diagnosis of invasive meningococcal disease.

The length of prodromal illness was less than 24 hours in eleven cases and less than 72 hours in a further seven. In six cases the patient had been unwell for up to a week, and the single case of cryptococcal meningitis presented after a period of six weeks of ill health.

Consciousness was impaired in nine cases and four had convulsions before or shortly following admission. In only fourteen was meningism recorded. Eight patients with meningococcal septicaemia had a characteristic purpuric rash.

Predisposing factors were present in three cases. One young girl developed a fatal meningitis following coagulase negative Staphylococcal colonization of her ventriculo-peritoneal shunt, and one man with pneumococcal meningitis had sustained a skull fracture in a road traffic accident six years previously. The patient with cryptococcal meningitis had normal granulocyte counts and immunoglobulin levels: antibodies to the human immunodeficiency virus were not detected. He was, however, a keen pigeon breeder.

All but one of the patients had a positive culture in either CSF or blood. Seven cases had positive blood and CSF culture, 14 had positive CSF culture alone, and 3 cases of *N. meningitidis* were diagnosed by blood culture alone.

N. meningitidis was the most common organism isolated (13 cases): five of these were found to be group B and three group C, one organism would not agglutinate any of the typing sera available and in four grouping was not performed. This implies an incidence of 4·6/100,000 for *N. meningitidis* during the period of study. *H. influenzae* type B was responsible for five cases and *Streptococcus pneumoniae* for three. Other organisms isolated included *Streptococcus pyogenes* (2 cases), *E. coli* (1 case), *Staphylococcus albus* (1 case) and *Cryptococcus neoformans* (1 case).

Culture of blood and CSF, together with peripheral neutrophilia were the most useful laboratory tests. Nine of the patients had a CSF glucose of less than 1·0 mmol/l. An organism was demonstrated on direct examination of the CSF in less than half of the cases. This is usual in cases of meningococcal septicaemia, but was also found for other pathogens. A rise in CSF white cell count was found less

often in cases of meningococcal disease mainly due to patients presenting with the septicaemic form of disease. In four patients, examination of the CSF on admission was completely normal. In two this was expected since their presentation was predominantly septicaemic, but in a further two cases no explanation could be found. These were all later culture positive. The details of the laboratory investigations are set out in the Table.

TABLE

Laboratory results in 26 cases of acute meningitis

	N. meningitidis **(a)**	*H. influenzae* **(b)**	*S. pneumoniae*	Others
Raised peripheral neutrophil count ($>10^9$/ml)	10	4	3	3
CSF protein				
<0·4 g/l	4	1	1	1
0·4 – 1·0 g/l	3	2	0	2
>1·0 g/l	3	2	2	2
CSF glucose				
<60% blood glucose	3	1	1	2
<1·0 mmol/l	4	2	1	2
Significant increase in CSF polymorphs ($>0{\cdot}9 \times 10^9$/l)	7	4	3	4
Organisms seen in CSF on microscopy	6	2	2	3 **(c)**
Positive blood culture	4	1	1	2
Totals	13	5	3	5

(a) includes three cases with CSF culture result only and one with no blood glucose.

(b) includes one case with no blood glucose result.

(c) Cryptococci only seen on later specimens.

During the period of the study there were two deaths: one child with meningococcal septicaemia, and another following coagulase negative Staphylococcal meningitis. Sixteen patients made an uncomplicated recovery. Four patients with meningococcal disease suffered transient sequelae; one had a respiratory arrest, one disseminated intravascular coagulation and three had significant skin necrosis. One patient with pneumococcal meningitis had congestive cardiac failure and at review was shown to have residual hearing loss. A single patient with *H. influenzae* had a sub-dural effusion.

DISCUSSION

As expected, the most consistently useful laboratory investigations were examination of the CSF and blood culture, together with the CSF and peripheral blood white cell count. All of the patients but one were culture positive. An organism was demonstrated on a direct Gram stain in less than half of the cases. Two patients with meningococcal septicaemia had normal CSF examination, but in a further two patients no explanation for the normal initial results could be found. This emphasises the need to make use of the antigen detection methods which

have become available for early diagnosis. Most of these are cheap and easy to perform and do not depend on sophisticated equipment.[7] Methods include latex agglutination, co-agglutination, and counterimmuno-electrophoresis. They are of particular value when the clinical and laboratory picture is clouded by pre-admission antibiotics. Although antigen detection techniques were not in routine use during this period, these methods were later introduced.

The outcome of infection was favourable in most cases. One death occurred in a young boy with meningococcal septicaemia whose illness followed a fulminant course. The second death was in a child with an intraventricular shunt who had had repeated episodes of infection in the past.

Bacterial meningitis is a continuing cause of morbidity and mortality in children in the area of the Southern Board. This study demonstrates important differences in the relative contribution of different pathogens. Goldacre's review showed the meningococcus to be the most frequent pathogen isolated in patients with meningitis in the Oxford region during the period 1969–1973.[8] Ispahani, studying a population in Nottingham between 1974 and 1980, demonstrated a similar pattern as did Davies *et al,* in Birmingham during the ten years 1968–1977.[9, 10] These figures may to some extent be distorted by the peak in the incidence of meningococcal meningitis which occurred in 1974.[2] During the 1980s *H. influenzae* became the most frequent pathogen reported by the Public Health Laboratory Service,[3] a pattern which has been present in the USA for many years.[11]. Our report indicates that this change in incidence has not yet occurred in the population studied. The distribution of pathogens is similar to the reports compiled by the communicable diseases report for Northern Ireland suggesting that this study provides a representative picture of the disease in the whole province. The meningococcus was the predominant organism responsible for reported cases of meningitis throughout the province throughout 1985–86.

The incidence of *N. meningitidis* infection during this study exceeds that of any health district in England and Wales during the 1986 "epidemic", and is comparable to the peak incidence in several other northern European countries.[11, 12] The rate of 4·6/100,000 suggests that the background incidence of *N. meningitidis* is higher in Northern Ireland or that an epidemic was occurring during the period of study. The meningococci isolated were of at least three different serotypes, B, C and non-typeable, which would favour the former explanation. During the period 1975–84 the average number of deaths from bacterial meningitis in Northern Ireland was 8·7 per year. This supports the idea that bacterial meningitis is more common than is demonstrated by notifications or laboratory reports. A final solution of this problem will only be revealed by a further study.

We conclude that the pattern of bacterial meningitis is following a different trend in Northern Ireland from that seen in the rest of the United Kingdom. In the succeeding years it would be interesting to chart the changes in the pattern of disease in the community. However, current reporting strategies will not permit this since during the study period only 15 cases of laboratory proven infection from this area were actually notified. Such deficiencies are commonly found in other European countries, including the notification of meningococcal infections in England and Wales,[2] and the Netherlands.[13] There is a real need to improve the standard of reporting. The category of "acute meningitis" is vague as it does not distinguish between viral and bacterial causes or between bacterial species. It is therefore not a useful epidemiological index for clinicians. A more

comprehensive reporting system for bacterial meningitis could include personal details of the patient, the aetiological agent and its sensitivities, together with the treatment and outcome. Such a system would provide a baseline from which to evaluate potential epidemics, to plan control measures and to assist the planning and allocation of resources for infectious diseases and microbiology. The appearance of penicillin resistant pneumococci and chloramphenicol resistant *H. influenzae* means that the surveillance of antimicrobial susceptibility in organisms causing acute meningitis is of increasing importance. Without more detailed information it will be impossible to understand changes in the pattern of this important disease in our community, to advise on empiric therapy and to institute control measures when necessary.

The authors wish to thank the physicians who made their cases available for study, the laboratory staff of all three hospitals, and the medical records officers.

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The application of DNA technology to tissue typing

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SUMMARY

A new method of determining class II HLA antigens by genotyping with HLA DNA probes is described. This method compares favourably to the traditional serological methods.

INTRODUCTION

HLA cell-surface antigens are involved in various functions of the immune system, such as co-operation between lymphocyte subsets, antigen presentation and allograft rejection.[1] Some HLA antigens have been shown to be associated with susceptibility to certain diseases.[2] Bone marrow transplantation usually requires an identical HLA match between donor and patient for class I (HLA-A, -B, -C) and class II (HLA-DR, -DQ, -DP) antigens.[3] Although a complete match is not an absolute requirement in kidney transplantation, there is a significant improvement of graft survival between the best and worst matches for HLA -A, -B and -DR antigens.[4, 5, 6]

HLA typing is traditionally performed according to immunological methods using antibodies reacting with HLA gene products on the cell surface. These methods rely on obtaining monospecific alloantisera from multiparous females.[7] In addition for class II typing suitable B lymphocyte preparations are required.

It is now possible to apply techniques of molecular biology to the problems of HLA typing. Restriction endonucleases that recognise specific nucleotide sequences are used to digest DNA. The resulting DNA fragments are then separated according to size by electrophoresis in agarose gels and transferred to nylon membranes. Radiolabelled complementary DNA probes are used to identify genes for class I and class II regions. Each probe hybridises with several fragments of DNA, some of which are constant in the general population whereas others called allogenotypes are present in some persons but absent in others. We describe the use of a single enzyme/single blot/multiple reprobe system of HLA-DR and -DQ (developed in this laboratory in conjunction with Dr J Bidwell, United Kingdom Transplant Service, Bristol) to assign by analysis of allogenotypes the HLA-DR and DQ specificity at the DNA level. We have applied this technique to typing renal patients in order to assess any differences obtained by this technique when compared to conventional serological methods.

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MATERIALS AND METHODS

The renal population consisted of 160 patients either transplanted consecutively since May 1986 or awaiting a transplant. DNA was prepared from peripheral blood leucocytes which were isolated from EDTA blood by two erythrocyte lysis steps using 2–3 volumes of red cell lysis buffer RCLB (10mM Tris·HCl pH7·6/5mM $MgCl_2$/10mM NaCl). The leucocytes were resuspended in 2ml RCLB followed by the addition of 13ml white cell lysis buffer (10mM Tris·HCl pH7·6, 10mM EDTA pH8·0, 50mM NaCl, 0·2% sodium dodecyl sulphate, 200µg/ml proteinase K), mixed gently and incubated overnight in a shaking water bath at 42°C. Proteins were removed by two extractions with 15ml phenol/chloroform/isoamylalcohol (25:24:1) followed by two 15ml extractions with chloroform/isoamylalcohol (24:1). DNA was precipitated from the aqueous layer by the addition of 300µl 3M NaCl and 15ml isopropanol. The DNA was harvested using a sealed glass pasteur pipette, washed three times in 4ml 70% ethanol, dried and resuspended in TE (10mM Tris HCl pH7·6, 1mM EDTA pH8·0). The DNA was assayed spectrophotometrically and stored at a final concentration of 1–2µg/µl at 4°C.

Samples of genomic DNA (8µg) were digested to completion with the restriction endonuclease Taq 1 (Bethesda Research Laboratories, 5 units/µg DNA) and resolved using agarose gel electrophoresis for 18hr at 40V in an agarose gel (25 × 20 × 0·6cm, 0·7% w/v agarose (Bethesda Research Laboratories) in TAE electrophoresis buffer (40mM Tris·acetate, 1mM EDTA containing 0·5µg/ml ethidium bromide)). The DNA was denatured, transferred to a nylon membrane (Amersham Hybond·N) and hybridised.[8] Membranes were hybridised sequentially with the following radiolabelled exon·specific cDNA probes; HLA–DRß pRTV1,[8] HLA–DQß pII–ß–I[9] and HLA–DQα pDCH1.[10] After autoradiography the membranes were dehybridised by washing, with gentle agitation, at 42°C in 500ml 0·4M NaOH for 30min followed by 500ml 0·2M Tris·HCl pH7·6 0·5% SDS for 30min prior to rehybridisation.

RESULTS

The figure shows the allogenotypes revealed using the DRß cDNA probe pRTV1. The allogenotypes DRß1, ß2, ß3, ß4, ß7, ß8, ß9, ß11, ß12, ß13, ß14 are associated with the corresponding HLA–DR serological specificities. Allogenotypes are also observed which are subtypes of individual DR specificities not previously differentiated by serological methods i.e. DRß3[1], 3[2] and DRß13a[1], 13a[2], 13a[3], 13a[4].

The figure also shows the results of rehybridising the membrane with the DQß and DQα cDNA probes respectively. Using the DQß probe, six DQß allogenotypes are identified (ß1a, ß1b, ß2a, ß2b, ß3a, ß3b). These allogenotypes correlate with known serological types of DQ i.e. ß1a and ß1b with DQw1 serotypes; ß2a and ß2b with DQw2 serotypes; ß3a and ß3b with DQw3 serotypes.

Using the DQα probe, five DQα allogenotypes are identified (α1a, α1b, α1c, α2 and α3). These describe another allelic series which also correlates with serological types of DQ, though to a lesser degree than the absolute correlation seen with DQß allogenotypes. Thus DQα1a, α1b and α1c correlate with DQw1 serotypes though α1b is also associated with DQw3, and α2 and α3 allogenotypes are both associated with DQw2 and DQw3 serotypes.

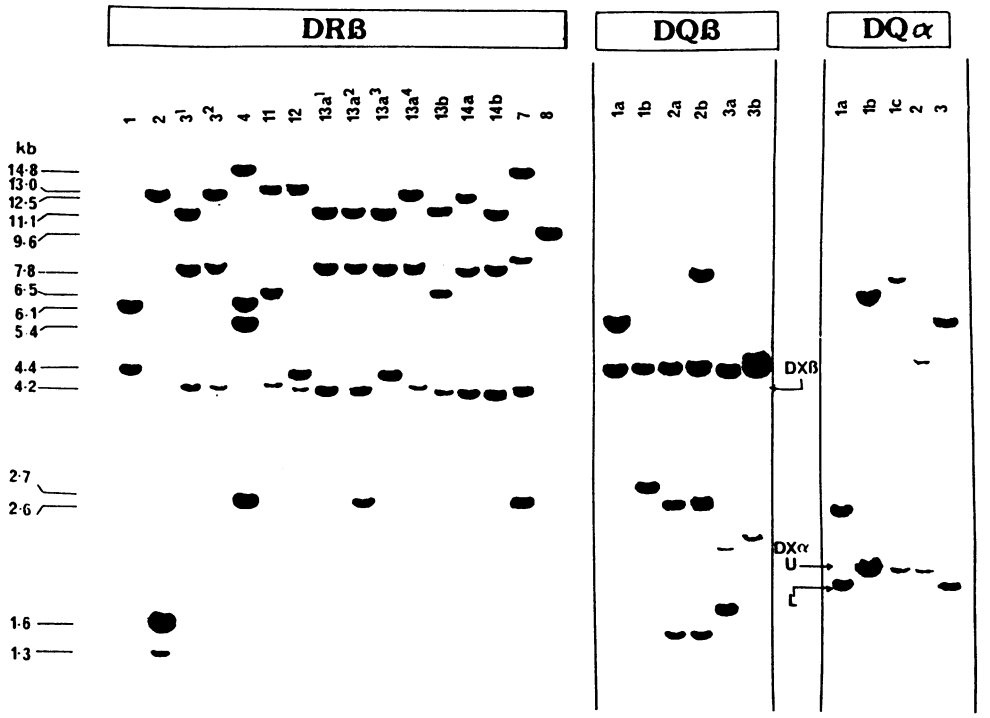

Figure. Allogenotypes found using Taq I and the DRß, DQß and DQ α probes.

When the results obtained by allogenotyping were compared to those obtained by serology, there was a difference in 25 (16%) of the patients. In 5 patients an incorrect antigen had been assigned by serological typing. On two occasions DRw13 had been assigned instead of DR3 and on a further two occasions instead of DRw12. On one occasion DRw12 was assigned instead of DRw13. In 20 patients an antigen detected by allogenotyping was not detected by serolgy. The antigen HLA-DRBr was not detected in 11 patients, HLA-DRwB was not detected in 2 patients, HLA-DRw14 was not detected in 2 patients and the following antigens were not detected on one occasion in separate patients; HLA-DR1, -DR3, -DR4, -DR9, -DRw13.

DISCUSSION

The methods described in this report permit the assignment of HLA-DR and -DQ specificities by the sequential use of short DRß, DQß and DQ α DNA probes. The DRß, DQß and DQ α allogenotypes in heterozygotes are interpreted by the summation of patterns demonstrated by homozygous control cells.

A major application of DNA typing is in the interpretation of HLA-DR and DQ specificities where serological assignment is not possible or where the results are equivocal. Serological assignment is influenced by poor quality or low numbers of circulating B cells and by the lack of reliable monospecific alloantisera for certain specificities.

Our results have shown that the majority (20/25) of differences between the serological and allogenotyping method are due to the detection by allogenotyping

of antigens which are not detected by the serological method. The remaining five discrepancies arose due to the difficulties involved in the serological typing of DRw13, an antigen for which it is difficult to obtain monospecific sera. Thus all the differences that have occurred are due to the unavailability of suitable sera. On all occasions when differences between the allogenotype and the serological type are found, it is possible, using the allogenotype result, to assess the quality of the HLA sera.

We can now assign DQ specificities using allogenotyping, which we could not do by serological means because all the DQ sera available to this laboratory are contaminated with DR antibodies. For example, all available DQw1 sera always contain antibodies against one or more of the DR antigens associated with DQw1 (DR1, DR2 and DRw6).

There are two main disadvantages at present to the allogenotyping: cost and length of time to perform the technique. The difficulties in cost are due to the initial setting-up of the technique. Once the techniques are established then the cost of DNA typing is very similar to the cost of serological typing. The technique is not readily applicable to cadaver donor typing since it is incompatible with the clinical urgency set by organ ischaemia time. In the future it should be possible to shorten the time taken by using allele specific probes.

The DNA methods can also be applied to disease studies. At present we are investigating multiple sclerosis patients with a variety of endonucleases to determine any differences in the size of the resulting DNA fragments between patients and controls. Previous disease studies, using Taq 1 restriction enzyme and DRβ and DQβ probes, have identified a restriction fragment pattern observed in 11 of 12 DR3/4 patients with insulin dependent diabetes mellitus but absent in all 12 DR-matched controls.[11]

A further possible application of DNA typing is the replacement of the mixed lymphocyte culture for measuring differences between donor and recipient in bone marrow transplantation. Using a variety of endonucleases and probes, it should be possible to determine if there are any differences between recipient and donor. This would be extremely useful in those instances where donor and recipient sibling are separated by geography or where the use of a non-related HLA identical donor is contemplated.

Already DNA typing has proved most useful in this laboratory. As the technique is developed, it should be possible to identify splits of DR antigens and more generally to provide further insight into the genetic complexity of the human major histocompatibility complex.

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Does cigarette smoking alter platelet aggregation in women receiving the contraceptive pill?

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SUMMARY

Platelet aggregation was examined in a group of 35 non-smokers and 15 smokers who were receiving the contraceptive pill (30μg ethinyloestradiol and norgestrel or norethisterone). Platelet aggregation induced by ATP was greater after 4–6 months on the contraceptive pill in both smokers and non-smokers, and there was no difference between the two groups. The increased risk of thromboembolic disease in smokers on the contraceptive pill would appear to be unrelated to effects on the platelet.

INTRODUCTION

Oral contraceptives have been available for over 20 years and, despite several highly publicised articles on their adverse effects, they have remained one of the most popular methods of contraception throughout the world. A variety of epidemiological studies have linked their use with an increased incidence of venous and arterial thromboembolism, and cigarette smoking has been strongly associated with this increased risk, at least with regard to arterial thrombosis.[1] Recent interest has focused on the effects of oral contraceptives on blood coagulation factors and platelet function.[2] Previous studies have shown an increase in platelet aggregation in women receiving the contraceptive pill.[3, 4]

Cigarette smoking is also a major factor for the development of cardiovascular disease. It has been suggested that both endothelial and platelet factors are involved in the effect of smoking. Although an effect on endothelial damage has largely been confirmed,[5, 6] data dealing with platelet function are conflicting.[7, 8] Since epidemiological studies show an increased risk for women who are smokers and are receiving the contraceptive pill,[9] this study was carried out to see if this increased risk could be related to effects on the platelet.

METHODS

Women who were intending to start the contraceptive pill were recruited from a local family planning clinic. Informed consent was obtained from all subjects. Ethical approval had been obtained from the Research Ethical Committee of the

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Queen's University of Belfast. Of the 80 subjects recruited for the study, only 50 completed the protocol. Their ages ranged from 17 to 31 years (mean 22 years). Thirty-five were non-smokers and 15 were smokers (mean consumption 15 cigarettes per day). Patients who were on any other regular medication, had any abnormality of their haematological indices or serum lipids, or who had received an oral contraceptive within the previous six months, were excluded from the study. Blood samples were taken for estimation of platelet aggregation before starting the contraceptive pill and four to six months after treatment. All subjects received a combination of 30μg of ethinyloestradiol in combination with 150–250μg of levonorgestrel or 1·5mg norethisterone.

For measurement of platelet aggregation, 20ml venous blood was obtained and immediately anticoagulated with 3·8% sodium citrate using nine parts blood and one part citrate by volume. After inversion and gentle mixing this was centrifuged at 200g for 10 minutes. The platelet-rich plasma was removed with a plastic pipette and diluted with saline to give a platelet concentration of 4×10^8 cells/ml. One ml aliquots of this were pipetted into siliconized cuvettes and placed in the heated block of an aggregometer. When the sample stablilized at 37°C a magnetic stir-bar and the impedence electrode were inserted into the cuvette. The aggregometer and recorder were then brought to zero at the pre-set baseline and calibrated using the integral 5 ohm resistance. The recorder was started immediately after the addition of 10μl of aggregation agent. After four minutes for ADP or seven minutes for adrenaline, the recorder was stopped, the pen deflection measured and the impedence of ohms calculated. These agents were used because they have been the two most commonly employed in similar studies examining platelet aggregation with the contraceptive pill. The aggregating reagents used were supplied by the Sigma Chemical Company and were designed to produce a final concentration of 2 mmol/l in the prepared sample. Comparisons between baseline and 4–6 months of therapy and between smokers and non-smokers were made using a paired t test. Values are expressed as the mean ± standard deviation of the mean.

RESULTS

Adrenaline failed to induce platelet aggregation in several patients. We therefore excluded those patients who had a response of less than 3 ohms (sensitivity of the method) from further analysis. On this basis we were able to analyse data in 22 non-smokers and seven smokers. By contrast, platelet aggregation induced by ADP occurred with all samples.

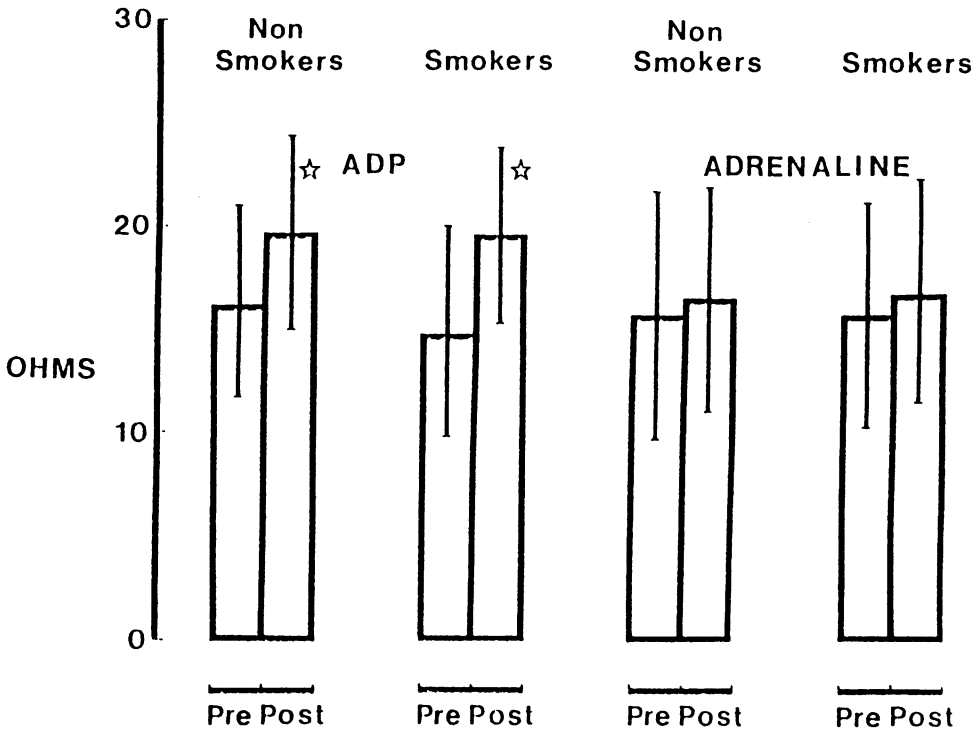
No change in adrenaline-induced platelet aggregation was observed, either between baseline and the effects at 4–6 months or between smokers and non-smokers. There was a significant increase in ADP induced platelet aggregation in the 35 non-smokers and 15 smokers ($p < 0{\cdot}05$). There was no significant difference observed between the two groups (Fig).

DISCUSSION

Two different approaches have been used to study the effects of oral contraceptives on blood clotting. One technique is to perform large-scale prospective and retrospective studies of women receiving the contraceptive pill and determine the incidence of thromboembolic complications as compared with a control group. The other method is to study the effects of oral contraceptives on blood coagulation and platelet function in smaller groups of women. The techniques

FIGURE

*Measurement of electrical impedance (ohms) due to platelet aggregation induced by ADP or adrenaline, before (pre) and 4–6 months after (post) taking the contraceptive pill in a group of smokers and non-smokers. * $p < 0{\cdot}05$ compared with baseline. Values are mean ± standard deviation.*



used in this study are widely used as an index of platelet function. Our results confirm those of other investigators that the combined contraceptive pill increases platelet aggregation as assessed by ADP. The reasons why we were unable to show an effect with adrenaline are unclear since platelet aggregation has been shown to be increased by other investigators using similar techniques.[4] The fact that adrenaline failed to induce platelet aggregation in some of the subjects, thus reducing the study number, could be a factor.

Although it is generally assumed that the techniques used in this study give some indication of platelet aggregation *in vivo*, the methods used are artificial and use supraphysiological doses of aggregating agents. The finding of increased platelet aggregation does not necessarily imply that this is associated with an increased risk of thrombosis. This study suggests that smoking in patients receiving the contraceptive pill has little effect on platelet function which is in keeping with most of the previous published literature. The principal effects of smoking would appear to be on the vascular endothelium [10, 11, 12] and those of the combined contraceptive pill to be on the platelet.

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Cutaneous remnants of the vitellointestinal duct: a clinico-pathological study of 19 cases

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SUMMARY

The presence of cutaneous vitellointestinal duct remnants was confirmed histologically in 19 cases in the period 1970–1984. These lesions occurred mostly in males (16 males, 3 females), and 80% in children under the age of five years. One case was identified in an adult, suggesting that these lesions may in some cases cause little inconvenience, and that their true incidence is underestimated.

INTRODUCTION

Cutaneous vitellointestinal duct remnants are encountered infrequently in clinical practice and although they usually cause little inconvenience they may be associated with intra-abdominal lesions. They are a reminder of the part which the yolk sac plays in human development.

Early in embryonic life the vault of the yolk sac (intra-embryonic yolk sac) forms the primitive gut which is connected to the extra-embryonic yolk sac, or yolk sac proper, by the yolk stalk. By about five weeks' gestation this communication has lengthened, narrowed and forms the omphalomesenteric duct or vitellointestinal duct. At about six weeks it is incorporated into the umbilical cord and by about seven weeks the lumen is obliterated. In most instances no trace of the duct remains at 16 weeks.[1] Abnormalities occur when all or part of the duct remains.[2]

In some cases the entire duct remains patent.[3] After the umbilical cord sloughs, the cutaneous part of the duct is represented by a velvety nodule. Sometimes a volvulus may occur around a persistent patent duct and very occasionally the ileum may prolapse through the patent duct lumen. In some cases only the cutaneous portion of the duct persists which results in either a vitelline polyp or sinus.[4, 5] Other remnants include Meckel's diverticulum which occurs in 1–4% individuals,[6] and represents persistence of the enteric portion of the duct. When both the enteric and cutaneous portions are obliterated, and the middle portion persists, the result is a vitelline cyst.

METHODS

We reviewed all the cases of cutaneous vitellointestinal duct anomalies which were submitted to the departments of histopathology at the Royal Victoria Hospital and Belfast City Hospital from 1970 to 1984. These two departments dealt with 89% of all surgical biopsy specimens examined in the province.

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Nineteen cases were identified. During the same period some 400 Meckel's diverticula and enteric cysts were seen in the two departments, but this study is confined to those remnants which have a cutaneous representation and these internal structures will not be further considered. The clinical details were obtained from hospital notes. Haematoxylin and eosin stained sections were examined, and special stains were carried out in selected cases.

RESULTS

Sixteen cases occurred in males and three in females. Age at presentation ranged from two days in the case of a boy with a vitelline polyp to 24 years in the case of a man with a vitelline sinus; 80% were under five years and 50% were under one year.

Sixteen of the cases presented with a mass in the umbilicus, variously described clinically as a polyp, lump or granuloma. In two cases with a patent vitellointestinal duct the main complaint was of a discharge at the umbilicus. One case (a vitelline sinus) presented with abdominal pain.

The pathological findings were of 15 polyps, two sinuses and two patent vitello-intestinal ducts. One case, a vitelline sinus, was associated with a Meckel's diverticulum, which had also been resected. Histological examination revealed 14 remnant structures which were lined by small intestinal type mucosa only, and five cases lined by gastric mucosa in addition to small intestinal mucosa. In no case was ectopic pancreatic mucosa found.

Sixteen of the subjects were born in the years 1970 – 1984 and during this period there were 404,000 live births in Northern Ireland. These lesions therefore occur with a frequency of at least 1 in 25,000 live births.

DISCUSSION

Cutaneous vitellointestinal duct remnants occur mainly in male subjects, an observation reflected in other studies, and most occurred in young children.[3, 4, 7] In Steck's series, 35% of cases presented in people over 18 years of age, but that study was based on the files of the USA Armed Forces Institute of Pathology and included cases treated at Veterans Administration Hospitals which would not be representative of the general US population. Most cutaneous vitellointestinal duct remnants present early, but they may persist into adult life and apparently cause little or no inconvenience.[8]

In our series, as in those of Steck[3] and Kutin,[4] the most common lesion was a vitelline polyp. We encountered only one case in which a vitelline sinus was associated with a Meckel's diverticulum, although Kutin suggests that additional anomalies may occur in up to 56% of cases where there is a cutaneous remnant. Two of our cases presented with vitelline sinuses, and two more had a patent vitellointestinal duct.

Fourteen were lined or covered by small intestinal type mucosa only and in five cases both intestinal and gastric mucosa were identified. This preponderance of intestinal mucosa is reflected in Steck's study although he encountered ectopic gastric mucosa more frequently. He also came across examples of colonic and pancreatic tissue in some remnants, which reflect the larger size of his series.

Two reports from major maternity hospitals in the United States of America estimate the frequency of patent vitellointestinal ducts between 6·3 and 6·7 per 1,000 births,[9, 10] whereas our figures suggest that they occur much less often

(0·4 per 1,000 births). Since the two histopathology laboratories serve most of the hospitals and general practitioners in Northern Ireland, it would be reasonable to assume that at least 89% of all the cutaneous lesions of vitellointestinal duct origin which occurred in Northern Ireland and required histopathological diagnosis would have been examined in one or other of the two departments, regardless of the patients age or the nature of the unit in which he received treatment. Our figures, although perhaps underestimating the frequency of these lesions by about 10%, probably reflect more accurately the occurrence of these lesions in the general population, than the previous estimates which were based on the rate of occurrence amongst neonates born in specialist obstetric units.

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The development of occupational medicine in Ulster — a personal memoir

J A Smiley

At least since the middle ages physicians have been interested in the relationship of man to his work but it was not until the beginning of the nineteenth century in Great Britain that this was recognised in legislation. Following the passing of the Health and Morals of Apprentices Act in 1802, in the absence of a registry of births, doctors were appointed to certify that the apparent age of young entrants to the mills and factories was above ten years and that they were not unfit. Some of these doctors using the opportunities thus presented to them made observations of clinical interest which prompted their social conscience to protest against the abuses of what came to be known as the Factory System. Such a man was CD Thackrah in Leeds, now regarded as the father of occupational medicine in Britain, but there were others in the 19th century such as Andrew Malcolm and a succession of Purdons in Belfast whose names must be added to the roll of doctors who influenced the legislation which mitigated the effect of factory life on the health of workers.[1] There is some evidence too in the reports of the factory inspectors of the time that a few philanthropic employers did engage the services of a physician to supervise the health of their workers. It would appear, however, that with a few exceptions the certifying surgeons took their work somewhat casually.

In general it was not until the 1914–18 war that the contribution that doctors could make to industry was more fully recognised. The huge proportion of recruits for the services who had to be rejected alerted the nation to the poor physique and inadequate state of nutrition of the working classes. As well two other factors came into operation — the toxicological hazards in the manufacture of armaments, such as trinitrotoluene and lead used by relatively unskilled labour, and the failure, beyond a certain level, to produce more shells by increasing the hours of work. These led to the setting up of the Committee on the Health of Munitions Workers — later the Industrial Fatigue Research Board. As a result the Ministry of Supply engaged a number of doctors in their munition factories to supervise the workers — many of whom were women. A number of these doctors continued in full time employment after the war — some in government posts but others in non-state companies like Imperial Chemical Industries. The economic depression of the thirties militated against any great recruitment of doctors to industry but in the inter-war years the Industrial Fatigue Research Board was re-established as the Industrial Health Research Board — later coming under the aegis of the Medical Research Council. There was a growing acceptance that medicine had a contribution to make to industry.

In 1937 in Great Britain (1938 in Northern Ireland) a new Factory Act was

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(Dr Smiley died on 31 March, 1988).

enacted. Increased responsibilities were given to certifying doctors who now had to examine the under 16 year olds entering industry and from time to time report on illnesses attributed to industrial processes. They were renamed Examining Factory Surgeons. This led after the second world war to an anomaly — that of pre-employment examinations being carried out both by the Government appointed examining surgeons and where such existed the works medical officer — for during the war years the number of doctors entering industry in either a full time or part time capacity was considerable. This dilemma was resolved by the Ministry of Labour recognising the works doctor as the examining surgeon — thus came into being the Appointed Factory Doctors system. To conclude this account of the metamorphosis briefly, it can be said that during the war years the health and nutritional standards of the young people had improved very greatly and the need for their protection had largely faded. As well, the School Medical Service claimed that during their school years the health of the children was adequately supervised by its staff. Although this was universally accepted, it was realised that changes had to be made.

From all this developed a statutory body — the Employment Medical Advisory Service — staffed initially in Northern Ireland by an experienced doctor (WH Hood) who had experience both as a part time and later full time industrial health physician. In 1987 its chief is Dr Gerald Hall and he has two assistant medical officers. Their function is to advise (on request) management, trade unions and individuals on health matters relating to employment and to co-operate with the Factory Inspectorate. From these elements of what was formerly called *"Industrial Medicine"* there began to emerge a specialty *"Occupational Medicine"* in that its practitioners found themselves dealing with problems arising in occupations other than industrial.

As the second world war loomed the manufacture of armaments had begun to absorb the hordes of unemployed — at first men and then women. Short Brothers, then in Rochester, because of their vulnerable position near the east coast were persuaded to open an aircraft factory in Belfast. As the certifying factory surgeon in East Belfast in whose district the new factory was situated I had been invited to train First Aid workers and soon found myself absorbed into the ARP (Air Raid Precaution) organisation. Soon I had become a part time member of staff and with the help of an able nursing sister (Miss IM King) organised a casualty service not only in Queen's Island but in a number of widely dispersed units outside Belfast. We insisted on the employment of competent state registered nurses to deal with casualties. The policy was widely approved both by the managements of the local hospitals (especially Brigadier TW Davidson of the Royal Victoria Hospital) and by the employers who reckoned that less time was lost by instant and local treatment than by transport to a hospital casualty department. As well these women (for at that time nursing was essentially a profession for women) made a very considerable contribution to the morale of the workforce (which now had a large number of women in it) especially in the dark days of the conflict. No account of the development of industrial medicine to occupational medicine would be complete without reference to the tremendous contribution made by the nursing profession.

It soon became evident however that the somewhat informal relationship of the works doctor with management, while it had advantages, was from time to time

fraught with difficulty. Because he was appointed by the company, paid by the company, accommodated by the company and had his stationery supplied by the company, the claim was made by the company's solicitors that the doctor's records belonged to the company and that when accident claims were made by injured workpeople, they the company solicitors must have access to them. For a number of years when claims were made the battle for the confidentiality of medical records had to be fought time and again. Eventually the lawyers came to recognise that like themselves doctors had to behave within the ethical parameters of their profession.

Because the service was regarded as a casualty service by a not very experienced management, observations and then recommendations by the doctors about working conditions were not always well received. Inevitably shop stewards found their way to the medical centres with complaints on alleged health matters and the difficulties of charting a course of action bore heavily on a sensitive conscience. Over difficult months and years the right of the doctor to comment on working conditions came to be recognised until eventually it was a responsibility imposed on him and his successors by legislation. At the outbreak of the second world war I had become a part time member of staff of what had now become Short and Harland's. After the air raids on Belfast my practice had dispersed to the country, and, although most of my time was spent in and around the various units of the aircraft company, I was pressed into service with the Belfast Ropeworks Company,[2] Chloride Electrical Storage Batteries (lead), Mallory Batteries (mercury), the York Street Flax Spinning Company (byssinosis)[3] and two flour mills — and later the British Petroleum oil refinery.

At the end of the war the shipyards were busy helping to replace shipping which had been lost and the board of Harland and Wolff fell into line with the growing practice of enlisting medical help to their management team. Dr FE Fletcher, who had served in the RAF during the war and who was also qualified in law, was their choice. Unfortunately, such was the pressure of casualty work and the consequent number of claims under the Workman's Compensation Act that little time was available to him for monitoring working conditions. There was, however, on the staff of the Ministry of Health's Referee Service Dr Ben Swain who later recognised the high incidence of asbestos-related disease and the sources of origin of these cases. Asbestos had been recognised early in the 19th century as being a risk to health but it was not until 1930 with the increasing use of the material that its serious hazards to those inhaling it were fully appreciated. There are those who feel that the Factory Inspectorate were at fault in not requiring safer working conditions, but during the war the need for production was deemed to be the prime consideration. The plight of those workers disabled and dying as the result of working with asbestos was taken up by Professor Peter Elmes who soon came to be a recognised authority on asbestos-related conditions.[4]

Boiler makers deafness was a condition by tradition recognised as inevitable for those who spent a lifetime in that trade. It was not a condition recognised for compensation purposes. (Although deafness is a social disability, it does not prevent the deaf from earning their living — such was the justification made for disregarding it). Years later when claims at Common Law were initiated and awards made, a number of large companies were brought to the verge of bankruptcy. In the aircraft industry the effect of exposure to noise was recognised

early. The protection of technical staff working at, or in the vicinity of, the wind tunnel was ensured by withdrawing from exposure where possible or by the use of ear muffs. There was however resistance by the workers on the factory floor to the use of ear protection from the noise of rivetting, and supervisory staff were at fault in not insisting on its use. This again demonstrated the somewhat anomalous position of the doctor in industry. He had no executive function (nor has he) outside his own department. It illustrates too another of his functions — that of educating management and men in health care, which in this case was not fully successful.

Because of rivetting, the vibration white finger syndrome was recognised as a problem but it was largely eliminated by altering the frequency of the vibration of hand-held tools. Nowadays whole body rather than segmental vibration is the subject of investigation. In the ropemaking trade there was a serious risk of deafness in the area where ropes and cords were plaited. Plaiting involved the clatter induced by inter-digitating metal cogs through the centre of which passed strands of yarn. The din caused by upwards of forty of these machines at a time was painful to the ear of the casual visitor. The workers communicated by sign language. Various attempts were made in the years following the war to mitigate the noise by mounting the machines on artificial rubbers and by substituting the newly discovered material, nylon, for the metal of the cogs. These experiments were successful only in reducing noise levels to approximately 100 decibels. As a result a policy was adopted of recruiting for this department only those who were already deaf! In the event by the 1950s the use of plaited ropes and window cords was diminishing and no new cases came to light. In the ropeworks and in the various spinning mills acquaintance with young people soon revealed what was called by them "Mill Fever". The condition occurred during the first few days of employment and usually subsided in 2 – 3 days. The youngsters felt miserable, complained of headache and usually had a slight elevation of temperature. The trades being familial, parents recognised the symptoms and in most cases insisted on the victims remaining at work. Their belief was that if they did not, on return later the symptoms would recur. With the greatly improved conditions in the mills and probably as the result of the substitution of man-made fibres for the natural fibres of flax, cotton and hemp, the condition is not now seen.

The story of the recognition of "pouce" as being identical with cotton byssinosis has been told,[3] but there were other problems of a transient and minor nature which caused much distress inside factories and offices where women were employed in large numbers. Following the air raids on Belfast the working population of the industrial areas of the city were dispersed. Whatever the cause, lice infested hair and scabies presented problems not only to the Public Health Authorities but to factory managements as well — giving rise to personnel embarrassments which had to be resolved by the nursing staff. By and large, however, the largest medical problem confronting industry both during and after the war was that of contact dermatitis. It was mitigated to some extent by improved washing facilities, so-called barrier creams and replacement inunctions — especially in those cases where the cause was oil and its various additives, rust inhibitors and antibacterial agents. The problem, however, remains with the introduction to industry of new and powerful adhesives — replacing to some extent welding. Soon after the war industry turned from the manufacture of

armaments to more peaceful projects. Shorts ventured into a number of fields, one of which was the provision of milk churns for a war devastated Europe. The introduction of this enterprise presented me with the one and only outbreak in my experience of metal fume fever. The components of the churn were made separately, galvanised and then welded together. Within hours a procession of men attended the medical centre at one of our dispersal units and being informed that there was a sudden "flu" outbreak, we went to investigate. A reorganisation of the process to ensure that the assembly was done before galvanising resolved the problem. Human error is curtailed in its effects only by being conscious of its possibility. Eight men out of a total of seventeen developed chrome ulcers, with in three cases complete perforation of the nasal septum, when an extractor fan in a lip extraction process was wrongly installed.

Lead poisoning was a not uncommon condition in the middle years of this century. It was caused mostly by the cutting of red leaded plates in the break-up of ships and shipyard cranes. When Chloride Electrical Storage (Exide) established the manufacture of storage batteries the supervision of the workers fell to me. The statutory requirement of examination at fortnightly intervals was farcical — examine buccal membrane for a "blue line", ensure that each worker could stand on tip toe and had no wrist drop! Blood lead estimations were at that time not feasible. Professor RE Lane, who was consultant to the company in Manchester and to whom the elimination of lead poisoning in the manufacture of accumulators was largely due, instituted a system based on his experience of the industry. Anaemia being an early objective sign, haemoglobin estimations and blood counts were done on those most exposed at monthly intervals. Eosinophilia occurring in an individual might not be significant to that individual but if a number of workers in a process displayed the phenomenon, in his opinion the workplace and the process had to be investigated, and no cases of intoxication occurred in that factory. In an adjoining Belfast factory (PR Mallory) there was a mercury hazard where miniature batteries were manufactured and several cases of erethism alerted us to some failures of technique.

So far as my own experience went, the incidence of scrotal cancer with a fatal outcome in a felting works was for some years deeply embarrassing, until the insurance company raised the insurance premiums to the extent that the offending firm went out of business. During all these post-war years other enterprises were being undertaken by colleagues. In particular Dr Edwin James in the Tyrone County Hospital was seeing and writing about farmer's lung which was very prevalent in his area and Dr Tom Milliken came to be recognised as a very considerable authority on the hazards of agriculture generally — and on his retirement from his hospital appointment has taken up a new career with the Belfast Corporation.

One of the civil engineering projects having a considerable medical input was the tunnelling from the Bog Meadows to the river Lagan. Most of the tunnellers employed had previous experience on the Silent Valley undertaking and travelled to and from work. There were occasions when anxious to get home they cut short the decompression process and by the time they reached Ballynahinch, felt the "bends", necessitating their return to the pressure chamber. My recollection is that a case came to court in which a tunneller, having developed neurological symptoms, attributed them to the nature of his work. These are only illustrations

of the rich variety of experiences which fall to doctors who have an interest in and a care for the work which their fellows undertake. To some extent gross excesses in faulty working environments have disappeared — especially in large scale industry but occupational diseases are not quite a thing of the past. They still occur from time to time in the many largely unsupervised factories and workshops where a substantial number of the workforce is still employed.

Occupational health physicians have now become familiar with work induced stress, with musculo-skeletal problems, with exposures which might affect behaviour or reproduction but the subject most discussed at present when they meet is the "sick building syndrome". This is the phrase used to describe that group of conditions brought about by air conditioning, visual display units, miniaturisation of assembly units and the effects generally of modern technology. In the immediate post-war years industry was thriving and the number of companies in Great Britain enlisting the advice of medical men greatly increased. Unfortunately (for whatever reason is debatable), absenteeism, especially absenteeism attributed to sickness, became a serious problem and managements looked to their medical advisors for help. It was some time before it was recognised that this was essentially a management problem and that the role of the industrial health physician in it was minimal although of course he was involved. In the meantime a number of doctors on a part time basis had been engaged in various undertakings to advise on this problem, and their appointments were continued in many cases even when it was realised that the solution lay mainly with personnel management. About the same time a not unrelated problem, that of stress occurring mainly just below board level, was causing anxiety, and numerous schemes for routine examinations of senior staff were inaugurated without much thought of their value. There is no doubt however that the full time occupational health physician fully acquainted with his colleagues and their work, on an informal basis and with ready access to senior management, proved to be useful. It should be mentioned too that a large company (Short's) with several hundred staff, pilots and technicians at any one time scattered over the world had a concern for their health and wellbeing. The responsibility for these and the implementation of an immunisation programme fell to the occupational health physician.

By this time I had left general practice and was engaged fully in the practice of occupational medicine, and Dr Fletcher was at Harland and Wolff. As early as 1935 in Great Britain a small group of full time medical officers had formed an association which met on a quarterly routine at the London School of Hygiene and Tropical Medicine, which I was invited to join in 1947. In the early 1950s a group of this rapidly growing association was formed in Northern Ireland composed of part time practitioners. Lord Nuffield in these years was distributing his massive benefactions to various medical interests — guided largely by the eminent London surgeon, Sir Ernest Rock Carling. Nuffield proposed to endow two university chairs of Industrial Health. This aroused a bitter controversy in which the medical officers of health, who by the inauguration of the National Health Service had lost control of the Poor Law hospitals, claimed that industrial health was part of their province. There was a suggestion too that a national industrial medical service be set up and integrated with the National Health Service. A committee, presided over by Judge Dale, recommended a "wait and

see" policy suggesting that until the lines of natural evolution emerged it was premature to make a decision. Eventually the chairs were established — RE Lane in Manchester and RC Browne in Newcastle. At the same time the Conjoint Board of the Royal Colleges, and the Society of Apothecaries, announced their plans for the award, following examination, of a Diploma of Industrial Health (DIH). After study in Manchester I took the diploma of the Conjoint Board and shortly afterwards gained an MD (Belfast) for a thesis on accident proneness. This led to an invitation to read in 1955 the Milroy Lectures to the Royal College of Physicians of London.[5] Subsequently, I read the Scott-Heron lecture[3] which led, after further investigation by the Department of Social Medicine QUB under Professor John Pemberton, to the definition of byssinosis being extended to include flax and hemp dusts (1965). In 1970 I read the BMA Mackenzie lecture[6] to a meeting of the Society of Occupational Medicine (successor to the Association of Industrial Medical Officers) in Dublin.

During the decade 1950–59 and succeeding years there was agitation stimulated by the trade unions that the benefits accruing to large scale industry able to employ full time doctors should become available to industrial workers generally. This presented difficulties as over 80% of all workers were employed in factories with a workforce of less than 500, where the employment of a full time doctor was neither necessary nor feasible. With encouragement from the Nuffield Trust an experimental Industrial Health Centre was established in a huge industrial estate in Slough (Buckinghamshire) — staffed by a number of full time doctors and nurses with a mobile dressing station which daily visited the factories associated with the scheme. At the invitation of Rock Carling I was invited to join the Slough Industrial Health Advisory Committee to share my experience of working in Belfast. Some years later a similar invitation from the Minister of Labour, Barbara Castle, to join the Ministry of Labour's Industrial Health Advisory Committee was accepted — as was the renewal by her successor, Ray Gunther. For some years I sat too on the British Medical Association Occupational Health Committee so it was no surprise that what was happening in Northern Ireland evinced considerable interest amongst colleagues in Britain. In 1963 the Association of Industrial Medical Officers (shortly to be re-named Society of Occupational Medicine) held a very successful four day meeting in Belfast. In 1967 I was elected its president.

During all these years the relationship of a multiplicity of industrial medical services was a source of controversy on many grounds but so far as the medical staff were concerned the two most important were the absence of a career structure and the lack of provision generally for education in what was coming to be recognised as a new discipline. It had become clear that the experience of the Slough experiment could be applied only to compact industrial estates, and the lesson of other experimental units (notably at Harlow) pointed the way to the employment of part time general practitioners under the leadership of a doctor experienced in the specialty. In Northern Ireland about fifty such appointments were made — some without much regard to competence or experience in the environmental aspects of the work. Although Belfast was and remains an important industrial centre, the university paid scant regard to the needs of the factory workers as such. In the Department of Social Medicine a few lectures were given to undergraduates each year and the small number of post-graduate

students for the Diploma in Public Health (DPH) examination were exposed to some aspects of occupational medicine. A few physicians in their ward rounds and teaching generally drew attention to occupational aspects of the subjects under discussion but in the main the local meetings of the Society of Occupational Medicine were the means by which the educational process was conducted, amongst those who had joined the society.

Professor Owen Wade and Professor Peter Elmes too evinced considerable interest in research in industrial health subjects. The aggregation of large numbers of work people where good records are maintained provide research workers with material for scrutiny. During the war Short's co-operated with the Medical Research Council in an investigation of the value of giving vitamins A & D in maintaining health, and later in the use of anti-flu vaccines. Wade and Elmes later worked there too on a series of men whose medical histories met the criteria to justify a diagnosis of chronic bronchitis — and in a controlled observation over a number of years charted the response to the daily exhibition of antibiotics. Meanwhile some full time appointments were being made — notably Dr Jack Stutt by Belfast Corporation and later Dr Rory L Carson by Du Pont in Londonderry — Carson having had experience of public health work. Two general practitioners who were active during these years were Dr William Colquhoun of Dunmurry and Dr Trevor Hamilton who had an interest in Mackies and the Great Northern Railway.

One of the difficulties experienced by the exclusion of the occupational health service from the National Health Service was that whereas trainees in the NHS were indirectly employed by the state while being trained, apart from a few large companies which could afford a junior doctor and give time for study, no specific provision was available for the large number of part time occupational physicians. However, in 1976 the Royal College of Physicians of Ireland established a Faculty of Occupational Medicine and for the purpose of accreditation as a specialist, the Board of the Faculty was recognised as advisor to the Accreditation Board. Two years later the London College also established a similar Faculty and the medical centre of Shorts was recognised by the JCHMT as a teaching appointment — the only one in Ireland. In passing it may not be inappropriate to suggest that the apprenticeship system in the absence of a formal academic programme is not without its value. Each of the young doctors who served in Shorts demonstrated this — one became an associate professor of occupational medicine in British Columbia, another after developing an interest in contact dermatitis became professor of dermatology in Melbourne, another was awarded a personal chair in epidemiology before becoming a vice-chancellor and yet another became professor of Army Health at Millbank. The man who succeeded me in Short's not only developed the service he inherited but has been making a considerable contribution to the community in general as chairman of the Northern Ireland Youth Committee and vice-chairman of the Police Authority.

The problem of providing educational facilities has to some extent been met by a Distance Learning course based on the Department of Occupational Medicine in Manchester. From this source some fifty specially prepared manuals are sent out at monthly intervals to individuals taking the course — in the main mostly paid for by the employer. In any one area the students meet from time to time and by means of a telephone link with an authority discuss the subject under review.

There is a local tutor in each area to supervise and organise the scheme. In Northern Ireland (at present, 1987) Dr Gerald Hall acts in this capacity.

Stimulus to these developments had been given by a number of appointments to statutory bodies and other large establishments — Dr WA Eakins to the Post Office, Dr Raymond Pritchard to the Northern Ireland Electricity Service, Dr James Sweetnam for a few years to succeed Fletcher at Harland and Wolff, (several medical advisors joined and left the shipyard within a few years), Dr Ben Bolton to the Civil Service and later, and perhaps most significantly, two full time medical advisors to the Royal Ulster Constabulary — the first such appointments to a police force in the British Isles. The appointment of Dr David McLean as occupational health advisor to Queen's University threw an interesting light on the occasional anomalous situations in which a physician may find himself. He was one of a number of doctors working in the Student Health Service. Over a number of years persons in the employment of the university found it convenient to use the service and registered as National Health Service patients with it. Occasions arose when an employee of the university gave medical reasons for his failure to carry out his duties. The personnel manager applied to the Student Health Service for advice but was unable to gain it as its staff was in a confidential relationship with the employee. Following consideration of the problem, and the effect of industrial legislation regarding safety at work which was made applicable to organisations other than industrial, a university occupational health service was set up.

Since about 1960 the use of artificial glues, resins and the manufacture and use of plastics has become commonplace in industry, giving rise to a number of skin, respiratory and other toxicological problems — notably so far as Northern Ireland is concerned in the aircraft industry. The evolution of isocyanates, giving rise to acute and long term respiratory embarrassment in particular, led to the introduction of routine respiratory efficiency testing on entry and therafter at yearly intervals, or at other times where considered necessary. During this period too, actions at law for alleged noise induced deafness led to the institution of audiometric monitoring, increased efforts to reduce noise levels and insistence on the use of hearing protection. The use of X-rays and radioisotopes especially for the inspection of aircraft parts and joints added new responsibilities which were shared by physicists and others. Another function which has recently fallen into the lap of the occupational health physician is that of advising on the preparation of Data Sheets. As a member of the Health and Safety Committee of British Petroleum, with others the company requires us to prepare information for the customer as to the hazard of using each and every one of its products and the precautions necessary to minimise or avoid risk. It will be seen that those who propose to enter the field of occupational medicine must prepare themselves to accept the impositions of these burdens.

The largest employer of labour in Northern Ireland is the National Health Service and until recently apart from the doctors who cared for the nurses in residence, no account was taken of other aspects of health care. Some years ago discussions foundered when the matrons on the informal committee set up by the Ministry of Health at Stormont insisted that they were acting *in loco parentis* to the nursing probationers, and that therefore they must have access to occupational health records. This demand proved to be unacceptable. The structure of hospital

management having changed in recent years, an occupational health service for the NHS is now (1987) in process of being formed. Almost inevitably difficulties have arisen as to the status of the occupational health physician, *vis-a-vis* the consultant staff in any particular hospital. Occupational medicine is a multi-disciplinary subject and there are on the staff already doctors and others who can speak authoritatively on almost every aspect of health care — immunisation programmes, radiation hazards and their use. With goodwill these difficulties will be overcome as others in different spheres have been resolved.

From my earliest days in industry the function of the doctor was to help provide and maintain the health and welfare of people at work. In the beginning and especially since the second world war much emphasis was put on trying to fit the worker to the job especially in the case of partially disabled persons. (On the few occasions on which students came to see the working of our occupational health unit we brought them to see two machinists who had been blinded by drinking methyl alcohol left by the Germans in a post they had overrun. They were astonished to find totally unsighted men working with machine tools). Under the Disabled Persons Act each company employing more than 25 workers was required to engage a quota (which varied from time to time) of disabled persons — whatever the nature of the disablement. This led to the practice of pre-employment examinations for all to ensure, for example, that candidates with respiratory embarrassment were not subjected to dust or fumes, men with elevated blood pressure were not required to mount cranes or work at heights or inaccessible places, that diabetics were facilitated as to their dietary arrangements, that epileptics were located and supervised in appropriate work places, and that patients on return to work after illness or injury were assisted in their rehabilitation. This latter in the main was achieved by organising concessions from management about times of starting or finishing work, rest periods and occasionally by changing jobs.

We had a duty as well to ensure that the workplace was safe. Safety from mechanical hazards was obviously the responsibility of the engineers but as the medical staff were the first to see the results of accidents and engineers regarded production as their prime aim, it was not long before the medical department staff had a role to play in prevention. Similarly the doctors had the earliest intimation of toxic and other hazards as they manifested themselves in the workforce. This, of course, we recognised as being quite unsatisfactory. However, in our efforts to make early diagnoses and remove those affected from further exposure, much information was gained, especially when the help of hygienists, chemists and physicists was enlisted. It was now possible to determine the levels of dust, fumes and noxious agents generally in the environment. Experience in the workshop and experimentation in the laboratories led to the establishment of threshold limit values (TLV) of a large number of substances used in industry, values which must not be exceeded. The occupational health physician now co-ordinates the work of a team of industrial hygienists, safety officers, physicists, personnel managers and engineers concerned to provide a healthy, happy and safe working environment.

In the half century of my experience the range of responsibilities and interests of occupational health physicians has been enormously extended. With them have come new techniques and scientific disciplines which if used effectively should

remove all but the human element from the hazards of industrial life. In the meantime, as the older risks are being eliminated new ones come to light. The use of agrichemicals in the agriculture industry, the hazards of compression in diving and accidental decompression in high flying, the behaviour of groups of people in varying industrial situations, the substitution of mineral fibres for asbestos, the carcinogenic effects of a host of materials, various kinds of radiation hazards, the total body vibration syndrome are only a few of the subjects which engage the attention of the occupational health physician to-day. But however much he is involved with others in helping to provide and maintain a safe working environment, he has and always will have a day to day personal relationship with those for whom he has responsibility.

This memorandum was prepared by request of Dr JS Logan, Archivist to the Royal Victoria Hospital. Only a minimum of biographical detail was included and only such as seems relevant to the story of the evolution from industrial to occupational medicine in Ulster. It may however be added that in 1985 Dr Smiley was elected to membership of the prestigious Ramazzini club (25 members in Europe, 25 in USA) and that in 1987 the academic respectability of the subject was recognised by the award of an honorary doctorate from the Queen's University of Belfast.

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The development of the examination for membership of the Royal College of General Practitioners

Extracts from the Provost's Address to the Northern Ireland Faculty, 5 October 1987.

I B Moran

THE EARLY YEARS

When the College of General Practitioners was formed in 1952 criteria for Foundation Membership included length of service in general practice and a commitment to continuing education. Membership was somewhat later made available to those whose application was supported by sponsors testifying to the applicant's high standards of practice. Some candidates had to submit to an interview: all had to give an undertaking to uphold and promote the aims of the College.

In 1954 an examination committee was established and eleven years later in 1965 a Court of Examiners was approved and the first examination was held. The aim of the examination was to test the competence of the ordinary general practitioner in his work by assessing his or her knowledge of the details of clinical medicine and the ancillary services.

Five sat and four passed that first examination from the whole of the British Isles. From 1965 – 1967 few candidates sat the examination and the November 1967 examination was cancelled because of lack of support. At the annual general meeting of the College in 1967 it was decided that the normal route to membership would be by examination only. The first compulsory examination for entrance to the College was held in November 1968. There were 32 candidates. In 1969 71 candidates passed and in 1970 67 candidates were successful. In 1970 the MRCGP became a registrable qualification with the General Medical Council.

The great development of the examination coincided with the introduction of vocational training. When vocational trainees became an increasing proportion of candidates the original aim of the examination "to test the competence of the ordinary general practitioner in his work" was revised in 1980 "to assess the knowledge and competence appropriate to the general practitioner on completion of vocational training".

ASSESSMENT OF GENERAL PRACTITIONERS

Assessment is defined as being "measurement by as objective a method as possible of the learning achieved by the learner". There are immense difficulties in devising methods of assessing a general practitioner on completion of vocational training. These difficulties include establishing what is already known by the learner before entering general practice, measuring any change during the training course and defining precisely what has to be learned.

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Terminal assessment, as in the FRCS, has been used for many years as an indicator of higher medical training. In this present decade the MRCGP is being used in a similar way. The great weakness of terminal assessment, from a training point of view, is that if it produces a negative result it is too late to give the individual concerned a chance to take remedial action.

THE DEVELOPING YEARS

The three year vocational training scheme became mandatory for general practice on 16 August 1982. As will be seen from Fig 1, making the examination compulsory for admission to the college had little effect on the rate of increase of college membership; the major influence was the introduction of mandatory vocational training.

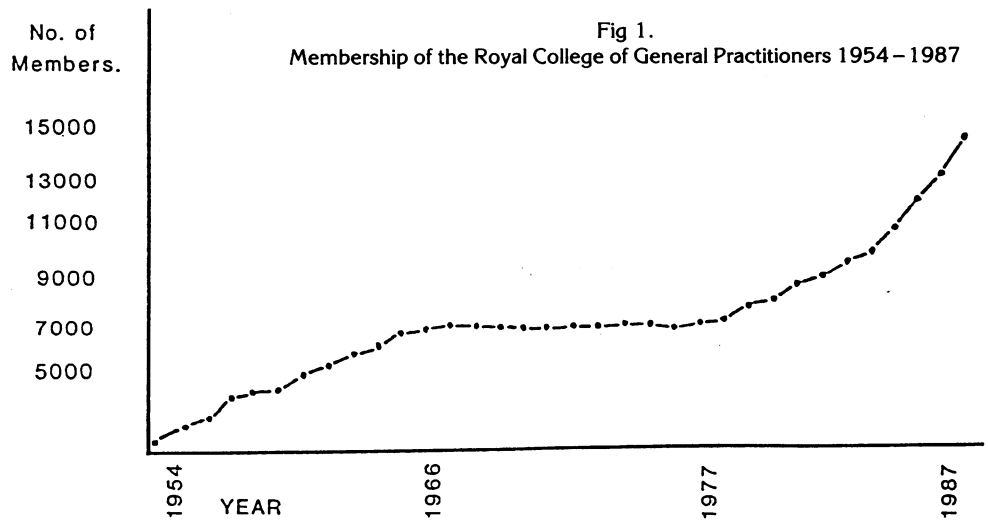

Fig 1.
Membership of the Royal College of General Practitioners 1954–1987

The number of candidates has risen from about 1,000 per year to nearly 2,000 per year since the introduction of vocational training. The pass rate has remained relatively constant at about 70% (Fig 2).

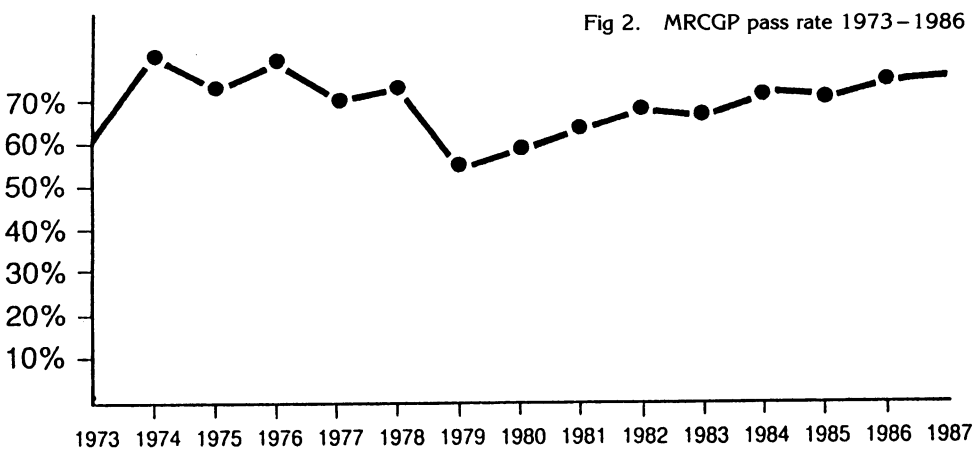

Fig 2. MRCGP pass rate 1973–1986

THE NORTHERN IRELAND SCENE

The number of candidates sitting in Belfast since the first MRCGP written examination was held here in 1971 is shown in Fig 3. The total number is 601 of whom 419 passed, giving an average pass rate of 69% which is about the national average. The top 2·5% of the pass list are awarded distinctions. From 1982–1986 we had 15 candidates who obtained distinction, which is well over twice the national average. Training for general practice became mandatory in 1982 and our selection system became highly competitive.

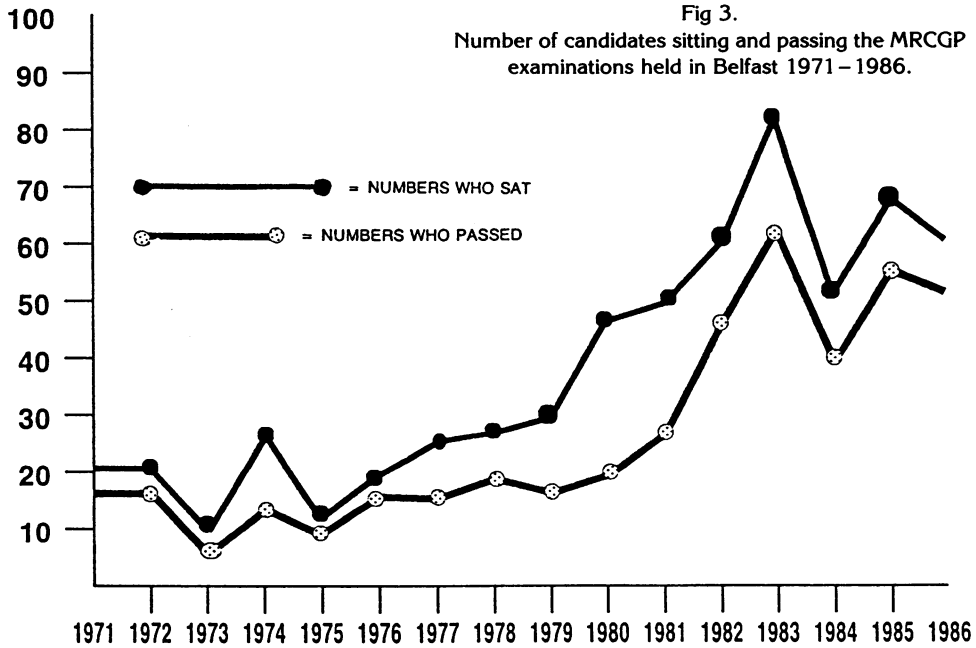

Fig 3.
Number of candidates sitting and passing the MRCGP examinations held in Belfast 1971–1986.

NI GENERAL PRACTITIONERS AND COLLEGE MEMBERSHIP

A previous study [1] recorded the progress of the college in Northern Ireland up to 1978. A comparison of the end point of that study and what has happened in the next eight years is shown in the Table. At the end of 1986 45% of the 33,000 general practitioners in Great Britain were college members. In Northern Ireland 55% of general practitioners who are in active practice are college members.

TABLE

Increases in college membership in Northern Ireland

	All general practitioners in Northern Ireland		*Fellows / Members / Associates of the Royal College of General Practitioners*	
Area Board	*1978*	*1986*	*1978*	*1986*
Eastern	309	381	126	278
Northern	167	202	57	112
Southern	127	171	27	89
Western	114	149	17	61
Total	717	903	227	540

Five points are highlighted by the RCGP examination:

Should the Joint Committee on Postgraduate Training for General Practice have a terminal assessment at the end of vocational training, or should all who complete the mandatory three years training be automatically eligible to practice, even though the MRCGP examination demonstrates that a tiny number of these know very little of what general practice is about? That is a problem for the Joint Committee: it should also be of concern to any general practitioner interested in maintaining standards.

Should passing the membership examination give the right to full membership of the college or should there be a diploma followed by a form of in-practice assessment leading to full membership? In other words should we have a smaller, more exclusive college?

Should there be an alternative route to membership? The present system has lasted since 1968. Should it always continue that way?

Should the college examination continue to be peer referenced as at present or should it be criterion referenced? At present, the college examiners, who are all active in practice, set the examination papers, construct marking schedules and apply them to the candidates' answers. In pairs, they assess the candidates in each of the two orals, judge the value of the answers and mark independently and they then agree a final overall mark. Criterion referencing implies there is a defined core content of general practice. No-one yet has produced such a definition. With the development of general practice in relation to hospital outreach through community paediatrics, and the introduction of psychiatric, diabetic and cardiac nurse specialists, keeping the criteria up-to-date and uniform would also present a problem. A further difficulty with criterion referencing is that if everyone meets the criteria there is 100% pass rate. If everyone does not there is a 100% fail rate. How would medical politicians react to that situation?

Lastly, should the membership examination have a clinical component? Any examination must be both valid and reliable. Validity relates to the extent to which the examination measures objectives appropriate to the tasks. It samples the attributes of the candidate over a whole range of topics. This is more important in general practice than in any other branch of medicine. Reliability relates to the consistency with which similar results are produced when candidates are repeatedly tested using the same technique or when the same characteristic is evaluated by using a variety of problems. The MRCGP examination has achieved a very high level of validity and reliability in assessing the attributes required of a doctor completing vocational training and about to enter practice as a principal.[2] If one introduced a clinical test it should be possible to make it satisfy validity. But what about reliability of the assessment in different centres, such as Edinburgh, Cardiff, Liverpool, London, Birmingham or Belfast, in examining 2,000 candidates per year? A major study has been commissioned by the college to test the reliability of an objective-structured clinical examination and college examiners are eagerly awaiting the outcome of this study.

I am grateful to Mr Tom Dastur, examinations administrator, RCGP, for access to membership data: to my colleagues on the panel of examiners who have taught me so much since I joined them in 1978: to Dr K Connolly, my partner, who has allowed me time off twice yearly to attend orals: to my wife and family who allow me to become a hermit four weekends a year while marking papers.

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The changing role of the physician

Ian Fraser

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THE FORMER GENTLEMAN OF THE PROFESSION HAS NOW BECOME A MANUAL WORKER

The Association of Physicians of Great Britain and Ireland had a very successful meeting recently in Belfast under the presidency of Dr John Weaver. This was its third visit to this town in its 88 years of existence. On the first occasion 61 years ago the president was James A Lindsay, at that time Professor of Medicine at Queen's University, a great admirer of Sir William Osler, the founder of the Association.

On the first occasion I was a young surgical registrar struggling to get a Fellowship; on the last occasion a very much retired old man. Although only on the touchline on both occasions I have been able to see the changes that have taken place in the role of the physician. The physician has changed from frock coat to shirt sleeves. In the old days one did not need to make an immediate diagnosis; Wednesday week would be good enough. When I was a resident pupil in the Royal Victoria Hospital under Professor Lindsay, treatment was uncomplicated. Medicines were simple — "give Pot Iod and trust to God". I suppose this was chemotherapy with alternative medicine added — "arsenic to cure all ills — has a tincture and two pills". This was very reassuring. "Tinct Ferri Perchlor" was given for almost everything. I remember my father, ill in bed with erysipelas, was prescribed "Tinct Ferri Perchlor"; with my help he washed it down the hand basin. At about the same time on a surgical round with Professor Sinclair I remember a patient presenting with a large abscess. The professor said "I am afraid this will require steel (iron) in the surgical sense; he meant a deep incision with a scalpel instead of the tincture.

When I read somewhere recently that surgery has become safe and that medicine is now so dangerous, it made me think of the changing role of the physician. He is certainly a different man to the chief whom I knew 60 years ago. We all get a small envelope from time to time which says at the bottom "Safety in Medicine". This of course is a misnomer and it should read "Danger in Medicine". None of us surgeons ever get an envelope which says "Danger in Surgery: give up varicose veins, etc."

When I was a student the physician carried hidden away a stethoscope and a thermometer, and across his waistcoat was a gold watch-chain with a bauble for playing with. The maximum of manual work that he ever did was to boil two test tubes full of Fehling's solution. When hot they were added together. The mixture changed, taking on the colour of either the Labour or the Conservative party — one was bad and the other good. He used to boil the top inch of a test-tube full of

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urine — it must be full to the top; this required the patient's co-operation. If it went solid with heat, like the white of an egg, the patient had albumin — Bright's disease — that was bad. A test meal was carried out regularly. The unfortunate patient had been starved and with no breakfast was feeling hungry. A Ryle's tube was passed, any residual fluid in his stomach was aspirated and kept: he was then given a delightful dish of gruel or porridge — food at last — but every 15–20 minutes some of this was removed. As students we were very glad if the patient had rapid stomach emptying as this meant the job was over in 1½ hours, but it could at times take anything up to 2½–3 hours. Often we wished that Johnny Ryle had never been invented. All of this was done in a gentlemanly manner; the physician never soiled his hands — the surgeon was the manual worker. The surgeon of that period was doing his operations on the kitchen table, perhaps in a farm house, or else in hospital in his shirt sleeves. Although the patient usually had no unpleasant memories of the actual operation, they all remembered for the rest of their lives being suffocated with the ether and chloroform mixture. It was such an incident that made Lord Nuffield found his Chair of Anaesthesia; he said he never forgot his first anaesthetic. Today that has all changed.

Today the surgeon walks in and lays his warm, comforting, competent, confident hand on the patient's "timid tum". The patient immediately feels safe, happy, relaxed, in fact all is well. The physician on the other hand, formerly such a nice kind chap, comes in and lifts a patellar hammer. He bangs the patient's leg and says to himself "where will this hurt most?". It is obviously the knee, because here the bone is covered only by a thin layer of skin. It has no protective cushion of muscle. He delivers three or four brutal blows. It takes three or four because as a rule he misses the target on the first occasion. If the patient gives a kick the physician knows that the patient is living, and so he now attacks that wiry-looking tendon behind the ankle. It is indeed in an awkward position, he often misses it and hits the couch or bed. In golf one would suggest that it is an awkward lie, and one would take either a niblick or a mashie — I must apologise: these nice names no longer exist. It is now a No 5 or a No 9. No one today even knows what a cleek was.

However, I must get back to the heel tendon. I am sure Mrs Achilles, when she got her young son and dipped him in the River Styx to give his body full protection, would not, knowing what goes on today, have held him by this tendon in the water to save him from being swept away downstream. She knew that this area was not protected, but she felt that this did not matter as her son could never, as a brave soldier facing the enemy, possibly get an arrow in that area behind the heel. However, like many mothers, she did not know what was going to happen. She certainly could not have anticipated that this unprotected area was going to be the daily target of the up-to-date modern physician. Had she known this she would certainly have dipped her son in the water by some other method. If we go back to this unexpected onslaught with the patellar hammer, just think what would happen if the police did that to any person under investigation. The policeman would be up for assault and battery and be taken before the 'Beak' in the morning. I suppose for protection the police could take out an MDU policy or else get an honorary FRCP to protect themselves.

Going back to the Fehling's solution, with the trouble and expense of boiling urine, this is all now done away with. A "dip stick" — I thought in my ignorance that a dip stick was something we used in the motor car to see if we were running short of oil, but not so. A "dip stick" is a mini totem pole. It tests everything, you

do not even need to think. It is hard to take this all in. I am also astonished what can be learned by amnio-centesis about a child whom you have never seen. The students tell me a drop of this fluid can tell you if the child has got hair on his chest, or not — not that it matters — much.

The cold hand on the fevered brow has gone. I am told that this is now replaced with a piece of inanimate sticking plaster impregnated with something so that one can see the patient's temperature at a glance. Taking the patient's temperature has changed greatly over the last 400 years. It started as a breathaliser, hot air into cold water: later a massive object was produced, too large for the mouth. It was about the size of a duelling pistol, in fact much the same shape, the bulbous part fitted into the axilla with the stem standing up vertically where it could be easily read *in situ.* The physician carried two of these in a box not unlike a brace of duelling pistols. Things have changed now. A small glass rod is stuck into the mouth while the nurse holds the patient's wrist. This is usually the only physical contact made with the patient. It has been said that this allows the patient to realise that the staff is really human after all. But surely this all must change. If a helicopter 1,000 feet up can tell the temperature of the ground below, surely a small nipple in the ceiling above the patient's bed could record the temperature on a machine on the Sister's desk, or even at GHQ in the Matron's office. Possibly the nurse could from her desk with a small hand machine focus the beam on each patient and so get the result without having to rise from her chair. This would save a lot of nurse man-hours. It is just a suggestion, but it is bound to come — sadly.

The patients now wear a wrist band with their name on it. In my day we knew the name of the patient, but even that is not quite true. The patient was often "a very good gall bladder third to the right" or "a nice Paget at the end of the ward". The disease had a name but the patient might be anonymous. I do object greatly to the M/s nonsense. You are either a Miss or a Mrs; you cannot be both. If this goes on we will soon be describing the patient on the wrist band with the old logo ♀ or ♂. We have been tagging dogs and cats for years with a collar with 'Fido' or something else on it and even pigeons have had a ring round their leg, but I never thought the time would come when we would have had to tag the humans. Perhaps, however, it would be better to be Mrs Annie Brown than, I am ashamed to say, it often was, "that lovely case of gangrene on the right". A patient does not usually like to be described as a case of good clinical material.

I think it was the discovery of the endoscope that produced the changes in the physician. He started to look down people's windpipes and then their gullets and then attacked them from the other end — something that was at one time reserved for the surgeons as their privilege, and theirs only. It speaks well for the surgeon that he has accepted this poaching into his field without any fuss. This would never have been allowed in our sister profession, the coal miners, where inflexibility is the key word. Nor indeed at Dagenham, where I am told the rule is 'one man, one screw'.

This takeover by the physician is very serious. They have virtually collared the peptic ulcer market. This is sad, since the surgeons have had for some years great fun with the vagus nerve, mutilating it in many different ways. The latest thing, indeed the last straw, is now to find that the physician has started to dissolve gall stones. It was indeed on the proceeds of the peptic ulcer and gall stones that the average surgeon could send his boys to Eton and Harrow, and also keep a small house in the Dordogne. These operations were the surgeons' bread and butter. I can see if this goes on that the average surgeon will soon be as hard up as the

physician. Today a physician thinks nothing of putting a catheter and a balloon into the coronary artery, something that would have been looked on as bad taste in my day.

The physicians have also led the radiologists astray. These nice people, at one time a branch of the photographers' union, are now breaking up kidney stones, again taking bread from the mouth of the surgeon. I watched them over in England doing this the other day with a machine, the name of which I could not pronounce. It sounded something like lithotripsy, in fact it sounded rather like striptease but not quite. A very worrying advance I read about in the *Journal* recently was that they hoped soon to have a mobile machine which could go round the country dealing with renal stones all over the place. One can easily see what is going to happen. The natural sequence is that this machine will appear as a side show in any country fair, in a large 'striptease' wagon. You can go to one tent to have your hand read, than off to the shooting range, then perhaps for a hamburger at another, and then get into the wagon and get your kidney stone cracked to bits. This indeed is the final straw for the penniless surgeon.

Whilst I am writing this a letter has come through the letter box. It has described the no touch technique of treating piles with the CO_2 laser. It is marvellous the post-graduate extra-mural teaching which the old retired folk now get. It is a sort of medical open university sponsored by the drug firms. I suppose you learn a lot from them. They can be divided into several groups. The old hat redressed: the impossible, not even understandable: and the DIY which is meant to make the difficult easy, but often is the reverse. The CO_2 laser for piles says it is a no touch technique. In food stores we often see a note — untouched by hand. I suppose this will soon apply to the hospital patient. The advertisement then goes on to say that it offers a "state-of-the-art solution to the problem". This is an overstatement as 60 years ago I was making my own CO_2 snow, making a fine pencil of it, to treat naevi in children. I got a cure, but did not know that many of these naevi often disappeared spontaneously. I suppose Ambroise Parré would have said, and very truly, "I dressed him, God cured him". If we take this no touch technique to its logical conclusion the time could come when the patient could be put on the moving belt with the affected area correctly exposed and the beam directed on the target for the correct time, the patient falling off the belt at the far end fully cured. We see this in bottling factories where the cap is put on accurately by some machine. No doctor will be needed; it looks in time as if no patient will be needed either. I do find it hard to keep up with modern progress.

Although on one hand the profession seems to glory in no touch technique, still the old stethoscope continues to exist. What a stupid name; it is not a scope, it is really a hearing aid. It has not changed much in the last 160 years. It started originally as a tubular piece of rolled paper linking the doctor's ear to the patient's chest. Later this was changed to a short straight hollow wooden tube and later, with the invention and discovery of rubber, the modern machine evolved. Although called modern it is the one and only medical tool that has remained unchanged over the years. It is a period piece. No patient will go away satisfied if he has not been asked to cough and say ninety nine. It is a sort of ritual like amen at the end of a church hymn. I was interested to see, when I was a student many years ago in Paris, that they did not use ninety nine but asked the patient to say 60–61–62–63–64 etc (of course in French). The length of the rubber tube is interesting. It started with the idea that a long tube would give the doctor some protection against the patient's fauna and flora. The students actually will tell you

that the length must exceed the distance that a super flea could hop, in fact an Olympic medal winning flea. Talking of fleas, it is interesting to see these are now an endangered species. Soon there will be a flea protection society as there is now for bats, badgers, hedgehogs etc. In the old days I had a large morning outpatient list in the Childrens Hospital. There were the usual named days in the week, but there was always one called "Flea Day". I was a carrier. The flea found something more succulent in the other members of the household. I was very unpopular. I suppose if I could have been bitten like the others I might have got away with it, but as it was there was great jealousy that I always remained untouched. It is sad that no longer in the case of undiagnosed pink rash can we confuse shingles with a flea bite. Why has our old friend the flea gone and where has he gone to?

I am always sad that the profession is no longer paid in guineas. To accept some guineas from a patient you felt you were doing them an honour in accepting a fee. But today with modern coinage you do not feel much different from the plumber or the butcher. Perhaps as a surgeon I should have chosen a different simile. The great advantage of the guinea certainly in dealing with farmers was that you give the shillings back as a 'luck penny'. No farmer ever sold a cow or sheep without the exchange of a 'luck penny'. After getting his hernia done, a 'luck penny' always gave him the confidence that his hernia would not come back — too soon.

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Case report

Acute epiglottitis: a case cluster

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Acute epiglottitis is most commonly recognised in children as an infection with *H. influenzae* and may be a severe and rapidly fatal disease. Less commonly, adults, compromised by chronic bronchitis, may develop acute epiglottitis with *H. influenzae* or with a variety of other organisms including *Streptococcus pneumoniae, Streptococcus pyogenes, Staphylococcus aureus,*[1] beta-haemolytic group C Streptococci[2] or *Klebsiella* spp.[3] The peak incidence in children is 2 – 6 years of age, due to the fact that infants receive passive immunity to *H. influenzae in utero* which decreases after birth. Susceptibility to infection then increases until a natural immunity to the micro-organism is developed later in childhood. There may be a seasonal predominance in the winter months. The main focus of infection is the supraglottic area, but the acute phase of the infection is more generalised, producing a state of toxaemia. The presentation is usually of sudden onset with dyspnoea, stridor, and profuse drooling. The severity of the infection usually requires immediate medical attention. Prompt treatment is directed towards relief of any airway obstruction and the eradication of the aetiological agent by the use of appropriate antibiotics.

PATIENTS

A cluster of four patients with acute epiglottitis presented to the Royal Belfast Hospital for Sick Children between September and December 1985. Three were less than 21 months and one was five years of age. Three were female and one male. All lived within 20 miles of the hospital. They were all previously healthy and had normal developmental milestones. They presented with sudden onset of inspiratory stridor, which in two cases followed a reported upper respiratory tract infection. The diagnosis of acute epiglottitis rather than laryngtracheitis was suspected due to the severity of presentation. All patients had pyrexia (up to 40°C), raised leucocyte count (up to 27 $\times$ $10^9/l$), and increasing, severe stridor. One patient had a history of penicillin allergy. Diagnosis was confirmed clinically at intubation and confirmed microbiologically by the isolation of *H. influenzae* from blood culture. All patients responded to chloramphenicol within five days with no complications. One patient received ampicillin in addition to chloramphenicol.

ORGANISMS

The organisms were all isolated from blood cultures using an automated radiometric blood culture detection system (Bactec) on chocolate blood agar

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in 5–10% CO_2. Identification was based on X (Haemin) and V (Nicotinamide Adenine Dinucleotide) growth factor utilisation on nutrient agar. Slide agglutination (Wellcome Ltd) showed the strains to be Pittman serotype b. Antibiotic susceptibility testing was by disc diffusion using the Stokes method[4] with the Oxford Staphylococcus NCTC 6571 as control. Minimum inhibitory concentrations were obtained in broth dilutions of antibiotics using inocula of 10^8 colony forming units/litre. Beta-lactamase production was detected using a chromogenic nitrocefin test.

All the isolates were sensitive to chloramphenicol (10 µg disc). Two isolates were resistant to ampicillin (10 µg). In one of these beta-lactamase production was demonstrated, but the other ampicillin resistant strain was not a beta-lactamase producer. Both had minimum inhibitory concentrations >8 µg/ml to ampicillin.

DISCUSSION

A cluster of four cases occurring in a three month period was remarkable as the frequency of cases brought to the attention of bacteriologists through positive blood culture is much lower. Most series report an incidence of about one per thousand paediatric admissions.[5] Incidence rates are erratic which may reflect the cluster-like occurrences over time. There was no reason to suppose that these cases were epidemiologically related.

Of *H. influenzae* isolates in Northern Ireland, 9·3% are resistant to ampicillin due to production of beta-lactamase.[6] One of the strains reported was ampicillin resistant but not a beta-lactamase producer. This form of resistance is uncommon and is probably due to permeability barriers or structural alterations in penicillin binding proteins. A much lower incidence of chloramphenicol resistance, 1·7% has been reported in a United Kingdom survey,[7] and this is therefore the drug of choice in life-threatening *H. influenzae* infection such as epiglottitis. Chloramphenicol is also more rapidly bactericidal than ampicillin against *H. influenzae.* Indeed even those rare strains producing enzymes which inactivate chloramphenicol by transacetylation have been successfully treated with high doses of chloramphenicol.[8] High initial doses of 100 mg/kg/day should be used, which can be reduced later when quantitative susceptibility tests are available and a clinical response is shown. Chloramphenicol assays of trough (pre-dose) and peak (1 hr post dose) serum concentrations should be performed to determine maintenance intravenous dosage aimed at a peak serum level of 15–25 mg/l, as there are large variations in pharmacokinetic responses due to age, liver enzyme induction, and protein binding displacement. A peak at two hours post dose should be taken when using oral administration. Marrow toxicity may also be anticipated by reticulocyte, haemoglobin, neutrophil, and platelet counts.[9] Some of the newer cephalosporins such as cefuroxime and cefotaxime which show good in vitro activity against *H. influenzae* have been used in the treatment of *H. influenzae* meningitis and epigolottitis,[10] and provide a suitable therapeutic alternative to chloramphenicol in epigolottitis without the potential problems of associated toxicity.

Household contacts under the age of six have a 500 times increased risk of *H. influenzae* infection,[11] and rifampicin prophylaxis should be considered in this risk group. Pharyngeal colonisation of contacts is reported to be up to 70%, so that culture from contacts is not helpful in identifying those at risk. In the United States of America, familial spread has more commonly been reported and antibiotic prophylaxis of close contacts is recommended. In this country the

incidence and rate of spread is much lower and the debate on antibiotic prophylaxis of contacts has not yet been resolved: there is, however, a reasoned trend towards antibiotic prophylaxis for close contacts of acute cases.

Bacterial capsular polysaccharide vaccines are now widely used in the United States of America at 24 months of age, and some studies report significant reductions in the incidence of *H. influenzae* type b infection in children over 24 months.[12] Protein polysaccharide conjugate vaccines are being investigated with the hope of improved protection to include children less than 24 months who are at greater risk from life-threatening infection.

We wish to thank Professor J Dodge, Dr S Keilty and Dr A Redmond for permission to report these cases.

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Case report

Laparostomy in acute pancreatitis

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Acute necrotizing pancreatitis is a severe disease with a mortality often greater than 50 per cent. Partial or total pancreatectomy in this condition is a major surgical procedure with a hospital mortality in excess of 40 per cent. Recently there has been a move towards limited resection and open drainage. We report a case which illustrates the new technique in the treatment of necrotizing pancreatitis of necrosectomy (local debridement of obviously necrotic tissue leaving the intervening recoverable tissue) and laparostomy (leaving the abdominal cavity open, to heal by granulation).

CASE HISTORY

A 36-year-old seaman was admitted with a twenty-four hour history of severe epigastric pain with vomiting. Two similar but milder episodes were reported in the recent past. There was no history of dyspepsia. He smoked forty cigarettes and drank ten units of alcohol daily. He was not on any medication.

On examination he was pale, distressed and sweating. His abdomen was rigid with marked tenderness and rebound in the epigastrium. There were no palpable masses and scanty bowel sounds were present. Initial investigations showed a haemoglobin of 17·2 gms/dl, white cell count of $13{\cdot}4 \times 10^9$/l, normal serum urea and electrolytes, serum amylase 341 iu/l, plasma glucose 13·8 mmol/l and an arterial blood pO_2 77 mmHg. X-rays revealed no free sub-diaphragmatic gas, localised ileus or gallstones.

He was taken to theatre on the night of admission because of continuing severe pain and peritonism. In spite of the normal serum amylase, laparotomy revealed pancreatitis with a swollen pancreas and typical "prune juice" peritoneal fluid (amylase content 1570 iu/l). The stomach and duodenum were normal and there were no gallstones. The peritoneal cavity was lavaged with normal saline and then closed.

Initial post-operative management included intravenous fluids, naso-gastric suction, oxygen by facemask and opiate analgesia. Over the following forty-eight hours his condition deteriorated, he developed the adult respiratory distress syndrome and required transfer to the intensive care unit for intubation and ventilation. He had continuing signs of intra-abdominal sepsis, a swinging pyrexia up to 39°C and a palpable abdominal mass. Abdominal CT scan twelve days

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after operation showed a markedly swollen pancreas with focal necrosis and several large peripancreatic fluid collections.

Laparostomy was performed two days later. The pancreas was explored, necrotic areas resected and the peripancreatic abscesses drained. The greater omentum was detached from the stomach, draped over the viscera and sutured to the wound edges inferiorly and laterally. The resulting gap between the stomach and omentum afforded wide access to the lesser sac while protecting the colon and small bowel from evisceration. Three corrugated capillary drains, each 5 cm wide, were inserted behind the stomach and the abdomen only partially closed. A tracheostomy was also carried out to facilitate the necessarily prolonged ventilation.

In the intensive care unit the level of anaesthesia was deepened every forty-eight hours to allow the insertion of a sterile gloved hand deep into the abdomen through the laparostomy (Figure). This allowed adequate breaking down of all loculi in the peripancreatic area. Fluid and necrotic debris were aspirated and the cavity irrigated with saline and tetracycline solution. This regime continued for a further 12 days during which his condition gradually improved. His temperature returned to normal, blood pO_2 rose and he required decreasing inotropic support. After 46 days of respiratory support he returned from the intensive care unit. His only problem was mild hyperglycaemia which was easily controlled with a 2000 calorie diet. Over the following two weeks his drain was removed and repeat CT scan revealed only an oedematous pancreas with no abscess formation. His wound granulated and contracted and he was fit for discharge 72 days after admission. Total alcohol abstinence was strongly advised. On review eight weeks after discharge, he was well, off all alcohol and his wound was much smaller. He will require repair of the muscle defect at a later date.

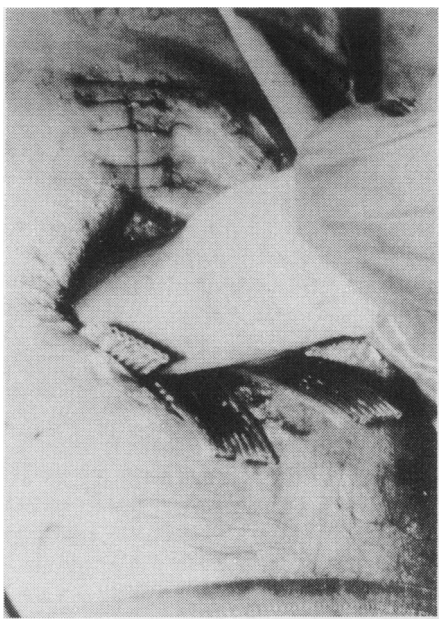

Figure. Insertion of a sterile gloved hand through the laparostomy to break down loculi.

DISCUSSION

Acute pancreatitis encompasses a spectrum of disease. It ranges from interstitial pancreatitis, a self-limiting disease with a low morbidity and mortality to necrotizing pancreatitis with persistent intra-abdominal necrosis and a high frequency of pulmonary, renal, cardiovascular and gastrointestinal complications.[1] Necrotizing pancreatitis occurs in 8–15 per cent of patients with acute pancreatitis and has a mortality rate with conservative management of 50–80 per cent. Indications for surgery in acute pancreatitis include uncertain diagnosis, correction of associated biliary tract disease, progressive clinical deterioration despite maximal supportive care and development of peripancreatic sepsis.[2]

The most controversial aspect is the role of surgery in patients with severe pancreatitis. Operative intervention has been advocated to reduce the high mortality in those who fail to respond to supportive measures in an intensive care unit. Pancreatic drainage was widely used during the early part of the century but fell out of favour.[3] There has been renewed interest in operative drainage over the past fifteen years,[4] sometimes combined with peritoneal lavage, although the latter alone has not been shown to be effective[5] because most of the necrosis is retroperitoneal.

There is controversy over both the timing and extent of pancreatic resection. Previously proposals for timing of operative intervention varied from one to seven days from the onset of symptoms.[6, 7] We believe that operation should be deferred until the second or subsequent weeks of the illness by which time areas of necrotic tissue will be clearly demarcated. Although the criteria of Ranson,[8] Imrie[9] and of McMahon[10] indicate severity and prognosis, they are not accurate pointers to which patients will require surgery. Clinical judgement based on the patient's overall wellbeing, abdominal tenderness and increased swelling, pyrexia, leucocytosis and need for increasing respiratory and inotropic support are the best guides to intervention.

Computerised tomographic scanning with enhancement has a higher sensitivity and specificity than either ultrasound or indium-labelled leucocyte scanning in evaluating the extent of pancreatic and peripancreatic necrosis. It avoids the risk of overlooking a fluid collection which should be drained at laparotomy,[11] but it is not accurate in defining whether infection is present. Fine needle aspiration under ultrasound control with fluid culture is more accurate in diagnosing sepsis.

Recommendations for the extent of resection range from total pancreatectomy to the more limited procedure of necrosectomy. The mortality rate for the major operative procedures varies from 35 per cent after distal pancreatectomy to 40 per cent after subtotal pancreatectomy and 67 per cent after pancreaticoduodenectomy.[12] Beger and colleagues[1] showed in a prospective trial that treatment by necrosectomy and post-operative local lavage reduced their mortality rate from an overall 24 per cent to six per cent.

The treatment of pancreatic abscess is prompt surgical drainage and removal of devitalized tissue followed by irrigation with saline and antibiotic solution. Wide sump drainage, or more recently, open packing of the lesser sac or prolonged post-operative irrigation of the pancreatic bed have been recommended.[13, 14] The mortality of untreated pancreatic abscess approaches 100 per cent. Laparostomy has been used in a few centres in Europe over the last ten years but has not been widely adopted. Mughal and colleagues[15] reported a series of 18 patients with intra-abdominal sepsis treated by laparostomy (including four with pancreatitis). They considered necrotizing pancreatitis to be the only indication for laparostomy as a primary procedure. Some have suggested the use of silastic sheeting or suturing of a zipper to the wound edges to reduce the risk of visceral trauma in laparostomy.[16] In the present case the use of wide corrugated drains placed over the omentum and transverse colon had the triple function of protecting the viscera, facilitating entry to the peripancreatic area as well as providing a drainage route. We believe that this case illustrates a technique which could markedly decrease the mortality rate from necrotizing pancreatitis, and have successfully treated two further patients by necrosectomy and laparostomy.

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Case report

Percutaneous nephrostomy in an unusual case of ureteric obstruction

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Ureteric obstruction from an iliac artery aneurysm is uncommon.[1] We report what we believe to be the first case in which percutaneous nephrostomy was used in the management.

CASE REPORT

A 70-year-old man presented with rigors. He had a history of severe cardiovascular disease. Examination revealed marked congestive cardiac failure, a palpable abdominal aortic aneurysm and a separate pulsatile mass on right-sided rectal palpation. Haemoglobin was 11·1 gm/dl, white cell count 23·7 × 10/1, serum urea 26·2 mmol/l and creatinine 330 mmol/l. Computerised tomography confirmed the presence of an aortic aneurysm and also identified a right internal iliac artery aneurysm which had caused noninflammatory dilatation of the ipsilateral ureter (Fig 1).

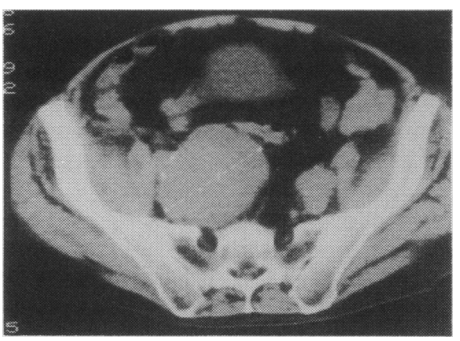

Fig 1. Computerised tomogram of the pelvis showing the right internal iliac artery aneursym.

Excision of the aneurysm was not possible due to the patient's poor cardiovascular condition. A right percutaneous nephrostomy was performed resulting in rapid clinical improvement, serum urea falling to 15·8 mmol/ and creatinine to 285 mmol/l. The pus obtained grew coliform organisms on culture, allowing the appropriate antibiotic to be commenced. It was intended to insert a double J ureteric stent but antegrade pyelography following catheterisation of the right

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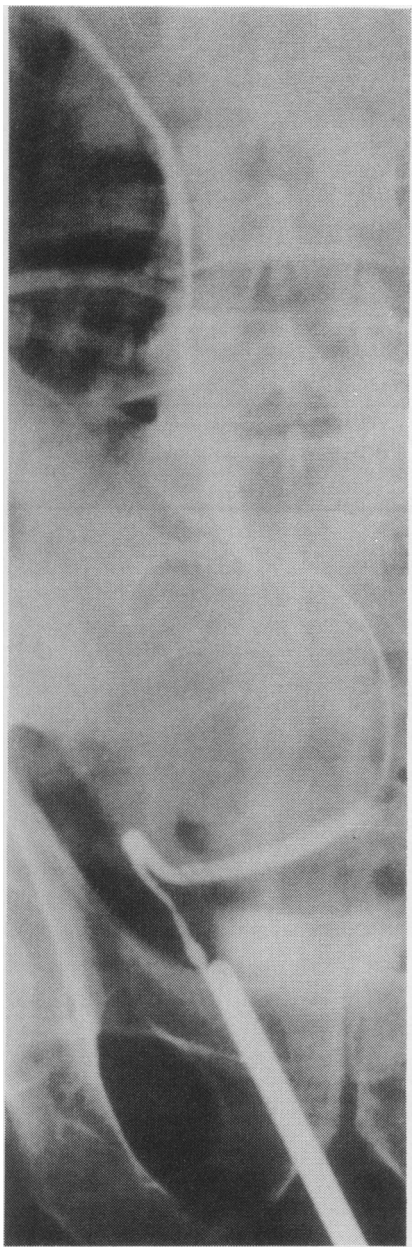

Fig 2. Antegrade pyelogram following ureteric catheterisation revealing acute angulation of the right ureter.

ureter (Fig 2) revealed acute angulation of the ureter, making it impossible to pass a guide wire from above or below. A percutaneous pigtail catheter was therefore left in place. During the following five weeks, despite the improved renal function, the patient suffered two further myocardial infarctions, the second proving fatal.

DISCUSSION

Iliac artery aneurysms occur one tenth as frequently as aortic aneurysms.[2] Ureteric obstruction can be caused by compression, reactive fibrosis secondary to atherosclerosis, or inflammatory aneurysmal disease similar to that seen in retroperitoneal fibrosis.[3] Only 10 cases of noninflammatory ureteric obstruction from internal iliac artery aneurysms have been reported,[1,4,5,6] all of which have been treated surgically. We report what we believe to be the first case in which percutaneous nephrostomy was used in the management.

Fifty percent of iliac artery aneurysms are asymptomatic,[2] but they may be associated with ureteric or bladder neck obstruction,[1,3] rupture into the ureter or rectum,[2] or pyonephrosis.[1] In 20% of cases abdominal examination reveals a pelvic mass or bruit,[2] although a pulsatile, expansile mass is more frequently found on rectal examination.[6,7] In previous reports the diagnosis has been made either at operation or by invasive arteriography or pyelography.[8] The present case shows the effectiveness of CT scanning.

Previous cases have been treated by surgical excision of the aneurysm or by ureterolysis. In our patient, definitive corrective surgery was deferred due to the risk of graft contamination from repair of the aneurysms in the presence of infection, the history of widespread cardiovascular disease and the poor general condition of the patient. Percutaneous nephrostomy, in which a catheter is inserted into the renal pelvis under ultrasonic or fluoroscopic control, allows relief of symptoms until definitive surgery can be performed. Although first introduced in 1955, its potential is only now being realised. Relief of uraemia and septicaemia occurs in over 90% of cases, and failure of catheter placement occurs in less than 3% of

patients; major complications, such as leakage of urine, severe haemorrhage, exacerbation of pyonephrosis or catheter displacement occur in about 4% of cases, although minor complications (wound infections or transient haematuria) occur more commonly.[9, 10]

In this case, percutaneous nephrostomy relieved the pyonephrosis, improved renal function and provided specimens for bacteriology. Although the patient subsequently died from his cardiovascular disease, the symptomatic relief and clinical improvement obtained from percutaneous nephrostomy indicates the effectiveness of the technique in cases of ureteric obstruction in whom definitive surgery is considered inappropriate.

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Case report

Salmonella reactive arthritis in established ankylosing spondylitis

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A patient with previously diagnosed ankylosing spondylitis developed a first episode of peripheral synovitis following enteritis with *Salmonella* group D. Ankylosing spondylitis and reactive arthritis are HLA – B27-related conditions and flare-ups have been linked to alterations in bowel flora. This case report supports recent suggestions that peripheral synovitis in ankylosing spondylitis is a form of reactive arthritis.

CASE HISTORY

A 44-year-old school teacher presented with acute synovitis of both knees and left wrist shortly after returning from holiday with his family. Two weeks earlier, while in Portugal, he had developed mild watery diarrhoea with minimal systemic upset twelve hours after eating barbecued chicken. There was no recent history of urethritis, conjunctivitis or back pain, but his joint complaints were severe enough to keep him from work. Fourteen years previously, he had sought specialist advice because of pain and morning stiffness in the low back and right hip. X-rays revealed bilateral sacroilitis and symmetric syndesmophyte formation in the upper lumbar spine. A diagnosis of ankylosing spondylitis was made and he was initially treated with phenylbutazone. He has subsequently had intermittent exacerbations of low back symptoms responding to short courses of anti-inflammatory drugs, but denied persistent stiffness.

Physical examination four weeks after the onset of diarrhoea revealed painful bilateral knee effusions, worse on the left, and a tender, swollen left wrist. There was loss of the lumbar lordosis but normal lower lumbar movements, and no peripheral enthesitis. He had no skin rashes, mucocutaneous lesions or ocular inflammation. Investigations showed haemoglobin 14·6 g/dl and white cell count $9{\cdot}8 \times 10^9/l$ with normal differential white cell count. ESR and C reactive protein were moderately raised at 38 mm/hr and 31 mg/l respectively. Tests for rheumatoid factor were negative, no autoantibodies were detected in the serum, and tissue typing was positive for HLA – B27. Antibodies to *Yersinia enterocolitica* and *Campylobacter spp.* were not detected. Inflammatous synovial

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fluid was aspirated from the right knee (WCC $10{\cdot}6 \times 10^9/l$) but no organisms or crystals were detected. Stool culture revealed *Salmonella enteritidis* and antibodies to salmonella somatic antigens were detected in high titre (1280). Radiographs of peripheral joints were normal. There was anterior syndesmophytic bridging in the upper lumbar and lower thoracic regions and considerable narrowing of the sacroiliac joints. The patient was treated with bed rest, wrist splint and indomethacin. One month later his symptoms had lessened although he still reported occasional peripheral joint pain. At four months there were no joint complaints but intermittent loose stools persisted, and stool culture remained positive.

DISCUSSION

The pattern of asymmetric, oligoarticular synovitis occurring two weeks after the onset of salmonella enteritis is typical of 'reactive arthritis'. The concept of reactive arthritis is now well established and refers to an aseptic inflammatory peripheral arthritis following infection at a distant site in subjects who usually possess the histocompatibility antigen HLA–B27. Enteric organisms including salmonella, shigella, yersinia and campylobacter are amongst the commonest inciting agents. Reactive arthritis occurs in up to 9·5% of individuals following salmonella outbreaks, most usually with *S. typhimurium* and *S. enteritidis.*[1]

In the present case, reactive arthritis occurred in a patient with pre-existing ankylosing spondylitis. Only two previous reports have documented proven reactive arthritis in such patients,[2,3] but in neither was the diagnosis of ankylosing spondylitis established (by rheumatological consultation and X-ray) years prior to the reactive episode. Peripheral joint synovitis is a recognised feature of ankylosing spondylitis and may run a clinical course indistinguishable from reactive arthritis. Alterations in bowel flora,[4,5] histological features of inflammation in the ileocolic region[6] and alteration in bowel permeability have been linked to episodes of peripheral synovitis in patients with ankylosing spondylitis. It has therefore been postulated that it is a form of reactive arthritis usually arising from sub-clinical infection with an enteric organism which alters bowel permeability and possibly allows penetration of exogenous antigen.[6] An analogous situation may occur in patients with inflammatory bowel disease in whom flare-ups of bowel symptoms accompany episodes of inflammatory peripheral joint synovitis.

The finding of peripheral joint synovotis triggered by salmonella infection in this patient with ankylosing spondylitis lends support to the concept that bowel infections may not only lead to the development of reactive arthritis in previously healthy individuals but may also provoke a flare-up of pre-existing spondyloarthropathy. Recent reports of the therapeutic benefit of sulphasalazine in the treatment of HLA–B27-related arthropathies[8,9] may therefore be due to effects on bowel flora,[8] and, if confirmed, may suggest new therapeutic approaches in reactive arthritis.

We wish to thank Miss H Whyte for typing the manuscript.

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Case report

Spontaneous splenic rupture: a unique presentation of Q fever

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Spontaneous rupture of the spleen is rare but can occur in illnesses which alter normal splenic morphology. These include infections, typically infectious mononucleosis,[1] haematological malignancies,[2] and miscellaneous conditions such as rheumatoid arthritis, sarcoidosis and polyarteritis nodosa. It has not previously been reported in association with Q fever.

CASE REPORT

A 55-year-old female, from a rural area, following a four-day history of 'flu-like illness developed sudden onset severe epigastric pain radiating to her back and left shoulder. On admission to hospital she was hypotensive (blood pressure 80/60 mmHg) with a sinus tachycardia of 110/min. Abdominal examination was unremarkable. Haemoglobin was 10 g/dl, PCV 0·32, WCC 24·3 x 10^3/l, serum urea and electrolytes were normal and the serum amylase 120 Somogyi units. Supine abdominal X-ray showed a soft tissue mass in the left upper quadrant. Despite vigorous resuscitation she remained profoundly shocked.

At emergency laparotomy three litres of blood were found in the peritoneal cavity associated with a major splenic rupture. Splenectomy was performed and because several sizeable splenunculi were seen, no re-implantation of splenic fragments was attempted. Subsequent close questioning of the patient and her relatives failed to establish any preceding history of trauma. The postoperative period was complicated by persistent pyrexia, left-sided pleuritic chest pain and haemoptysis. ESR remained elevated at 55 mm/hr associated with a marked leucocytosis and elevated serum transaminases (AST 251 μ/l, ALT 334 μ/l).

Chest X-ray on admission had shown an area of oval shadowing in the left mid-zone which progressed postoperatively to collapse and consolidation of the left lower lobe. Repeated cultures of sputum and blood were negative. An atypical pneumonia was diagnosed radiologically and the clinical findings resolved with tetracycline therapy. Serological tests on acute and convalescent sera using the complement fixation test (Microtitre system) demonstrated a rising antibody to Q fever phase 2 antigen from $< 1:10$ to 1:80, thus confirming recent infection with *Coxiella burnetti.* Histopathology of the spleen demonstrated

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features of a non-specific infective process without evidence of leukaemic or neoplastic infiltration, connective tissue disorder, emboli or infarctive changes. Four months postoperatively the patient remained well with no evidence of residual Q fever.

DISCUSSION

Spontaneous splenic rupture, first described by Atkinson in 1874[3] is rare. The mechanism is unknown although disruption of the splenic capsule and blood veeesls by disease processes such as leukaemic deposits, microabscesses, rheumatoid arthritis and polyarteritis have been suggested. In such circumstances a sudden increase in portal pressure produced by coughing, vomiting or defaecation may precipitate splenic congestion and rupture. No obvious precipitating event occurred in this patient.

Q fever was first reported in Northern Ireland in 1962.[4] Since then there has been a high incidence of the condition in the province,[5] with 262 cases diagnosed up to 1986 when this case occurred. The condition commonly presents as a mild flu-like illness often associated with pneumonitis, although up to 30% may demonstrate an acute hepatitic picture. A recent Australian report[6] suggests that splenomegaly is present in over 30% of patients particularly if endocarditis is present. This case report is the first of spontaneous splenic rupture.

Correct preoperative diagnosis of this complication is difficult, often being confused with other intra-abdominal catastrophes.[7] The diagnosis should be considered in any patient with an illness known to involve the spleen, who presents with unexplained abdominal pain and cardiovascular collapse. A high index of suspicion should reduce diagnostic delay, accelerate appropriate surgical intervention and reduce mortality.

We would like to thank Dr J H Connolly, Consultant Virologist, and the staff of the Virus Reference Laboratory, the Royal Victoria Hospital for carrying out the serological tests; and Dr J Sloan, Consultant Pathologist, the Royal Victoria Hospital for the histopathology studies on the spleen.

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Case report

Torulopsis glabrata fungaemia

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Opportunistic yeast infections have emerged as an increasing problem in immunocompromised and debilitated patients. Although *Candida albicans* is the yeast most commonly isolated from the body and that which most commonly causes candidiasis, several reports have documented the emergence of *Torulopsis glabrata* as an important pathogen. We describe cases of fungaemia caused by this yeast in compromised patients which illustrate noteworthy predispositions.

CASE 1

A female child with a birth weight of 919 g was born by premature normal delivery at 25 weeks gestation and was transferred to a neonatal intensive care unit. Respiratory distress syndrome developed and mechanical ventilation was commenced. Total parenteral nutrition was administered via peripheral venous cannulae. An eight day prophylactic course of penicillin and gentamicin had been commenced at birth. Following the isolation of coagulase negative staphylococci from peripheral venous blood culture and accompanying signs of clinical sepsis, a further 22 days of flucloxacillin and netilmicin was given. The clinical response was poor despite *in vitro* sensitivity of the micro-organism. Her respiratory secretions were colonised with *Pseudomonas spp.* and when clinical pulmonary infection developed, the drug regimen was changed to ceftazidime. Her clinical deterioration continued and one day later splenomegaly was noted and the platelet count fell further to $80 \times 10^9/1$.

Four sets of blood cultures and urine grew *Torulopsis glabrata* and she was commenced on amphotericin 1 mg/kg/day and flucytosine 80 mg/kg/day. During the next week there was no clinical response and amphotericin and flucytosine doses were progressively increased to 2 mg/kg/day and 160 mg/kg/day respectively. Her clinical condition improved and the platelet count rose steadily to $160 \times 10^9/1$. A total of 21 days antifungal therapy was administered, during which liver and renal function remained within normal limits and she subsequently made satisfactory progress.

CASE 2

A 74-year-old drowsy and dehydrated woman was admitted to a geriatric medical unit with increasing confusion, and urinary incontinence. She had poorly controlled non insulin dependent diabetes mellitus, ischaemic heart disease,

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cerebrovascular disease and mild dementia. The plasma glucose level was 56 mmol/l, white cell count 21 x 10^9/l, and serum urea 26·5 mmol/l. Intensive therapy with insulin, fluids and diuretics was commenced, and oral ampicillin 500 mg four times daily. A urinary catheter was passed to monitor output. She was slow to respond and hyperglycaemia persisted in spite of this treatment. After ten days she developed abdominal distension and signs of subacute obstruction. The antibiotic regimen was changed to cephradine and metronidazole. Direct microscopy of a catheter specimen of urine showed a white cell count >1000 cells/cu mm, and yeasts were seen in large numbers.

The peripheral blood white cell count rose from 17 to 23 x 10^9/l. All antibiotics were stopped and intravenous amphotericin commenced at a dose of 0·3 mg/kg/day which was increased to 0·5 mg/kg/day after two days. *Torulopsis glabrata* was isolated from urine and three sets of blood cultures. There was a slow clinical improvement, accompanied by a slight fall in blood urea and creatinine levels and a normalisation of blood glucose. Amphotericin was continued for 11 days, by which time she was considered clinically to be no longer septicaemic. Catheter specimens of urine were clearer but some yeasts were still seen on microscopy. Unfortunately intractable congestive cardiac failure and right basal pulmonary consolidation led to death 34 days after admission.

CASE 3

A 60-year-old woman was admitted to hospital with a large perforated gastric ulcer. A partial gastrectomy was performed and she was commenced on mezlocillin and metronidazole. Postoperatively the patient was pyrexial, and amoxycillin was substituted for mezlocillin, with netilmicin added. A subclavian central venous catheter and an arterial catheter were inserted and the patient required assisted ventilation in a respiratory intensive care unit. Ten days postoperatively the gastroduodenostomy anastomosis failed and the abdominal wound burst, requiring emergency repair. A tracheostomy was also performed. Over the succeeding eight weeks the clinical course was stormy with a persistently spiking pyrexia, bile drainage, respiratory difficulty, depressed level of consciousness and a progressive flaccid peripheral motor neuropathy.

During this period clavulanate potentiated ampicillin, azlocillin and ceftazidime were successively prescribed while netilmicin and metronidazole were administered almost continuously. *Candida albicans* was first isolated from a catheter specimen of urine four days after admission and was subsequently isolated intermittently from sputum and wound swabs. Morphologically different yeasts, initially reported as "non pathogenic", were first isolated from a wound swab on day 15 and from sputum ten days later. Sixty days after admission her temperature rose to 40°C and two sets of blood cultures grew yeasts which were identified as *Torulopsis glabrata.* Culture of the subclavian catheter tip, removed two days later also yielded this organism. Amphotericin therapy 1·5 mg/kg on alternate days was given. The patient became apyrexic after 24 hours and remained so. Treatment was continued for eight days at which time netilmicin and metronidazole were also stopped. The urinary catheter was removed 12 days later, and she was discharged 21 weeks after admission.

CASE 4

A 42-year-old man presented with small bowel infarction which was treated by resection followed by jejunostomy. Thereafter he was maintained on total parenteral nutrition. He suffered repeated episodes of coagulase negative

staphylococcal infection related to central venous catheters over a period of four months. These were treated by various antibiotics and replacement of the lines. A mild fungaemia in which *Candida albicans* was isolated from blood and catheter culture responded to removal of the catheter without antifungal therapy. A tunnelled venous catheter was inserted to allow continued parenteral feeding. Three days later he became profoundly septicaemic and hypotensive. *Torulopsis glabrata* was isolated from four sets of blood cultures and also from the catheter following its removal. Low dose amphotericin (0·5 mg/kg/day) and full dose flucytosine were commenced. After one week amphotericin dosage was increased gradually to 1·5 mg/kg/day and the patient responded temporarily but continued to show intermittent pyrexia of 38·5°C. Antifungal therapy continued for three weeks after which time he appeared stable and afebrile, but he died from bronchopneumonia two weeks later.

DISCUSSION

Torulopsis berlese, the name first coined by Berlese in 1884 included a number of yeast species. *Torulopsis glabrata* was first recognised by Anderson in 1917 [1] and was thought to be a species of Cryptococcus because it produced no ascospores or hyphae. *T. glabrata* grows on routine bacteriological diagnostic culture media. Unlike *Candida spp.* the genus *Torulopsis* is distinguished by the complete absence of hyphal or pseudohyphal forms under all cultural conditions because it reproduces by budding only. The assimilation of trehalose and glucose but not other sugars confirms the identity,[2] but because of its close antigenic relationship to Candida it is often identified as "Candida species/non albicans" on the basis of germ tube tests. The organism is sensitive to amphotericin and normally also to flucytosine, but it is generally resistant to ketoconazole. Susceptibility to miconazole is variable, up to 40% of strains being resistant.[3]

T. glabrata was formerly considered to be wholly saprophytic but has become increasingly recognised as an opportunistic pathogen in the altered host. Like *Candida spp.* it may be found as a commensal in the gastrointestinal, respiratory and genitourinary tracts of normal individuals. Particularly at risk are those who have undergone major gastrointestinal surgery requiring prolonged intravenous feeding, have had broad spectrum antibiotics, diabetics, or immunocompromised patients.

The spectrum of infection ranges from mild fungaemia to endocarditis,[4] pyelonephritis,[5] pneumonitis,[6] and peritonitis.[7] The compromised host is particularly liable to tissue invasion and metastatic infected foci. Experimental pathogenicity studies on *T. glabrata* have demonstrated that the yeast does not produce a progressive infection in normal mice, but the capacity for tissue invasion was enhanced in steroid-treated, alloxan-diabetic and irradiated mice.[8] Normal human plasma inhibits the growth of *T. glabrata* more than other yeasts *in vitro,* the inhibitory factor being a combination of transferrin, IgM and other unidentified proteins.[9] The clinical presentation is undistinguishable from systemic candidosis, and can vary from an indolent low grade pyrexia to profound septicaemia with shock.

The first case illustrates the pathogenic potential of the yeast in a very pre-term neonate. Multiple invasive procedures, the prescription of broad spectrum antibiotics over a long period along with intravenous feeding enabled the organism to invade systemically. A very high dose antifungal regimen was required to produce a clinical cure. This was tolerated well by the infant, with no

evidence of renal or haematological toxicity. Case two became fungaemic following urinary catheter colonisation. Impaired renal function does not demand a reduction in amphotericin dosage since only 3% is excreted in the urine.[10] However, low dosage regimens are being increasingly used to reduce toxicity, particularly when the drug is used in combination with flucytosine. Since flucytosine is mainly excreted through the kidneys, the addition of this drug is particularly appropriate in treating fungaemia associated with renal tract infection. The regimen has been combined with local instillations of amphotericin into the bladder,[5] but bladder colonisation cannot be eradicated without catheter removal. Poor diabetic control adds to the difficulties of treatment since hyperglycaemia and acidosis interfere with polymorphonuclear phagocytosis.[11]

The third and fourth cases represent typical examples of initial colonization of a central venous catheter and subsequent fungaemia due to the yeast after gastrointestinal surgery, prolonged antibiotic therapy and intravenous feeding. Removal of intravenous catheters and subsequent alternate day treatment with amphotericin alone for 10 days was successful in rapidly eradicating the infection in case three. Case four showed spontaneous recovery following removal of the central catheter, but general deterioration compromised his ability to respond to the subsequent *T. glabrata* septicaemia. Metastatic seeding may have occurred prior to diagnosis or to catheter removal, or to the establishment of therapeutic antifungal serum concentrations. Such events are often only diagnosed at post-mortem. Systemic infection with *T. glabrata* is a life-threatening event in the metabolically or immunologically compromised patient. It is particularly likely to occur following broad spectrum antibacterial therapy or to the prophylactic or therapeutic administration of imidazoles.

We wish to thank Dr H L Halliday, Dr D H Gilmore, Mr C J J Russell and Dr R King for their permission to report the cases. We are also grateful to Miss Caroline Simpson for technical help and Mrs Cecilia McIlhatton for secretarial assistance.

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Case report

Hypothalamic tumour presenting as anorexia nervosa

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The term anorexia nervosa is used to describe a condition of food refusal in pursuit of thinness, often accompanied by self-induced vomiting and purgative abuse. Patients are typically very active and have a disturbed body image. Usually it is seen in pre-pubertal or pubertal girls. However, Bruch [1] describes atypical anorexia nervosa, which can occur in either sex, usually later in puberty. These patients recognise that they are thin and are more often depressed than hyperactive. This paper describes a patient whose initial clinical presentation resembled that of anorexia nervosa with some atypical features, but he was eventually found to have a hypothalamic tumour.

Case History. The patient, a schoolboy from a rural area, presented to hospital at the age of 14 years. His parents felt that his problems had started three years previously when he moved to a secondary school at which he was unhappy because he was the object of frequent bullying. However, they did not recall any physical symptoms until one year prior to admission when he complained of polyuria and polydipsia. There was no glycosuria and his symptoms settled fairly quickly. Two months before admission he complained of nausea and he vomited after eating. He stopped eating and lost weight. He became withdrawn and his parents found him "difficult". He agreed that he was thin but did not feel that he had any problem with eating. He lived on a farm with his family, and travelled to school in a nearby town. He was the youngest in a family of five, with four older sisters aged 17–23 years. There were stressful family events at the time of his presentation.

On admission to hospital he weighed 29 kgs and measured 141 cms in height, both figures well below the third centile for his age. He was withdrawn and unco-operative during the examination which revealed no abnormal neurological signs. The following tests were all normal:- haematological and biochemical profiles, morning and evening serum cortisol, urine culture, chest X-ray and barium meal. A diagnosis of anorexia nervosa was made and he was treated initially with intravenous fluids and nasogastric feeds and later with total parenteral nutrition. He continued to avoid food, and persisted in vomiting any food he managed to take. He failed to gain weight.

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He was referred to the metabolic unit where, under a similar regimen with the addition of chlorpromazine, he gained 9·5 kgs. He remained very difficult and unco-operative on the ward and, as his physical condition had improved, he was transferred to a psychiatric unit. His condition remained fairly static under a strict regimen in which privileges were lost or gained depending on changes in his weight or behaviour.

One month later he became acutely ill and was investigated in the paediatric medical unit. He initially complained of an occipital headache but within a few hours he developed drowsiness and slurred speech and appeared severely dehydrated. Cerebrospinal fluid examination revealed 100 leucocytes per ml with 65% polymorphs. However, the protein and sugar content were normal and no organisms or neoplastic cells were identified. The serum sodium was 165 mmol/l and urea 17·1 mmol/l. In spite of his severe dehydration he had a urinary output of 2 l/day, with an inappropriately low urinary osmolality. Skull X-rays and a CT scan (Fig 1a) showed periventricular calcification and an appearance suggestive of intraventricular tumour. He still had no neurological signs on clinical examination, although visual field perimetry now revealed bitemporal scotomata. In the neurosurgical unit, examination via a right frontal craniotomy revealed a mass of firm tissue on the floor of the right lateral ventricle in relation to the head of the caudate nucleus. The portion removed for histological examination showed gliosis. After the operation he felt better, vomiting decreased and there was no further weight loss.

Four months later his symptoms recurred and CT scan (Fig 1b) revealed extension of the lesion with encroachment on the third ventricle. After radiotherapy the tumour regressed (Fig 1c), he gained weight, his appetite improved and he became more co-operative. The field of vision in his right eye returned to normal although a temporal defect persisted on the left side. Improvement continued for a year, his weight remaining around 41 kgs. Then his appetite diminished, his weight dropped slightly and there was no further gain in height. CT scan now showed further spread of tumour although it remained confined to the ventricular system.

He was re-admitted to the metabolic unit as he had become increasingly unco-operative, was refusing to eat and had lost 3·6 kgs in weight. Nasogastric feeding was recommenced and cortisol and androgen given intramuscularly, as there was biochemical evidence of hypopituitarism. A week after admission he had an epileptic fit and developed cerebrospinal fluid rhinorrhoea. Lumbar puncture revealed uniformly blood-stained fluid and large nucleated cells. He became increasingly drowsy and further CT scan (Fig 1d) showed marked spread of tumour which involved the left frontal lobe. He died the following day. A post-mortem examination was not performed but from the malignant appearance of cells identified in the CSF, and the clinical and radiological behaviour of the tumour, a presumptive diagnosis was made of ependymoma.

DISCUSSION

There have been reports in the literature of hypothalamic tumours mimicking anorexia nervosa in adolescents and adults.[2,3] The diencephalic syndrome of infancy and childhood consists of severe emaciation with a happy, active disposition.[4] However, Bauer[5] demonstrated, in a review of 60 patients with lesions in the hypothalamic region, that only 44% had weight change, and the majority of these (26%) became obese.

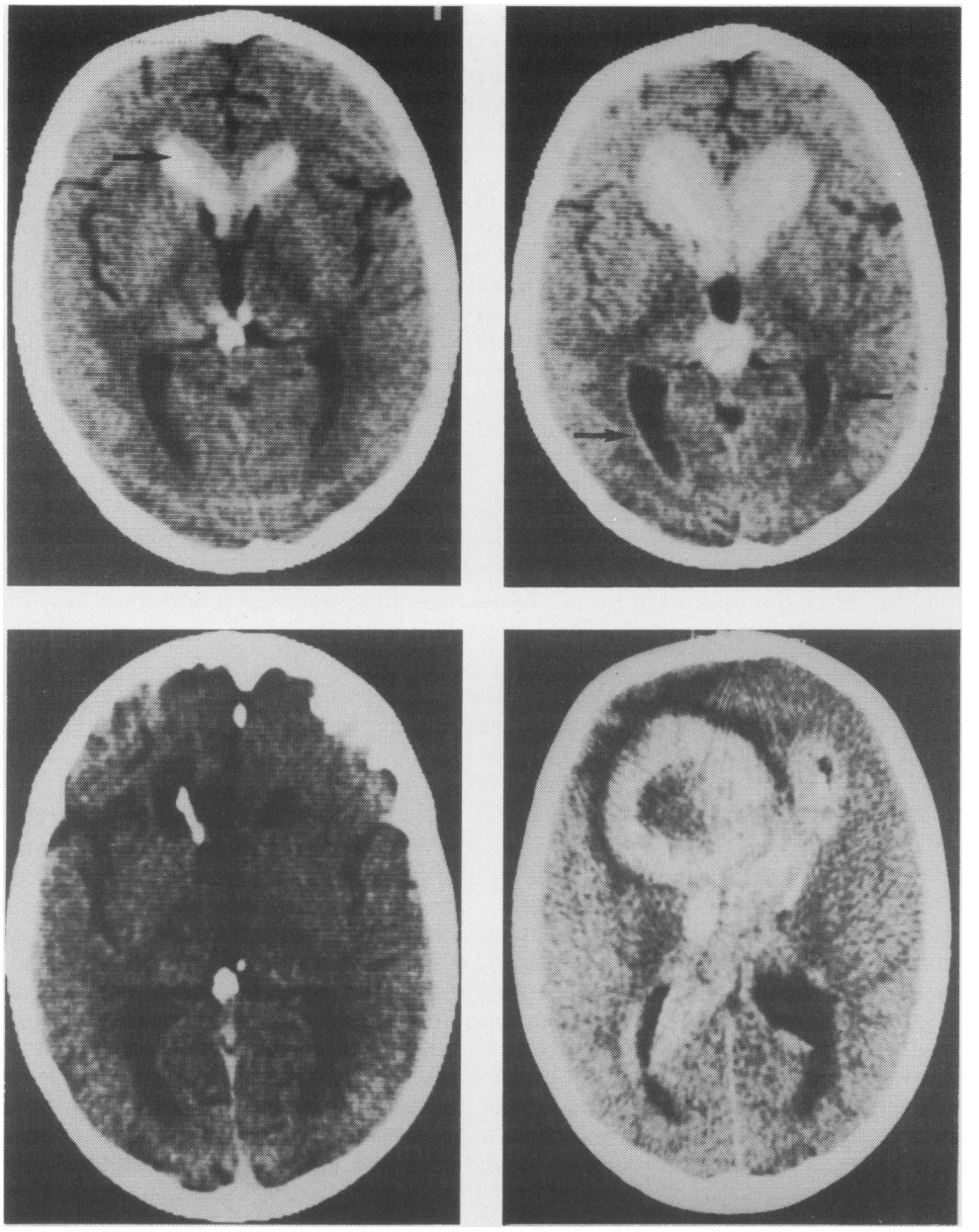

Fig 1. CT scans at the level of the third ventricle.

(a – top left) Six months after initial presentation, there was calcified tissue around the frontal horns of both lateral ventricles (arrow).

(b – top right) Four months later, tumour had enlarged and spread to posterior third ventricle and margins of lateral ventricles posteriorly (arrows).

(c – bottom left) Following radiotherapy, only frontal periventricular calcification remains.

(d – bottom right) Last scan, shortly before death, shows massive recurrence in the left frontal region with extensive periventricular spread bilaterally.

Various endocrine changes in anorexia nervosa[6] suggest an underlying hypothalamic dysfunction. Most of these changes are similar to those found in starvation states from other causes which suggests that they are initiated by weight loss. However, the changes in the hypothalamic-pituitary-gonadal axis and the hypothalamic-pituitary-adrenal axis seem to be unique to patients with anorexia nervosa. Thus it is perhaps not surprising that difficulty arises in diagnosing a hypothalamic tumour which appears to present as a case of anorexia nervosa. There were no specific neurological signs or symptoms in this case, and there were several features in keeping with anorexia nervosa — his lack of awareness of the problem with his eating habits, his difficult personality, his weight gain on "enforced" treatment and the age of onset.

According to Bruch[1] the less typical features of this case — his awareness of weight loss and depressed lethargic state — are found in secondary anorexia nervosa. The refractoriness of his condition, the deterioration of his personality and finally the acute episode of severe dehydration secondary to the onset of diabetes insipidus led to the correct diagnosis. This case, and others previously reported, demonstrate the need to consider the diagnosis of hypothalamic lesions in patients with anorexia nervosa. Response to treatment of anorexia nervosa does not exclude a hypothalamic tumour. A CT scan should be considered in any patient with atypical features.

We would like to acknowledge the co-operation of Dr D R Hadden, Consultant Physician in the Metabolic Unit; Mr D S Gordon, Consultant Neurosurgeon; Dr S McKinstry, Consultant Neuroradiologist; and Prof Ingrid Allen, Consultant Neuropathologist, all from the Royal Victoria Hospital; and Dr Clare Adams, Consultant Psychiatrist at the Windsor Unit, Belfast City Hospital, in the preparation of this case report.

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NOTICE.

"Liberality, Consideration and Dignity"

THE ROYAL MEDICAL BENEVOLENT FUND

The words in the title were used by an elderly medical widow whose circumstances changed suddenly and she did not know where to turn to or how she was going to manage. The Royal Medical Benevolent Fund was contacted and as a result of her application and the back-up report of the Area Visitor, this lady has been helped and her worries have been lifted from her shoulders. These words of thanks sum up the Fund's work so accurately they make a good beginning to this article about the work of the medical profession's own charity.

The Royal Medical Benevolent Fund was founded in 1836 by the same group of doctors who had earlier founded the British Medical Association. During the last 152 years the two organisations, though independent of each other, have worked closely together and with other medical charities which may be more locally based (such as the Royal Medical Benevolent Fund of Ireland, which is Dublin based). The founding fathers saw a need for a charity for 'the relief of medical men and their families who were under severe and urgent distress occasioned by sickness, accident or other calamity'. These are still the aims of the Fund, but it was made clear in 1836 that the Fund was not designed to relieve medical men of the necessity of providing for their families in 'ordinary life assurance and such other means as prudence dictates'. In the Fund's first year of action £17, 15. 0. was distributed and in 1987 that figure had grown to £520,000. The sum paid out to beneficiaries increases every year and sadly there seems to be no decrease in the number of people needing help from the Fund. The age range of beneficiaries is from a baby of a few months old to a widow of 97 years.

The best way to show how the type of help to be given is arrived at is to follow an enquiry from its first arrival at the office until the recipient gets their help. The enquiry itself may come through a colleague, the family doctor, the Social Services department, Guild members — these are active supporters of the Fund's work, or the local Area Visitor. The Area Visitors are a network of medical wives who visit the beneficiaries and act as the eyes and ears of the Case Committee. The applicant is sent a two part application form which gives details of the personal and financial situation. The Area Visitor will go to see the new applicant and then write a report for the Case Committee. She is the person who lets the Case Committee know about the applicants actual state — the paint peeling off the wood-work, the frayed carpet, the single bar electric fire, the garden which was once a great joy but can no longer be kept in order due to infirmity and inability to afford help for even the most basic cutting of grass and hedges — an overgrown garden is a singularly depressing sight. All these things and many more can be brought to the notice of the Case Committee. The Case-Workers in London, who are professionals, check all references and prepare case-notes for

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the Case Committee and give suggestions for the type of help which may be appropriate, but it is the Case Committee at its monthly meeting that makes the final decision about what help is to be awarded to each applicant or what variation to existing awards are to be made. To return to my example, the Case Committee might decide to award her a weekly grant which will allow her to live in dignity with a little surplus which might allow her to employ someone to help in her house or to help tidy up the garden. A grant might be given to cover the cost of re-painting (estimates would be asked for before this would be paid), and possibly a grant to allow for the purchase of a safer and more economical type of electric fire. Most beneficiaries have their cases reviewed annually and so the help can be kept tailored to what is required. Sometimes there can be exceptional needs — the cooker may cease to function and can no longer be repaired and a grant can be given for its replacement. Recently in Northern Ireland we have given help with the changeover from mains gas to bottle gas or electricity if the statutory grant has been insufficient.

Sadly, some of the cases that come before the Case Committee involve doctors who, through a combination of illness, financial mismanagement and subsequent debt reduce the family to income support or supplementary benefit level. In these cases, once all state aid has been taken up we can give a small weekly grant, also provision for television rental and licence, telephone calls and rental, and gifts to cover occasional items. The Case-workers in the office liaise closely with the Social Services to ensure that we give all available help, but do not affect the recipient's rights to state benefits. All income support beneficiaries are offered the facility of the "clothes room" which can supply a wide range of first-class garments. Other beneficiaries also have the use of the clothes room and I feel it is a very worthwhile place for the misfits of any wardrobe to end up — needless to say everything is 'good as new' and great care is taken by the ladies who make up the parcels — they mix and match articles to give a range of outfits and they do not forget accessories like scarves which can be the finishing touch to a nice suit.

We also have an increasing number of deserted or divorced wives whose husbands do not pay maintenance as they should. The Case Committee is looking into ways of making loans rather than grants to this group, so we will not subsidise unscrupulous husbands. Sometimes help may be given as a loan to allow someone to complete a course of study and become independent. Sometimes help is required with nursing home fees and once all state aid and family support has been obtained then, and possibly in conjunction with other charities, help will be given to allow an elderly person to remain in the nursing home of their own choice. As well as help throughout the year, all beneficiaries receive Christmas cards and presents — the Christmas gifts come from the proceeds of the Fund's annual Christmas Appeal and they are a source of great joy at that time of year. Older beneficiaries also receive birthday presents and cards.

All this help for the less fortunate members of the profession requires a lot of money, and all is raised from members of the Medical Profession and their families. The easiest and most cost effective way of subscribing to the Fund's work is by Deed of Covenant — this not only benefits the Fund in that it gains the additional refund of tax from the Inland Revenue, but they also know that for a number of years that that amount of income will be coming in. Legacies are also valuable and just recently a house, expected to realise £200,000, has been left to the Fund — a truly magnificent gift. Apart from the individual subscribers there are many groups, usually organised by medical wives, known as Guilds, which

run a great variety of fund-raising functions — in this respect really anything goes as long as people enjoy themselves and are prepared to pay for the privilege!

I have given you a very brief outline of the work of the RMBF — it is the doctor's own charity and as such it is wholly dependent on doctors for its support. Sadly, the need for the Fund does not diminish — in fact when I became the Northern Ireland Area Visitor in 1984 there were three beneficiaries and there are now eight.

As you all know, in the present political climate self-help is encouraged and unearned benefit is frowned upon, so we will have to help to a much greater extent both in the realm of nursing home fees and support of young people from broken marriages. Please help us to be able to continue to care for all who need it with 'Liberality, Consideration and Dignity'.

Lesley Donaldson.

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Book reviews

Belfast: 100 years of Public Health. By R Blaney. (pp 84, illus. £2.95). Belfast City Council and the Eastern Health and Social Services Board, 1988.

Dr Blaney's book "100 years of Public Health in Belfast" coincides with the centenary of this great city. In fact the book concentrates on the first 30 years or so of Belfast's city status. This may be because the successors to the Victorian public health reformers were ineffective; or it may simply reflect the fact that they were left with little upon which to improve, though I doubt this; or perhaps it is because the records of those in the twentieth century are less complete and accessible than those of their predecessors. Whatever the explanation, the description we have is well worth reading. At £2.95, with many handsome illustrations, it is good value for anyone interested in public health or medicine in Belfast. My only significant complaint is the production in "coffee table format", which makes it impossible to place in any conventional bookcase. While facilitating the use of large illustrations, such a format will ensure that most copies of this useful book will not survive the next hundred years.

PMB

Clinical endocrinology and diabetes. Edited by MC Sheppard and JA Franklyn. (pp 191, illus. £22.50). Edinburgh: Churchill Livingstone, 1988.

There are now a great number of small paperback books in the different specialties which are geared directly to the sub-specialties of present day medical practice. It is clear that there is a market for such small disease-orientated booklets just as it is equally clear that a comprehensive treatise of all aspects of internal medicine is too big to handle, to say nothing of expense.

This undergraduate and postgraduate summary of present day endocrinology has the great advantage of being produced by colleagues from the same medical school who knew each others work and who are consistent in their approach. The clinical work and research activities of the group of endocrinologists who have trained and practiced in Birmingham in the past 10 years is widely respected and this book does credit to them all. There is a reasonable degree of unanimity amongst the endocrinologists in the United Kingdom in terms of what we would consider standard investigative procedures and this book certainly acts as a reasonable and convenient reference source. The section on diabetes is intentionally brief and would have to be supplemented by a separate monograph for full information.

I suppose £22.50 is a reasonable price by present day standards. There is a case for having a copy of a brief clinical book of this type available for all clinical students attached to a ward during their clinical clerkship. This is the sort of stimulating introduction to a topic which might well be the stimulus for the next generation of clinical endocrinologists.

DRH

Paediatric respiratory diseases: 4th meeting of the European Paediatric Respiratory Society, Cracow. Editors: J Rudnik and R Kurzaura. Rabka: National Research Institute of Mother and Child, 1985: pp 387.

This book is a compilation of papers presented at the 4th meeting of the European Paediatric Respiratory Society held in Cracow in 1983. The papers are arranged into five parts: (I) epidemiology of paediatric respiratory disease, (II) diagnosis of paediatric respiratory diseases, (III) asthma in children, (IV) bronchitis and non-specific respiratory disease in children, (V) miscellaneous, dealing mostly with therapy and social aspects of paediatric respiratory disease.

Understandably, as the authors of the various papers come from different countries, there is a great variation in terminology in the papers. This does not lead to easy reading. This book would be of interest mainly to paediatricians concerned with respiratory disease, particularly those involved in definitive research projects. As it is not totally comprehensive of respiratory disease, it is not suitable for those studying for higher paediatric examinations. The papers on standardization of lung function tests in infancy by Helm presents an excellent review of respiratory function in the young child.

AR

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Understandably, as the authors of the various papers come from different countries, there is a great variation in terminology in the papers. This does not lead to easy reading. This book would be of interest mainly to paediatricians concerned with respiratory disease, particularly those involved in definitive research projects. As it is not totally comprehensive of respiratory disease, it is not suitable for those studying for higher paediatric examinations. The papers on standardization of lung function tests in infancy by Helm presents an excellent review of respiratory function in the young child.

AR

The Ulster Medical Journal, Volume 57, No. 2, p. 231, October 1988.

Book reviews

Belfast: 100 years of Public Health. By R Blaney. (pp 84, illus. £2.95). Belfast City Council and the Eastern Health and Social Services Board, 1988.

Dr Blaney's book "100 years of Public Health in Belfast" coincides with the centenary of this great city. In fact the book concentrates on the first 30 years or so of Belfast's city status. This may be because the successors to the Victorian public health reformers were ineffective; or it may simply reflect the fact that they were left with little upon which to improve, though I doubt this; or perhaps it is because the records of those in the twentieth century are less complete and accessible than those of their predecessors. Whatever the explanation, the description we have is well worth reading. At £2.95, with many handsome illustrations, it is good value for anyone interested in public health or medicine in Belfast. My only significant complaint is the production in "coffee table format", which makes it impossible to place in any conventional bookcase. While facilitating the use of large illustrations, such a format will ensure that most copies of this useful book will not survive the next hundred years.

PMB

Clinical endocrinology and diabetes. Edited by MC Sheppard and JA Franklyn. (pp 191, illus. £22.50). Edinburgh: Churchill Livingstone, 1988.

There are now a great number of small paperback books in the different specialties which are geared directly to the sub-specialties of present day medical practice. It is clear that there is a market for such small disease-orientated booklets just as it is equally clear that a comprehensive treatise of all aspects of internal medicine is too big to handle, to say nothing of expense.

This undergraduate and postgraduate summary of present day endocrinology has the great advantage of being produced by colleagues from the same medical school who knew each others work and who are consistent in their approach. The clinical work and research activities of the group of endocrinologists who have trained and practiced in Birmingham in the past 10 years is widely respected and this book does credit to them all. There is a reasonable degree of unanimity amongst the endocrinologists in the United Kingdom in terms of what we would consider standard investigative procedures and this book certainly acts as a reasonable and convenient reference source. The section on diabetes is intentionally brief and would have to be supplemented by a separate monograph for full information.

I suppose £22.50 is a reasonable price by present day standards. There is a case for having a copy of a brief clinical book of this type available for all clinical students attached to a ward during their clinical clerkship. This is the sort of stimulating introduction to a topic which might well be the stimulus for the next generation of clinical endocrinologists.

DRH

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