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CASE REPORTS

The Ulster Medical Journal, Volume 59, No. 2, October 1990.

The Ulster Medical Journal

The Journal of the Ulster Medical Society. First published in 1936.
Successor to the Transactions of the Ulster Medical Society (1884–1929), and the Transactions of the Belfast Clinical and Pathological Society (1854–1862)

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presented in a clear format, with easy to read text and clearly labelled illustrations. There is emphasis throughout on patient care. The suggestion that planning, organisation and critical thinking skills should be developed for effective trauma radiography, will stimulate interest in many experienced radiographers.

The first five chapters of the book provide information on relevant aspects of trauma care and include practical hints and guidelines. The sixth and main chapter of the book presents 131 adaptive techniques for radiographing trauma patients. These are described in an unorthodox way and must be learnt. Once learnt, however, the technique can be applied with any patient position.

This book deals thoroughly and lucidly with the subject. It will be of practical value in preparing students and radiographers to meet the challenge of trauma and mobile radiography. It is a useful reference book and a welcome addition to school and clinical libraries.

M McMILLAN

The respiratory care workbook. By Howard B Surkin, Anna W Parkman. (pp 372, illus. £16.17) Philadelphia: F A Davis Company, 1990.

This book is a general respiratory teaching manual. It consists of lists of recommended American text books, few of which are in general use in the UK. It uses the knowledge from these to prepare sections on multiple choice and short questions related to basic sciences, diagnosis, investigation and therapy of respiratory conditions. It also supplies the answers. In therapeutic terms, it suffers from the different availability of drugs in the USA. Thus asthma is treated by "avoidance of allergens, cromyln sodium and steroids as needed for infection", and cardiogenic pulmonary oedema by "lasix, morphine and digitalis". Despite these criticisms, it is probably a valuable revision book for those interested in a full time career in respiratory medicine.

CF STANFORD

Clinical procedures for medical assistants. (Third edition). By Bonewit. (pp 606, figs, illus. £21.50). Philadelphia: W B Saunders, 1990.

This book has little or no relevance to British medical practice as it is directed towards medical assistants who carry out a multitude of practical procedures in physician's offices throughout the United States. These "totipotent" practitioners usually have a nursing background, and are an essential link in ambulatory medicine, although the tasks they undertake may include physiotherapy, ophthalmology, pharmacy or radiology. We have no equivalent practice in the United Kingdom so that this book is unlikely to be of interest or use to paramedical personnel this side of the Atlantic.

BJ ROWLANDS

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The Ulster Medical Journal, Volume 59, No. 2, October 1990.

THE ULSTER MEDICAL SOCIETY

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The Ulster Medical Journal, Volume 59, No. 2, pp. 103 – 105, October 1990.

Editorial

Medical education in the United States

"Today's medical students are trained primarily to be medical scientists and technicians. Emerging systems of health care are forcing them to be bureaucrats and businessmen as well. There is a conflict between the humanistic needs of the patients and the financial and regulatory constraints of the health care system. Today's physician is often an unwilling bureaucrat, filling out unnecessary forms and attending unnecessary committee meetings and having little time to sit in a leisurely fashion by the bedside to ease a patient's concerns".[1]

What about the current situation with the United States medical schools, with an average of less than two applicants for each position? It is stated by some that applicants are stagnating due to the high cost of tuitions and fees, the endless grind, and the loss of prestige. The number of applicants increased during the 1960's and early 1970's, peaking at 42,264 in 1974. From 1981 to 1988, there has been a 27% decline. However, women applicants, who were conspicuous by their limited numbers up to about 1970, have increased and today are about 40% of the total. The quality of the applicants, as gauged by their science grade point average in college, has shown no significant change over the years.

Tuition costs continue to increase and much faster than inflation. The median for private medical schools was $1050 in 1960–61; $2900 in 1974–75; and $17,454 in 1980–90. Tuition, however, pays less than half the cost of the education. The average debt of indebted medical school graduates increased 119% between 1980 and 1988. By 1988 57% of graduates were reporting debts of about $37,000, with a few as high as $100,000. Today the graduates must start to pay back their loans during residency training. Another concern is that these debts can influence the decision to train for the most lucrative medical specialties.

Concerning the selection of medical students, one has listened to debates that there is too much emphasis on high scores in the physical sciences, that we should admit more students who have majored in social sciences and humanities, so that we get more humanism into medicine. There is no evidence, however, that college students who have a strong interest in the natural sciences are less concerned about humanism than other students. The interview which is a requisite part of the selection process, also serves to recruit candidates to the schools. However, it is no guarantee of the admission of the best candidates. If we were truly objective, we would list all the applicants who had appropriate qualifications and offer admission to the required number by lottery.

Few would disagree with the concept that medical education should include the patient as a living organism, as a member of society and as a person. The first

expresses the roots of medicine in the natural sciences; the second, in the social sciences; and the third, in the humanities.[2] Subjective debate continues, however, on the optimal process to educate medical students. It has been stated for example that to train physicians for the twenty-first century, medical education should be integrated rather than subject based, should be faculty rather than department based, and should be "active" rather than "passive"; that student evaluation should be subjective as much as objective; and that steps should be taken to define objectives and promote literacy.[3] However, the prime factor in successful education is not in adopting the latest fashionable theory, but in establishing a faculty of competent teachers who can interest and motivate the students. When we reflect on key events in our education, seldom are the merits of particular videocassettes mentioned, but, rather, a certain teacher or teachers who had a key influence on our career. For the future, we need to find ways to evaluate the quality of faculty teaching and include this in the determination of academic promotion.

Discussion is also active in the future role of the physician in biological research, reinforced by the current trend to favor molecular biology in providing grants for research and training, with diminished emphasis on the regulation of the integrated responses of the body systems in health and disease. It is suggested that the clinician-investigator is an endangered species and that the United States may become a consumer rather than a producer of medical research. Those training for medical careers can be discouraged by the length of training necessary to become competent both in clinical practice and research. Indeed, it can be argued that, with the increasing complexity of medical practice and the exponential output of papers in biomedical research, each is a full-time occupation.

Another factor is the uncertain long-term institutional financial support for research and the necessity for competing for funds from the National Institutes of Health every three to five years, with the realization that today a successful candidate must attain the 12–14 percentile in the peer-review system to be successful. In seeking solutions it is acknowledged that clinician-investigators are necessary to relate the advances in the basic sciences to practice and to bring the problems from the patients to the laboratory. I like the words of Jack Masur, designer and first director of the Clinical Center at the National Institutes of Health, "Research enhances the vitality of teaching. Teaching lifts the standards of service. Service opens avenues of investigation".

As the new graduates of our medical training programs emerge to take their place in American medicine, they will face the fact that medicine has devised more health care than we can afford to deliver. It has been said that the United States health care system is the most expensive, least efficient and least equitable in the developed world, with costs for medical care exceeding more than 10% of the gross national product. About 22% of this is stated to be due to wasteful administration to which can be added excessive malpractice insurance and the increasing impediment of physician-effectiveness in time-consuming efforts of surveillance and reviews, such as the so-called "paper-trail" commanded by the

Joint Committee on Accreditation of Health Care organizations. Each country faces the challenges of affordability, availability, quality, and priorities for medical care. The answer to these questions will require gumption, which is a relatively rare human trait. Those who lack it fall into the category of one of the most delightful sayings of antiquity, that of Heraclitus, on his predecessors: "They had much knowledge, but no sense". Hopefully some of those graduating from medical schools will provide further leadership in meeting these challenges by a combination of knowledge and gumption.

John T Shepherd, MD (Belfast), DSc, FRCP,
The Mayo Foundation, Rochester, Minnesota 55905, USA.

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Functional decline and survival in dependent elderly people

Vivienne Crawford, Henrietta Campbell, E Hodkinson, R W Stout

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SUMMARY

A survey of dependency levels was carried out in 1985 and 1989 in 41 residents of old peoples homes and 25 patients in geriatric continuing care wards. There was an increase in all levels of dependency for those in both types of care. In the hospital patients surveyed in 1985, mortality was greatest in the most dependent, particularly those with impaired mental function. There was a positive correlation between length of survival and mental function. The results of this study emphasise the important role of dementia in the health and survival of old people.

INTRODUCTION

In order to plan for the needs of elderly people in the future it is not only necessary to predict the number of people in the oldest age groups but also to examine how the most dependent of old people make use of institutional care.[1] This study measured the changes in dependency levels that occurred over a four year period in a group of elderly people who were in residential or hospital care, and related these changes to survival. The results emphasise the importance of impaired mental function in prognosis.

METHODS

In 1985 the disability level of all the residents of the residential homes and continuing care wards in the South Belfast district was measured[2] using the survey questionnaire of the Clifton Assessment Procedure for the Elderly (CAPE).[3] The questionnaire comprises 12 questions on information/orientation (I/O) and six questions relating to physical disability (Pd). The survey score is calculated by substracting the Pd from the I/O score and applying the result to a five point dependency grading system. In this system each subject is assigned a disability score from A (independent) to E (most dependent). A further survey, using the same methods, took place in these institutions in 1989.[4] It was

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therefore possible to compare the results for those who were resident on both occasions, and to assess the predictive value of the 1985 measurements on survival.

Statistical analysis: after testing for skewness the samples were analysed using an independent samples t-test which compared the measurements between the outcome groups. A two-tailed t-test was used to examine the relationship between total dependency score and length of survival.

RESULTS

Sixty six subjects were tested in both 1985 and 1989, 41 in residential accommodation (15 men, mean age 73·1, range 62–83 and 26 women, mean age 81·6, range 66–94), and 25 in the geriatric medical unit (5 men, mean age 79·4, range 74–83 and 20 women, mean age 81·0, range 72–92). In each case there was a significant decrease in the level of information/orientation, increase in physical disability, worsening of mobility and continence, and an increase in the total disability score. Thus, over a four year period there was a significant increase in dependency in the residents of both institutions (Table).

TABLE

Mean (± S.E.M.) scores for some measures of dependency on the CAPE survey for residents and patients studied in 1985 and 1989. All differences were highly significant between 1985 and 1989

	1985	*1989*
Residential Accommodation (n = 41)		
Information/Orientation*	9·10 ± 0·48	6·95 ± 0·68
Physical disability+	3·12 ± 0·36	4·49 ± 0·44
Mobility+	0·37 ± 0·09	1·20 ± 0·12
Continence+	0·10 ± 0·05	0·59 ± 0·11
Dependency score*	5·98 ± 0·73	2·49 ± 1·00
Geriatric Medical Unit (n = 25)		
Information/Orientation*	6·54 ± 0·68	3·62 ± 0·91
Physical disability+	7·00 ± 0·58	8·40 ± 0·48
Mobility+	1·56 ± 0·12	2·00 ± 0·00
Continence+	1·12 ± 0·17	1·59 ± 0·15
Dependency score*	−0·33 ± 1·18	−4·75 ± 1·31

*Lower value indicates increased dependency.
+Higher value indicates increased dependency.

Using the 1985 results, a comparison was made of the 78 patients who died in hospital before the 1989 assessment with the 25 who survived and were reassessed. Those who died were significantly more dependent in the survey score ($p = 0·04$) and in information/orientation ($p = 0·028$) but not in physical disability score, compared with those who survived. There was a significant correlation between the total dependency score and survival ($r = 0·263$, $p = 0·033$), the least dependent surviving the longest. When the dependency

score was broken down to its two components, the information/orientation score was positively correlated with length of survival ($r = 0 \cdot 328$, $p = 0 \cdot 007$), (Figure) whereas there was no significant relationship between the physical disability score and length of survival. Age and sex were not related to survival or length of stay.

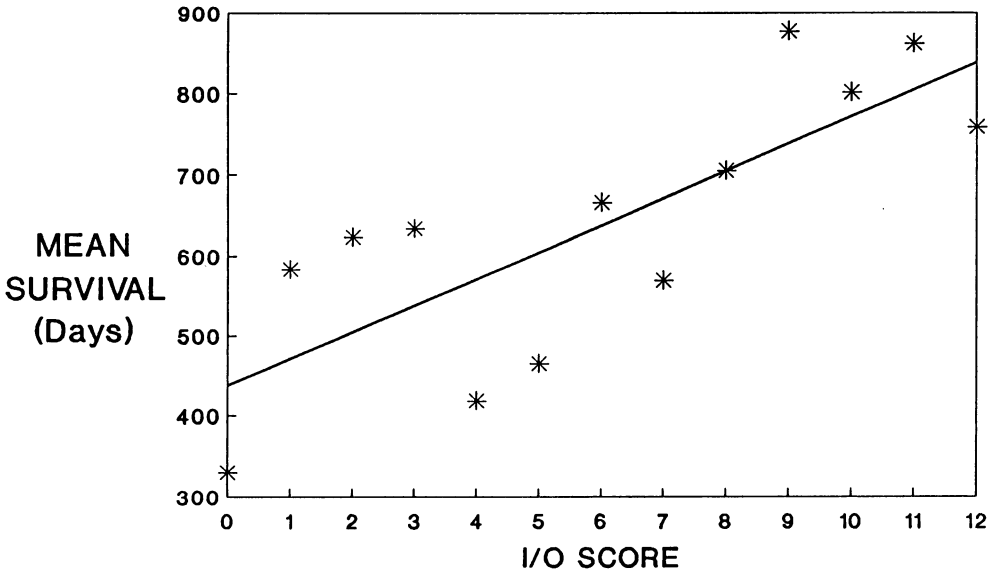

Figure. Relation between the information/orientation (I/O) score and length of survival of patients in the geriatric medical unit ($r = 0 \cdot 328$; $p = 0 \cdot 007$)

DISCUSSION

As might be expected, there were significant increases in all measures of dependency in institutionalised old people surviving over a four year period. As those in the geriatric wards were already very dependent in 1985, it is not possible to compare the rate of change in dependency in the two types of care. This group of elderly people in hospital or residential care is not typical of the elderly population as a whole as it represents the most disabled old people. A recent survey of elderly people in the community, using a slightly different survey instrument, has reported that cognitive impairment is associated with early death in these people.[4] Thus, it is likely that a relation between cognitive function and survival is a general feature of old age.

Since this study was completed, questions have been asked about the accuracy of the CAPE method in diagnosing dementia.[5, 6] It seems that an information/orientation score of 7 or less has a low sensitivity but high specificity in that it accurately identifies severe dementia, but mild dementia is under-diagnosed. However, the linear relation between information/orientation score and survival found in the present study does not depend on an arbitrary cut-point. It must also be accepted that the CAPE system provides a measure of the degree of mental or physical disability, but does not identify the cause of the disability.

The results of this study emphasise the important role of dementia in the health of elderly people. People with dementia were disproportionately represented among the residents of residential homes and the patients in long stay wards.[2, 7] Thus,

dementia is a major contributor to the development of severe dependency in elderly people and the need for institutional care. The present study shows that dementia also predicts mortality of dependent elderly people. Previous studies have shown a higher death rate among institutionalised elderly people with impaired mental function.[8, 9] The present study directly relates survival to mental capacity.

Although there has been an increase in the age of admission and length of stay of old people requiring continuing hospital care in the last three decades,[1] with many of these people suffering from dementia,[7] this and other studies show that people with dementia have diminished survival. Therefore it is difficult to calculate whether the need for services for the increasing number of frail elderly people will be balanced by the reduced survival of those with dementia.

We are grateful to the Assistant Director of Social Services for the South Belfast district, the proprietors of the nursing homes, and the consultant physicians in the geriatric medical unit, Belfast City Hospital, for permission to study the residents or patients under their care; and to Miss Andree Best for preparation of the manuscript. The opinions expressed in this paper are those of the authors and do not represent the views of the Department of Health and Social Services, Northern Ireland.

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The self and its brain

R J McClelland

Accepted 19 April 1990.

Based on a Lecture delivered to a joint meeting of the Ulster Medical Society and the Ulster Neuropsychiatric Society on 16th March 1989.

In 1973 Philippe Pinel, physician in charge of the Bicetre asylum in Paris, literally removed the chains from his patients and ordered that they should be treated with kindness and understanding.[1] This action reflected two important phenomena. One was the spirit of humanitarianism abroad in France at the time. The other was an emerging view of mental illness as a natural or biological phenomenon. Those afflicted were sick, they were mentally ill.

Prior to this time the contents of the human mind — thoughts, feelings, motivation — were very much the concern of the Church. Because the mind was conceived as a free agent, free from bodily and physical constraints, human beings were considered responsible for any abnormality in their behaviour or beliefs. Those with a disorder of thought or perception were usually believed to be in consort with the devil. It is perhaps paradoxical that the scientific reductionism dawning in the 18th century was associated with a new and liberating doctrine which sought to restore human dignity to the mentally ill. Taking a wider view — from the Greek scientist-philosophers through to the modern psychobiologists — the issue that has most profoundly exercised the minds of men is the relation between the mind, of which each of us is personally aware, and the body. Is there a relationship, and if so what is its nature? The search for an explanation has come to be known as the mind-body problem, described by the philosopher Schopenhauer as the world knot.[2] But the mind-body issue should not be the remote province of philosophers; the brain and the mind are as weft and warp in the fabric of psychiatry — in the evaluation, diagnosis, and care of the mentally ill. The physician or general practitioner who takes account of this psycho-soma relationship enlarges his understanding of his patients and their illnesses.

I. *Defining the problems*

One of the biggest problems is that we have a certain commonsense picture of ourselves as conscious, free, rational agents; my existence as a self is a reality beyond any possibility of doubt (Fig 1). This view, however, is very hard to square with our overall scientific concept of the physical world: a world that science tells us consists of mindless, physical particles. How can it be that the world contains nothing but inanimate particles and yet that it also contains self-consciousness?

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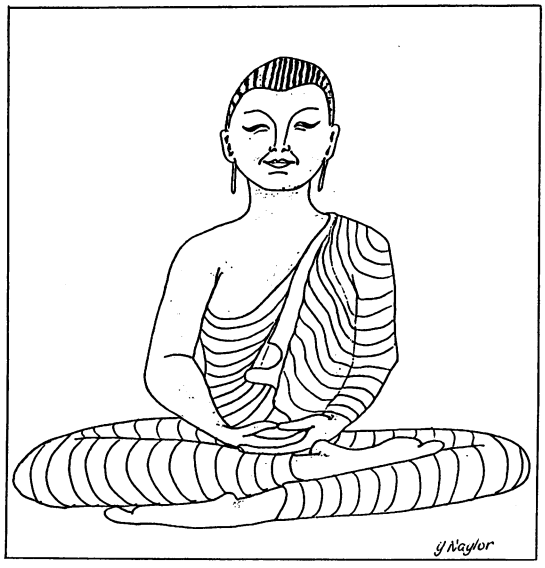

Fig 1. Buddha, who found enlightenment through prolonged contemplation under the Bodhi tree — symbolising *res cogitans* (thinking substance).

Fig 2. René Descartes (1596–1650).

Before proceeding further with our analysis of the issue I would like to trace the contribution of science to the world knot. This has its origins in the 17th century with the philosopher René Descartes, regarded by many as the father of modern science (Fig 2). Descartes asserted that the body was a machine, and this had a profound effect on medical science. First, it had a liberating effect on biology, allowing scientists to investigate animals as if they were machines.[3] Second, it taught that the human body was not sacred, but could be dissected and studied in the same way as any other physical system could be, except in so far as the rational mind was concerned. However, Descartes also accepted the traditional view that the rational mind, soul, or self was immaterial and immortal and hence accessible only to philosophy and theology, but not to science. Herein we see the origin of an important obstacle to the scientific study of mind; but an even greater problem was to follow.

The mind, within this framework was not a natural phenomenon. It stood outside nature. The effect of this conceptualisation was to leave this self, the inner person, perched precariously on the edge of matter and in strange conjunction with its body[4] (Fig 3). Soon the question was posed of how an immaterial non-natural mental thing, the mind, could act on a material body? With the subsequent rise in status of the physical sciences, the status of mental entities has been generally downgraded. A central thesis of modern scientific reductionism is that all physical substances, including the human body, can be reduced to simple particles and the forces acting on them. With this comes a determinism according to which the human mind is feeble and unfree. Thus most of the recent materialist conceptions of the mind — such as behaviourism and physicalism — have ended up by denying implicitly or explicitly that there is any such thing as a mind as we ordinarily think of it: ideas or feelings, for example, are at most mere epiphenomena and of no causal significance.[5] With these two contributions of

science to our problem we have inherited a cultural resistance to treating the conscious mind as a biological phenomenon.

There are three features which seem impossible to fit into our scientific conception of the world as made up exclusively of physical things: consciousness, mental causation, and subjectivity. First is the notion of consciousness itself. It is hard to see how a physical system, the central nervous system, could have consciousness and yet you, the reader, at this moment are presumably conscious. Once again Descartes was the first of the modern philosopher-scientists to address the problem. In his *Méditations* of 1630 he introduced the method of doubt as a technique for identifying the essence or true nature of things. Regarding the reality of the self, he speculated whether he might be the victim of a perceptual illusion, that perhaps his whole life was a dream. Then he noticed what seemed like a solid rock of truth — whatever doubts one may have about the truth of the content of one's thoughts there is never any reason to doubt one is having thoughts, that one is thinking. Any attempt to doubt or deny that one is thinking is quite nonsensical, since the very process of doubting or denying is itself thinking?

Fig 3. In the dualist philosophy of the mind and the brain, *res cogitans* (thinking substance) lies outside the body.

For Descartes this was the central truth regarding the reality of existence of the mind or the self — *cognito, ergo sum,* I think therefore I am. For the neurobiologist J Z Young, even more fundamental was the reality "I know that I am alive".[6] For the philosopher Sir Karl Popper the realisation of death is one of the great discoveries associated with full human self-consciousness — "to know that I will someday die is to recognise that I am alive, that I am, that I am a self".[7]

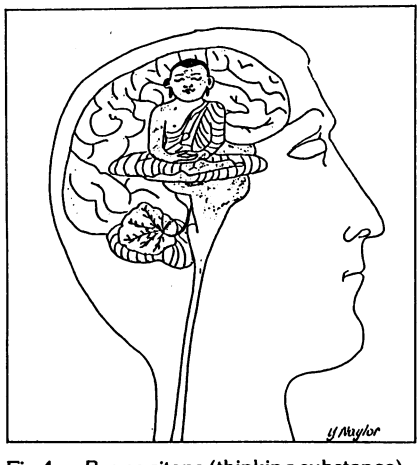

Fig 4. *Res cogitans* (thinking substance), or the self, often conceptualised as a person or homunculus within.

Consciousness is a dominating feature of existence, yet it is hard to characterise. The typical method of overcoming the difficulty is to speak of oneself as an inner entity, an inner thing, which leads to the postulation of an agent or person within. This was the origin of Descartes' homunculus[3] (Fig 4). The notion of a distinct and separate mind-thing and a body, or the dualist philosophy of the mind, is part of established tradition and therefore at the crux of my theme.

The second intractable feature is mental causation. We all assume that our thoughts and feelings significantly affect our action, that our mind does have a causal effect on the world around us. But if our thoughts and feelings are truly mental and immaterial, how can they affect anything physical? To take the illustration provided by the American philosopher John Searle, "are my thoughts not just as the froth on the wave is to the movement of the wave?" If the froth were conscious, it might think to itself "what a tough job it is pulling these waves up on the beach and then pulling them out again all day long!" but we know that the froth does not make any important difference. Why do we then suppose that our mental life is any more important than froth on the wave of physical or scientific reality?[5] Are we supposed to think that our thoughts and feelings can somehow produce effects on our brains and on the rest of our nervous system? How could such a thing occur? And yet unless there is some kind of connection between the mind and the brain it would seem that the mind cannot have any causal influence on the physical world.

A third important feature of the mind is subjectivity — for example I see the world from my point of view, you see it from yours. It is a characteristic feature of the present era that we have come to think of scientific reality as something that is objective — which is accessible to all observers. How can we reconcile the reality of subjective mental phenomena with this objective perspective of science?

Thus far we have defined the hard problems associated with three special properties of the mind: consciousness, mental causation, and subjectivity. The problems are that philosophy tends to split consciousness from body or brain, that scientific reductionism tends to deny mental causation, and that science resists the reality of the subjective.

TOWARDS A POSSIBLE SOLUTION

These features are what make the mind-body problem so difficult. Yet they are all very real features of our mental lives and any satisfactory account of the mind and of mind-body relations must take account of them.[5] On the one hand there are mental things such as our thoughts and feelings; we think of them as conscious, subjective, and immaterial. On the other hand there are physical things; we think of them as having mass and as interacting causally with other physical things. How can we account for the relationships between these two apparently completely different kinds of things? As J Z Young has commented, philosophers generally have paid little attention to the fact that knowledge and thought are somehow related to the brain.[6] Nevertheless it in imperative for neuroscience and medicine that we close this gap that keeps the study of mind a scientific anomaly. Fortunately over the last decade there has been an increasing interchange between philosophers and scientists.

I would like to examine briefly two modern responses to our problems. To begin with, the very expression "mind-body problem" suggests that mind and body are two separate entities. Yet we do not speak of the "motion-body problem" in mechanics or of the "lung-respiration problem" in physiology. Popper suggests one reason we have become confused about mind-body issues is that biology emphasises that organisms are hierarchies of structures rather than hierarchies of processes.[7] The philosopher John Searle reminds us that the mind should not be considered as a thing but rather as a process, a high level process of the brain. Such processes have parallels in other organ systems, for example in the way excretion is not a thing but a process or series of processes of the kidney embracing such specific functions as filtration and reabsorption.

Secondly the mind-brain identity theory, which has become the strongest thrust in materialist philosophy, has itself undergone substantial change. Initially it was strongly reductivist, holding that a complete account of mental processing is possible, in principle, in neural terms. The introduction in the mid-1960's of the opposing view of consciousness as an emergent process has been followed by transformations in the identity theory. Within this framework mental phenomena, while constrained by neural activity, also obey rules that are different in kind from those of their constituent neural material.[8] Mental laws are involved in determining behaviour and are necessary to explain behaviour.

It is important therefore to recognise that mind and brain are not identical: there is no more brain-mind identity than there is lung-respiration identity. To consider mind as a process, as an emergent function of brain, opens up several new possibilities. Note however, that such a mentalist position is not dualist but monist. Conscious processes are properties of the brain. Subjective events are generated and exist only by virtue of brain activity. They are inseparable from their physiological substructure. Yet once generated from neural events, higher order mental patterns have their own subjective qualities, operating and interacting by their own causal laws and principles. Compared to the physiological processes, conscious events are more molar.[8] The mental entities transcend the physiological, just as the physiological transcends the molecular. The meaning of experienced mental phenomena matters, and on the basis of such meaning we react. A catalogue of evidence from clinical psychiatry and behavioural neurology testifies to the importance of meaning as a causal factor in mental and behavioural adjustment and maladjustment.

Such psychoneural monism reduces to the following thesis: that all mental states and processes are processes in brains, and these states and processes are emergent relative to those of the cellular components of the brain.[9] The whole is more than the sum of its parts.

RESISTANCE

In considering our natural resistance to giving up the simple *dualistic* notion of a separate mind entity and brain entity, it is helpful to recount an earlier debate in the history of science. Biologists and philosophers have for a long time thought it was impossible to account for the existence of life itself on purely biological grounds. Some other additional element must be necessary. It is difficult today to realise how intense the dispute was between vitalism and mechanism even a generation ago. We now know there is no vital substance. Living things are physical systems made up of a small selection of the elements that make up the rest of earth. Moreover in nearly all respects the combination of these elements in living things behave like those in the organic world. Nearly, that is, but not entirely, and here we find two properties of all living things which give insight into our problem of conscious selves.

The first property is causality — all living things are in fact causal, they act in ways to ensure survival. They are not just the passive effects of lower forces. Their actions may be constrained to a large degree by molecular determinates, a reductionist account. However, living organisms also pursue aims, they act for some purpose so that teleological accounts are also valid.[6] Indeed if we accept Darwinian evolutionary theory then teleological accounts of biological activities, including mental activities, are just as valid as reductionist explanations.

Second and related is the notion of emergence. Popper proposes that there have been two major emergent phenomena in evolution — life itself and consciousness.[7] Life has arisen unexpectedly as an emergent property of the relationships of large amino acid structures. Consciousness is yet another highly improbable emergent event. It emerged in evolution in order to confer advantages for survival. Self-consciousness is a major upward step in our very long phylogenic history. A key conclusion from this analysis is that logic strongly suggests that all mental events are associated with changes in the brain. Intellectual activity and emotional experience all require activity in the brain and we are utterly dependant upon it.

Accepting the foregoing analysis one can talk about mental phenomena without leaving the biological ground.[9] Modern psychobiology seeks to explain the correlations that exist between neural activities and mental or behavioural events.

CODING, REPRESENTATION AND LANGUAGE

We now examine some of the contributions of modern neuroscience to our theme. One difficulty is the relative youth of neuroscience. For example Cajal's neuronal hypothesis of the brain is less than one hundred years old, but the last three or four decades have seen major conceptual developments. What had been solely the topic of philosophical speculation has now become open, at least in part, to laboratory investigation. A major theme in neurobiology to-day is representation or coding. What is the coding system with which the brain operates when it performs its remarkable feats?

The basic unit of function is the nerve impulse. Individual impulses are all alike and could not represent anything: it is only by their grouping in various ways that they can do so. Such grouping is central to coding information just as in morse code.[6] The grouping of nerve impulses is patterned both in time within each nerve fibre, the frequency code, and in space — that is among many fibres — the place code, which depends on which fibres are active. In studying the brain one is struck by the immense number of cells and nerve fibres which direct the action of the body. Even a relatively simple action like the movements of the chest in breathing is regulated by thousands of cells in several parts of the central nervous system. How much more complex must be the systems or programmes of activity for speaking, for feeling and ultimately for thinking? One complex high level process which is central to our understanding of the nature of human self-consciousness is language. Popper suggests that the self-conscious mind, in which the I is conscious of itself is only possible through language and through the development of imagination in that language.[7] Much of our understanding of the cerebral organisation of language and speech comes from clinical science. One hundred years ago the early advocates of duality of mind considered the two hemispheres of the brain to be functionally identical. The very idea that certain mental functions might be localised within specific brain areas was an anathema. Not surprisingly the early independent reports by Dax and Broca suggesting speech localisation in the left hemisphere provoked a harsh response. Such an asymmetry hypothesis was phrenological nonsense and not worthy of scientific attention. When Broca reported eight new cases supporting a localisation of expressive speech in the third frontal convolution he qualified his observations "I dare draw no conclusions and I await new facts".[10] Soon after, clinical studies revealed that the left superior temporal convolution and surrounding areas were critical for the understanding of speech.

Of course such knowledge arising from study of lesions should not hide from us the incredible complexity of the encoding and decoding involved in speech and language. Beginning with Penfield and his associates, stimulation of the cerebral cortex has provided new and important evidence on the organisation of language functions. One important finding from the recent work of Ojemann is that the language cortex is discretely organised. In almost half the sites studied, only single specific functions were affected by stimulation — for example in one bilingual subject naming in English and in Greek were separately affected.[11]

A third clinical dimension which sheds light on the mind-brain problem is the effect of transection of the corpus callosum for the amelioration of intractable epilepsy. These commissurotomy patients have been systematically investigated by Sperry and his associates[12] and one outstanding discovery is the uniqueness of the left hemisphere in conscious experience.[8] While the right hemisphere continues to perform at a very superior level, indeed better than the left hemisphere in pattern recognition, none of the goings on in this hemisphere give conscious experience to the person. Indeed the subject disclaims responsibility for the actions initiated within this hemisphere. Such evidence supports the view of the Nobel Laureate Sir John Eccles that activities in the right hemisphere in normal intact subjects only reach consciousness after transmission to the left hemisphere.[7]

The exclusive association of speech and consciousness with the left hemisphere raises the question: are there some special anatomical structures in this hemisphere that are not matched in the right? It has now been shown that about 80% of human brains possess anatomical asymmetries with special developments of the cerebral cortex in the regions of the speech area (Fig 5). There is hypertrophy of a part of the left superior temporal gyrus, the planum temporale. Similar asymmetries in this region have been reported in infants.[13] Why is this region of the inferior parietal lobule utilised for language?

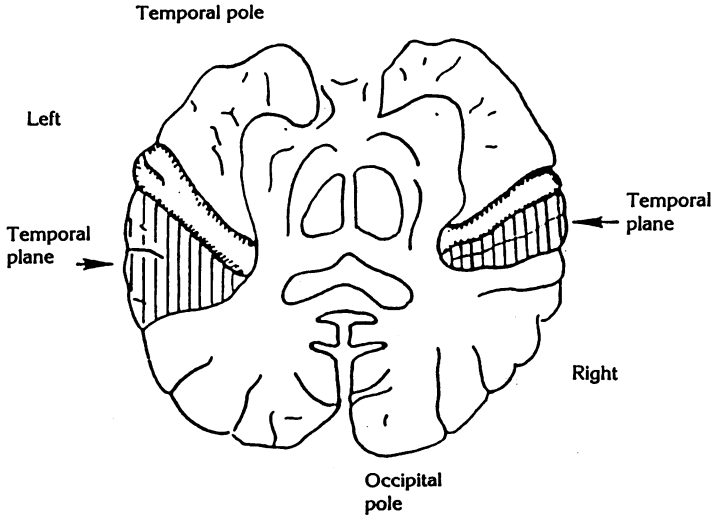

Fig 5. The upper surfaces of the temporal lobe has been exposed by a cut on each side in the plane of the Sylvian fissure. The temporal plane (vertical lines) is bordered anteriorly by the posterior border of Heschl's gyrus, posteriorly by the posterior border of the Sylvian fossa and laterally by the Sylvian fissure. Note the right-left differences in the temporal plane.

In primates this region (Broadmann areas 39, 40) has been shown to be the site where touch, visual and auditory information converge. Geschwind proposed that these area are unique in having developed to enhance the ability for cross-modal associations — a prerequisite for the acquisition of language,[14] and Teuber commented that language freed us to a large extent from the senses and gave access to concepts that combine information from different sensory modalities and is thus intersensory or supersensory.[15]

No doubt influenced by studies of clinical lesions, we have tended to focus our investigation of brain functions on specific anatomical loci. The great Russian neuropsychologist Alexander Luria cautioned on the dangers of a narrow localisationism, on the false premise that higher cognitive processes have a focal basis.[16] It seems most likely that higher cerebral functions emerge from high level neural networks which integrate and organise local brain regions. An understanding of such intermediary networks is probably critical to any further understanding of the relationship between cerebral activities and thinking, and new insights into these high level cerebral processes are beginning to emerge from modern brain imaging techniques.[17]

CONCLUSION

After this brief reflection on the self and its brain, Pinels' pioneering journey into reductionism as a physician and scientist may be less threatening — increased knowledge may not detract from human dignity. On the contrary, reductionism seeks an understanding of ourselves beyond the simple impressions of the senses. The properties of mind are determined in large part by the properties of highly organised neural networks of the brain — but not, I suggest in its entirety. Any theory of the self and its brain if it is to be effective in accounting for the vagaries of human behaviour, normal and abnormal, must also be able to account for the reality of the mind and its meanings.

A major aspiration of modern neuroscience is that further investigation of psychophysical relationships will provide a more precise description of the ways in which bodily states influence the mind and vice versa. At the very least we might hope to define the rules that determine the correlations between mental and physical events. The essence of our present position was eloquently summed up by Aristotle: "Soul and body, I suggest, react sympathetically upon each other: a change in the state of the soul produces a change in the shape of the body; and conversely a change in the shape of the body produces a change in the state of the soul".[7]

The title of this article was chosen in recognition of the valuable contribution to the topic by Sir Karl Popper and Sir John Eccles and reviewed in their book with the same title.

Figure 2 was reproduced by kind permission of the Hulton Picture Company London, and Figure 5 by Paul Eleck (Scientific Books) Ltd.

Figures 1, 3 and 4 were drawn by Mrs Yvonne Naylor.

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The changing pattern of fetal hydrops

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SUMMARY

*Fetal hydrops (*hydrops fetalis*) remains a significant cause of fetal and neonatal mortality. The decreased incidence of rhesus iso-immunisation due to prophylaxis with rhesus immune globulin (anti-D), improved antenatal ultrasound screening, and advances in neonatal intensive care have greatly altered the clinical outlook in this condition. A retrospective review of all 27 liveborn cases of hydrops in the Royal Maternity Hospital, Belfast in the period 1974 – 89 showed that in the last five years 40% of cases were non-immune in origin. The mortality rate fell from 100% in the first part of the study to 50% in the second.*

INTRODUCTION

There have been two major changes in the management of fetal hydrops in the last twenty years — the use of anti-D immunoprophylaxis which has greatly reduced the incidence of the disease and the development of perinatal intensive care. This has led to major changes in both the prenatal and postnatal management of the gravely ill fetus. We reviewed the records of all liveborn cases of fetal hydrops in the Royal Maternity Hospital, Belfast in the period 1974 – 89 to assess changes in aetiology, incidence, management, and prognosis.

METHODS

Fetal hydrops was defined as fluid accumulation in some or all serous cavities (peritoneal, pericardial, and pleural) with generalised skin and placental oedema. In the 15 year period 1974 – 89, there were 27 liveborn babies with hydrops among the 52,177 liveborn babies in the Royal Maternity Hospital, an incidence of 1 in 1932. We chose not to include stillbirths because in some cases a full assessment to ascertain cause had not been performed.

RESULTS

The specialised neonatal intensive care unit was opened in this hospital in 1978; we looked at babies managed before and after this date (Table). Of the 11 babies born in 1974 – 7 there were no survivors. In this early period, 10 were due to rhesus-D incompatibility, one to rhesus-E incompatibility, and none to other blood group incompatibilities or non-immune causes. The management of the cases of iso-immunisation, both pre- and post-natal, is outlined in the Table.

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TABLE

Clinical details and management of 27 cases of fetal hydrops

	1974–7	*1978–89*
Number of babies	11	16
Gestational age (weeks) (mean ± SD)	28·9 ± 3·2	30·9 ± 4·3
Birth weight (g) (mean ± SD)	1607 ± 741	1975 ± 802
Cause		
Rhesus-D	10 (91%)	10 (63%)
Rhesus non-D	1 (9%)	1 (6%)
Other iso-immunisation	0	1 (6%)
Non-immune	0	4 (25%)
Mortality — all causes (%)	11 (100%)	8 (50%)
Management of babies with iso-immunisation		
Number	11	12
Intra-uterine fetal transfusion	5 (45%)	5 (42%)
Exchange transfusion	10 (91%)	10 (83%)
Died in labour ward	1 (9%)	1 (8%)
Mechanical ventilation	0	11
Mortality (%)	11 (100%)	7 (58%)

In the later period, half of the babies with hydrops survived. Ten were due to rhesus-D incompatibility, 1 to combined rhesus — c and E antibodies, 1 to Kells antibody, and 4 to non-immune causes. The management of the four babies with non-immune hydrops included mechanical ventilation (in three), thoracocentesis (in two), paracentesis (in one), and treatment with digoxin and diuretics (in three). These cases were due to congenital cytomegalovirus infection, chronic feto-maternal transfusion, congenital heart disease (Ebstein's anomaly) and no cause was found after extensive investigation of the fourth leaving a diagnosis of idiopathic non-immune hydrops. Three survived, one with a mild residual hemiplegia.

DISCUSSION

In the past, the vast majority of cases of hydrops were due to iso-immunisation. Non-immune hydrops was first described in 1943 by Potter,[1] who defined it as hydrops due to any cause other than feto-maternal blood group incompatibility. Prior to 1970, Macafee[2] reported that 17% of cases of hydrops were non-immune in origin. In the last five years of our study, 40% of cases were non-immune, which has also been the experience of others.[3] Since the introduction of anti-D prophylaxis, an increasing number of cases of materno-fetal blood group incompatibility are due to groups other than rhesus-D. We have had cases of hydrops due to rhesus-E, rhesus-c, and Kells blood groups. O'Sullivan[4] found that the proportion of patients delivered in the Royal Maternity Hospital, Belfast with antibodies other than rhesus-D had risen from 11% in 1970 to 37% in 1980.

Hydrops has many causes, and new ones are reported every year. In a review by Machin[3] of 1414 cases published in the 1980's, 63% were due to five fetal disease processes: cardiovascular disorder, chromosome abnormalities, thoracic disorder, twin-twin transfusion, and anaemia. Formerly, the appearance of hydrops was considered to be a very poor prognostic factor in cases of iso-immunisation with death almost inevitable.[5] Recently, the prognosis has improved — we report a mortality rate of 50% in cases of rhesus iso-immunisation in the last eight years, and others have reported rates as low as 25%.[6] For non-immune hydrops our mortality rate was 25%, which compares favourably with reports of rates up to 80–95%.[7,8] The change in prognosis for the hydropic fetus is probably due to many factors, including delivery of the hydropic infant by elective caesarean section in a controlled setting with a neonatal team in attendance to perform intensive resuscitation.[9]

The prenatal diagnosis of hydrops is improving due to the use of high resolution ultrasound scanning. In the last eight years all cases of hydrops in pregnancies complicated by iso-immunisation were identified in the antenatal period so that management could be planned in advance. However, only 50% of our cases of non-immune hydrops were identified prior to birth. It has been suggested that certain maternal conditions can be used to predict the presence of non-immune hydrops. In one report, if maternal anaemia, hypertension or polyhydramnios had been used as indications for ultrasound examination, 80% of babies of greater than 28 weeks gestation with non-immune hydrops would have been detected.[8] If these criteria had been applied to our babies, all would have been identified prior to birth.

In the future, it is probable that the mortality rate in fetal hydrops will continue to fall due to better antenatal detection and more widespread use of cordocentesis for direct fetal intravascular transfusion in severe haemolytic disease.[10] For this to be achieved, the obstetrician must be alert to the possibility of this disease.

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The failed back syndrome: the diagnostic contribution of computed tomography

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Up to 30% of patients who undergo surgery for lumbar disc disease experience recurrent symptoms postoperatively.[1, 2] This so-called 'failed back syndrome' is a complex phenomenon influenced by organic, psychological, economic and social factors.[3, 4] Radiological examination is frequently requested in order to exclude an anatomical basis for the patient's complaints[3] (Table I). Re-exploration in the presence of significant epidural scarring (fibrosis) from previous surgery may lead to further more severe scarring,[5] although some surgeons may consider microsurgical lysis of scar tissue (DP Byrnes, personal communication). Neural compression due to recurrent disc protrusion or canal or lateral recess stenosis may be relieved surgically.[1, 6] Accurate radiological diagnosis of these and other possible abnormalities is therefore of paramount importance,[1, 7] especially in view of the difficulties in performing re-exploration of the lumbar spine.

TABLE I

Possible causes of the failed back syndrome

Epidural scar
Recurrent disc prolapse
Spinal or lateral recess stenosis
Arachnoiditis
Discitis
Facet joint arthropathy
Painful disc degeneration without rupture
Spondylolisthesis
Pseudomeningocele

Myelographic appearances following surgery are non-specific, and in addition, myelography is insensitive to the effects of unsuspected bony changes, such as lateral recess stenosis.[3, 8] Experience with high resolution computed tomography (CT) has led to its preference in the radiological investigation of the failed back syndrome.[9] Use of intravenous radiological contrast media has been reported further to improve accuracy.[7, 10–15] In this report we describe our experience with CT scanning in 100 consecutive patients with the failed back syndrome.

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MATERIALS AND METHODS

Between October 1987 and December 1989 102 consecutive CT scans were performed on 100 patients at 131 disc levels where surgery had been carried out. The initial operation had been for disc prolapse on 125 occasions and at six levels laminectomy had been performed for spinal stenosis. The patients were aged between 19 and 68 years, and scans were performed between six months and 13 years after the previous surgery, except in two cases scanned within two weeks of operation. The scans were performed on a Siemens Somatom DR3 scanner. Contiguous slices 4 mm thick were obtained from the pedicle above to the pedicle below each disc space, angled parallel to the disc itself. All three lower lumbar discs were included regardless of the level of previous surgery. At 86 levels, scans were repeated following an intravenous bolus of 100 ml contrast medium containing 300 mg/ml iodine (Niopam 300, E Merck Ltd).

The CT scans were analysed for the presence of any significant abnormality involving the nerve roots or spinal canal. Where a soft tissue mass was seen at the disc margin with possible compromise of the adjacent root, differentiation of recurrent disc herniation from scar tissue was based on published criteria[9] (Table II, Fig 1). Scans were also classified as showing a mixture of disc fragment and scar tissue, or as indeterminate.

Scans were classified as showing scar posteriorly at the laminectomy site or in the adjacent posterolateral spinal canal (not involving a nerve root) only if no other clinically significant abnormality was present. The CT density of any abnormal tissue was measured using a computer generated region-of-interest facility on the scanner video monitor.

TABLE II

Differentiating features of scar tissue and recurrent disc on CT

CT feature	*Scar*	*Recurrent disc*
Site	May be above or below disc space and may be continuous with posterior scar	Usually contiguous with disc margin
Shape	Typically follows contour of thecal sac and may cause retraction of it	Mass which indents thecal sac and displaces nerve root
Density	< 50 CT units	> 65 CT units
Enhancement	Usually present	Absent

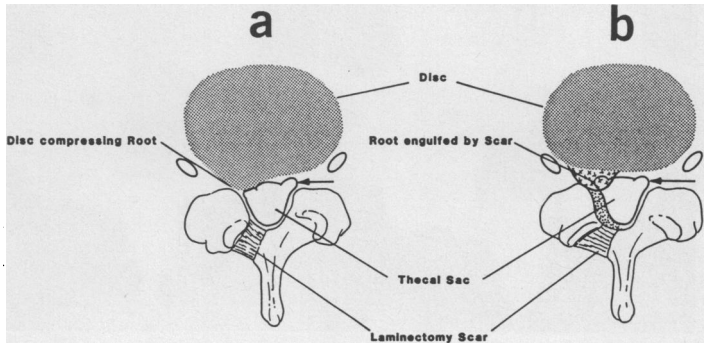

Fig 1. Diagrammatic representation of recurrent disc protrusion (a) and pure postoperative epidural fibrosis (b). Arrow = normal nerve root emerging from thecal sac. Note scar tissue engulfing root on side of previous surgery in (b) (cross-hatching), also scar extending along margin of thecal sac (stippled area).

Changes in abnormal tissue following intravenous contrast were assessed subjectively and objectively using the region-of-interest facility. The phenomenon of 'enhancement' of tissue on CT following intravenous contrast medium depends to a large extent on the vascularity of the tissue, and also on its tendency to accumulate the medium.[16] In theory, such media should enter vascular scar tissue causing a rise in CT density (enhancement), whereas avascular disc material should show no change. Definite enhancement was diagnosed when the CT density rose by at least 10 units following contrast administration. Clinical follow-up was obtained in all patients and where subsequent re-exploration was carried out, the surgical findings were compared with the CT diagnosis.

RESULTS

CT diagnoses at the 131 previously explored disc levels are shown in Table III, and illustrated in Figs 2–6.

TABLE III

CT diagnoses at previously explored disc levels

Diagnosis		*Number (%)*
Recurrent disc protrusion (Fig 2)		18 (14%)
Fibrosis:— at disc margin (Fig 3)		29 (22%)
— posteriorly		43 (33%)
Disc fragment and fibrosis (Fig 4)		16 (22%)
Spinal or lateral recess stenosis (Fig 5)		10 (8%)
Other (Fig 6)		8 (6%)
Disc bulge only	3	
Facet arthropathy	2	
Spondylolisthesis	1	
Discitis	1	
Equivocal		7 (5%)
	Total	131

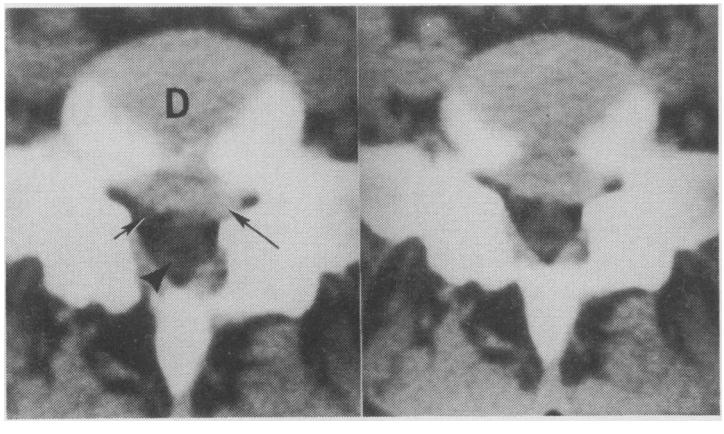

Fig 2 (a) and (b). Recurrent disc protrusion. CT scans before (a) and after (b) intravenous contrast. Non-enhancing disc material displaces adjacent root (long arrow): compare normal root on opposite side (short arrow). Arrowhead = thecal sac.

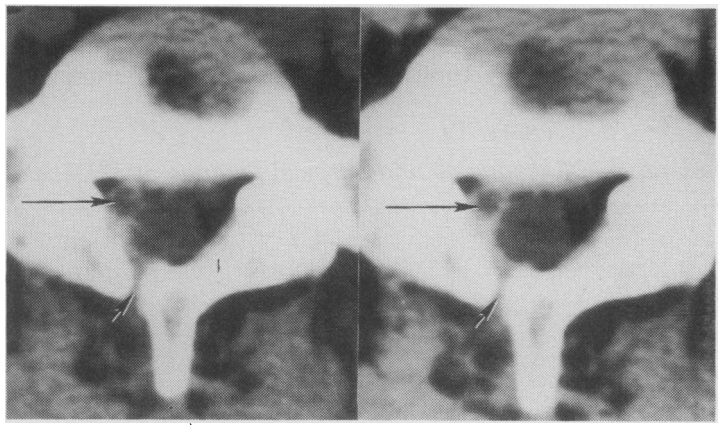

Fig 3 (a) and (b). Pure epidural fibrosis. CT scans before (a) and after (b) intravenous contrast. Non-enhancing nerve root (long arrow) is surrounded by enhancing fibrosis. Note enhancing tissue extending along margin of thecal sac to laminectomy scar (short arrow).

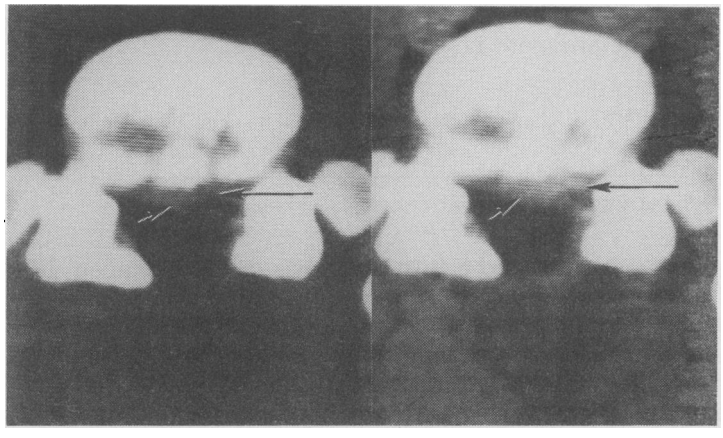

Fig 4 (a) and (b). Mixed recurrent disc protrusion and epidural fibrosis. CT scans before (a) and after (b) intravenous contrast. Non-enhancing disc material indents thecal sac (short arrow); enhancing fibrosis is seen laterally around nerve root (long arrow).

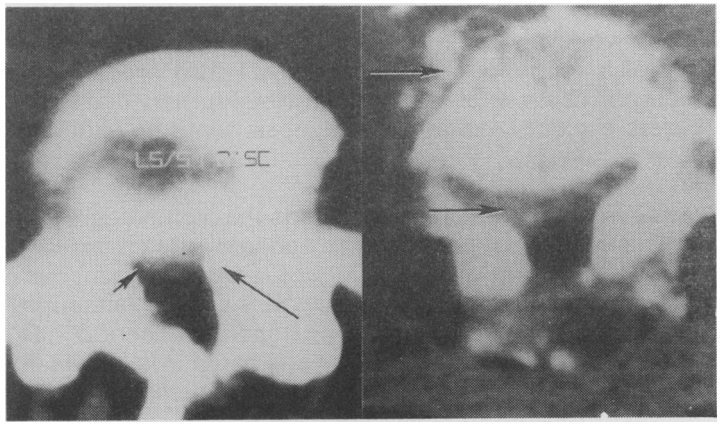

Fig 5 (left). Lateral recess stenosis. CT scan shows massive hypertrophy of the right interfacetal joint with encroachment on the lateral recess and nerve root (long arrow). Compare normal side (short arrow).

Fig 6 (right). Postoperative infective discitis. CT scan shows abnormal soft tissue and multiple fragments of bone around the margins of the narrowed disc space (arrows).

In 34 patients, subsequent re-exploration was carried out at 36 levels. The results of both the unenhanced and enhanced scans at these levels were compared with the surgical findings in order to assess the contribution of intravenous contrast to the accuracy of CT (Table IV).

TABLE IV

Re-exploration: comparison of CT diagnosis and surgical findings at 36 levels

	CT diagnosis	*Confirmed at surgery*
Unenhanced CT scan		
Recurrent disc prolapse	8	6
Epidural fibrosis	10	8
Spinal or lateral recess stenosis	1	1
Bone fragment	1	1
Disc bulge only	1	1
Enhanced CT scan (indeterminate on unenhanced scan)		
Epidural fibrosis	6	4
Disc fragment and fibrosis	5	5
Indeterminate	3	*

*All 3 of these levels were found at surgery to have disc fragments and fibrosis.

Use of intravenous contrast never altered a firm diagnosis on unenhanced CT, which was correct at 17 out of 21 levels. There were two false positives for disc recurrence. At one level, an epidural vein was found at surgery, but due to patient discomfort a delay occurred between contrast administration and repeat scanning which may have been responsible for the apparent non-enhancement in this case; at the other level, suspected recess stenosis was confirmed but the presumed disc recurrence was not. In two cases of presumed epidural fibrosis on CT, small disc fragments were found embedded in scar tissue. In one case a recurrent disc protrusion was correctly diagnosed, but a pseudomeningocele was missed.

Fifteen levels were indeterminate on unenhanced scans. One patient had magnetic resonance imaging (MRI) instead of enhanced CT. In the other 14, enhanced CT gave a correct diagnosis at nine out of 11 levels, with three remaining indeterminate. At two levels where CT predicted pure epidural scar, small disc fragments were also found at re-exploration. CT also predicted a further significant abnormality at 12 levels at which no previous surgery had been performed. Five of these were explored and the CT diagnosis confirmed at four.

DISCUSSION

Our experience with CT scanning confirms previous reports that some degree of epidural scarring occurs in the majority of patients who undergo surgical exploration of the lumbar spine.[7, 9, 17, 18] In contrast, the incidence of true recurrent disc herniation is low.[7, 9] The exact mechanism of scar formation is unclear, although scanning of asymptomatic volunteers in the early postoperative period has suggested that it may be linked to the soft tissue swelling and haemorrhage which occur acutely in most cases.[17, 19] Although microsurgical techniques have been

advocated as a method of reducing the incidence of postoperative epidural scarring,[20] no direct correlation of CT findings with the type of surgery has yet been demonstrated.[12]

Where epidural scarring is confined to the laminectomy site and/or the lateral spinal canal, it is probably of no clinical significance, and no diagnostic difficulty should be encountered on CT scanning.[9] However, where a soft tissue mass is seen adjacent to the disc margin, differentiation from recurrent disc herniation must be made. Teplick and Haskin in 1983 based this on the known appearances of virgin disc prolapse on CT.[9] Disc material was characteristically high in density, and indented adjacent structures, whereas scar tissue was usually low in density and caused retraction of adjacent structures. However, several authors also described "mass-like" scars which could simulate disc material,[10-12] and in addition the accuracy of CT density measurements in the postoperative spine was questioned.[13, 16] Use of intravenous contrast to improve diagnosis was first described in 1982,[10] the assumption being that contrast medium administered intravenously should not enter avascular disc material but would enter the highly vascularised scar tissue, which would therefore show enhancement on CT. Many papers followed confirming this experience and recommending contrast, usually at high dose, in all cases where a possible disc recurrence was seen.[12-14, 21]

Dixon subsequently reported that use of intravenous contrast did not alter a firm diagnosis of disc or scar on unenhanced CT.[22] He suggested its use only in those cases where unenhanced CT was equivocal, and also questioned the need for very high doses of contrast. Our experience is similar; use of contrast never altered a firm diagnosis on unenhanced CT, and we also routinely were able to identify enhancement using a moderate dose of contrast. Reserving contrast for use in indeterminate cases reduces costs and avoids the morbidity associated with its use. Retrospective review of our case material has led to a more selective use of contrast in our department.

Our accuracy rate for distinguishing disc from scar was 72% at 32 levels (excluding the patient who had MRI instead of enhanced CT). To our knowledge, this represents the largest reported series of surgically confirmed cases. Other authors have reported an accuracy of 74% (23 cases),[13] 67% (number unspecified)[15] and 100% (13 cases of disc prolapse only).[21] In all other series the number with surgical confirmation has been ten or less. This compares with an accuracy rate for CT and magnetic resonance imaging in virgin disc prolapse of 72-97%.[23]

The most difficult scans to interpret are those in which the appearances are not absolutely characteristic of scar or disc material. Typically these are cases in which a soft tissue mass is seen adjacent to the disc which is intermediate in density between scar and disc, which does not cause marked indentation of the thecal sac, and which enhances unhomogeneously. The differential diagnosis is then between nerve root surrounded by scar tissue and a disc fragment embedded in scar. It has been suggested that this is usually not difficult,[11, 12] but we agree with others that this distinction sometimes cannot be made reliably and that the radiological report should state that this is so.[13, 14] Failure to detect such fragments within scar was responsible for four of our incorrect diagnoses on CT, and inability to exclude their presence was responsible for our indeterminate scans. However, CT was always correct in predicting the presence of significant scar, whether or not a small disc fragment was also present. There were no false negatives for "true" recurrent disc and only two false positives.

Earlier reports indicated that unsuspected spinal canal or lateral recess stenosis were common causes of the failed back syndrome,[1] but our series agrees with more recent reports that such abnormalities are found infrequently.[9,24] This may be related to the widespread adoption of CT as the primary radiological investigation for lumbar disc disease, as lateral recess stenosis is more accurately demonstrated by this method than by myelography. Accurate demonstration of facet joint pathology is a further advantage of CT. The severity of disease may be a guide to the likely outcome from facet anaesthesia in patients with appropriate symptomatology.[25, 26] Other causes of failed back syndrome are rarely seen. Conflicting reports have appeared concerning the value of CT in early discitis. Reliable diagnosis is probably only possible when frank infection with soft tissue swelling and vertebral end plate destruction are present as in our case. CT appears to be as sensitive as plain films and radionuclide scanning in such cases, but magnetic resonance imaging may be more sensitive in so-called "aseptic" discitis.[27] Pseudomeningocele due to dural tear at surgery is also a rare finding and in retrospect should have been suspected from the CT scan in our case. Myelography was diagnostic but would have been required in any case for planning of surgery; it should be noted that not all pseudomeningoceles will fill with myelographic contrast material.[9]

Although we found a low incidence of pathology at levels not previously explored, we continue to scan all three lower lumbar discs routinely in view of the risk of missing a lesion not demonstrated on previous radiological studies. A particular consideration would be an unsuspected far lateral disc protrusion, where only myelography was previously performed.[28] Although we advocate CT as the preferred investigation, there is no doubt that myelography still has a place. It remains the only reliable method of diagnosing arachnoiditis,[8] although CT and MRI changes have been described.[12, 29] Myelography also allows a more dynamic study of the lumbar spine which may be important in cases of suspected spinal instability and in spondylolisthesis. CT has been combined with myelography but to no definite advantage.[7, 8]

Recent reports comparing MRI with CT have indicated its superiority in differentiating scar and recurrent disc, especially when gadolinium-DTPA enhancement is used.[24, 30] MRI also detects the early changes of disc degeneration without rupture,[31] although discography may still be required as a diagnostic provocative test.[32, 33] The availability of MRI is likely to remain restricted and in addition CT remains better for demonstrating bony changes including the facet joints and lateral recesses.[32] Our current practice is to refer for MRI only those patients in whom CT is negative or equivocal.

The surgical decision to re-explore must only be based on the radiological findings in conjunction with the history and physical signs. With careful selection, the results of re-exploration can be rewarding,[6] and CT with the selective use of intravenous contrast offers an accurate non-invasive method of helping to make this difficult decision.

We wish to thank our neurosurgical and orthopaedic colleagues for referring patients to us and for providing details of their operative findings. Figure 1 was prepared by Mr Brendan Ellis of the Department of Medical Illustration.

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Cardiac pacing in Northern Ireland 1979 – 1988

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SUMMARY

The implantation rate of cardiac pacemakers in Northern Ireland rose from 55·8 per million population in 1979 to over 180 per million in 1988, with the emergence of a second pacemaker implantation centre. However, the implantation rate in Northern Ireland remains less than that of many other countries. Over the period complete heart block fell as an indication from 45% to 34% and sinus node disease rose from 16% to 28%. Symptoms necessitating pacing remained unchanged, syncope being the most common. Ventricular demand pacemakers constituted a lower proportion of implants than anywhere else in the world and more atrial demand and dual chamber pacemakers were implanted than in most other countries. Insufficient patients are being referred for pacing in Northern Ireland but a high proportion of those who are referred receive modern sophisticated pacemakers.

INTRODUCTION

The first fully implantable cardiac pacemaker was inserted in 1959[1] and the first implant in Northern Ireland was in 1969 by Dr J Geddes. Since then there have been rapid advances in pacemaker technology, including the development of stable atrial and ventricular leads that can be inserted intravenously, and the development of smaller and longer lasting pacemakers with sophisticated functions which can be implanted under local anaesthetic with minimal morbidity. Permanent pacemaker implantation has become a relatively straightforward technique and the indications for pacing are no longer restricted to life-threatening bradycardias. This has resulted in a rapid increase in the numbers of pacemakers implanted worldwide.

This paper documents the change in pacing practice in Northern Ireland over the ten year period 1979 – 1988 and compares with other parts of the United Kingdom and the rest of the world.

METHODS

Permanent pacemakers are implanted at two centres in Northern Ireland: the Belfast City Hospital and the Royal Victoria Hospital. These hospitals register the details of permanent pacemaker implantations with the British Pacing Group database. Data for the two hospitals were obtained from the British Pacing Group with permission from the cardiologists in each hospital. This was compared with hospital records to exclude duplications, omissions or other inaccuracies.

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Sex, age at pacemaker implantation, electrocardiographic indications for implantation, symptoms requiring pacing, and pacing modes employed were recorded. These results were compared with published world data for 1986.[2]

RESULTS

Only first time implantations are considered. The total number of pacemakers implanted over the ten year period in both centres was 1935, with the greater proportion in the Royal Victoria Hospital (63%). The numbers implanted annually increased from 87 in 1979 to 290 in 1988 (Table I). The proportion implanted in the Belfast City Hospital rose from 17% to 53% over this period. The initial implantation rate per million population was 55·8 in 1979 rising to 152 in 1986 and 185·9 in 1988 (Northern Ireland population 1·56 million — 1981 census). The overall United Kingdom figures are 111 per million in 1979 and 148 in 1986. The average age at implantation has gradually increased from approximately 67 in 1980 to nearly 72 in 1988, with a slight excess of males in most years.

TABLE I

Numbers of first time pacemaker implantations in two hospitals in Northern Ireland, and implantation rate per million population

	Pacemaker numbers			*Implantation rate*
Year	*RVH*	*BCH*	*Total*	*(per million)*
1979	72	15	87	55·8
1980	98	26	124	79·5
1981	105	31	136	86·6
1982	143	35	178	114·1
1983	124	56	180	115·4
1984	131	81	212	135·9
1985	150	85	235	150·6
1986	141	96	237	151·9
1987	122	134	256	164·1
1988	137	153	290	185·9
Total	1223	712	1935	124·0

Indications for pacing

The main indications for pacing were heart block (including complete heart block, second and first degree heart block, bundle branch block and atrial fibrillation with bradycardia) and sinus node disease. The number of pacemakers implanted for complete heart block gradually increased over the first five year period but remained approximately 95 per year thereafter. The relative frequency of this diagnosis fell from 44·8% to 33·8% of the total over the period. There was a rise in numbers of pacemakers implanted for sinus node disease from 14 in 1979 (16·1%) to 78 in 1988 (27·8%). No patients were paced for carotid sinus hypersensitivity in 1979 whereas in 1988 this indication accounted for 5·3% of the total. There was no change in the frequency of implantation for other forms of heart block, with second degree heart block accounting for approximately 9%, bundle branch block 4%, and atrial fibrillation with bradycardia 6% throughout the period.

Symptoms

Symptoms at the time of pacing have remained fairly constant. Syncope has been the most frequent indication, around 50% for the whole period. Dizzy spells have risen from 12% to 20%. These two symptoms represent definite indications for pacing about which there is little controversy (if they are caused by bradycardia). Less definite symptoms such as bradycardia alone have decreased over the ten year period from 18% to 6%. These three symptoms have consistently accounted for more than 75% of those implanted, but their proportion has decreased as other indications, such as pacing for tachycardia have increased.

Pacing modes

This is the area where there has been the greatest change over the ten year period. In 1979 single chamber ventricular demand pacing was the predominant mode of pacing and accounted for 87% of the total, the other two modes used being atrioventricular sequential (DVI) and atrial demand (AAI).

Over the period the frequency of these three modes of pacing reduced and this was associated with the development and use of other more sophisticated modes of pacing — two more dual chamber modes (DDD and DDI), rate responsive pacing (VVIR) and antitachycardia pacing (Table II).

TABLE II

Different pacing modes used in 1979 and 1988

		Pacemakers implanted (%)			
Mode		*1979*		*1988*	
Single chamber:					
Ventricular demand	(VVI)	76	(87)	120	(40)
Atrial demand	(AAI)	5	(5)	22	(8)
Dual chamber:					
Atrioventricular sequential	(DVI)	6	(7)	25	(9)
	(DDD)	0	(0)	89	(31)
	(DDI)	0	(0)	11	(4)
Rate responsive	(VVIR)	0	(0)	20	(7)
Others		0	(0)	3	(1)

UK and world data

In 1986 the implantation rate per million population was lower in Northern Ireland and in the UK as a whole, compared with most other Western countries (Figure). Ventricular demand mode accounted for a much lower proportion of the total in Northern Ireland, meaning that the more sophisticated atrial demand and dual chamber pacemakers were used more frequently than in most other countries. Symptoms resulting in pacemaker implantation differed. In Northern Ireland and in the UK as a whole relatively small numbers were implanted for symptoms such as bradycardia, whereas this indication was more common in those countries with higher implantation rates.

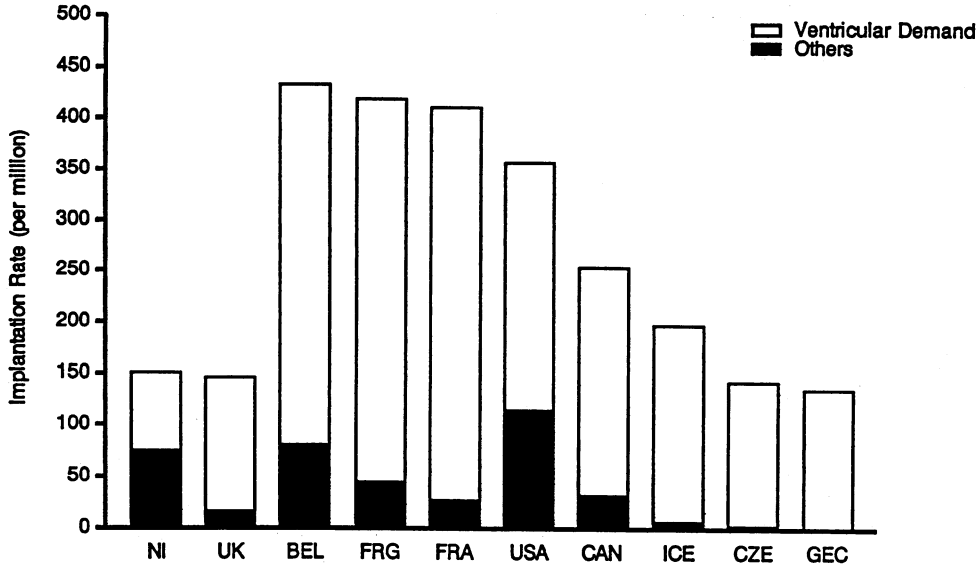

Figure. Implantation rates, and proportion ventricular demand mode, for Northern Ireland and the UK compared with other countries for 1986. (Bel = Belgium, FRG = West Germany, Fra = France, Can = Canada, Ice = Iceland, Cze = Czechoslovakia, Gec = Greece).

DISCUSSION

During the ten year period many advances have occurred in pacemaker technology, and permanent pacemaker implantation is now a straightforward procedure performed under local anaesthesia, with a minimal morbidity and mortality. There is now no doubt that mortality in patients with complete heart block, whether symptomatic or not, is significantly improved with pacing and all patients with this condition should be paced.[3] Second degree heart block is also a frequent indication. Mobitz type II block occurs when the block is below the level of the atrioventricular node and usually progresses to higher degree atrioventricular block with Stokes Adams attacks and the risk of sudden death.[4] Chronic type I second degree block (as opposed to the transient type that occurs following an inferior myocardial infarction) was generally believed to have a relatively benign course, and pacing was considered only for extreme bradycardia or if the patient was symptomatic.[5] Recent work would suggest that the prognosis in this condition is not so good and pacing should be considered.[6]

Patients with bifascicular block are at higher risk of development of high-grade atrioventricular block,[7] but the best means of predicting those most at risk is not clear. At present patients with bifascicular block and a prolonged PR interval with a history of syncope for which no other cause can be found probably require pacing.[8] It is generally agreed that mortality in sinus node disease has not been shown to be improved by pacing, and that patients with this condition should only be paced for relief of such symptoms as syncope and heart failure.[9] Carotid sinus hypersensitivity resulting in syncope is now another frequent indication for pacing, accounting for 5·3% of all cases in Northern Ireland in 1988. Pacing improves symptoms in a significant proportion of these patients.[10] Tachycardia control and termination represents a small but significant proportion of pacemakers implanted.

The implantation rate in Northern Ireland and throughout the UK is much lower than in many countries throughout the world. The most recent figures available are for 1986, when most western European and North American countries were implanting more than 200 per million, and some countries more than 400 per million population.[2] The reasons for these differences in implantation rates are not clear. Possible explanations are a lower prevalence of conditions requiring pacemaker implantation in the UK, underdiagnosis or undertreatment of conditions requiring pacing, or overimplantation in other countries.

The high rate of bradycardia as a symptom and sinus node disease as an indication for pacing in some countries with high implantation rates may reflect inappropriate pacing of asymptomatic patients with sinus node disease. It was suggested in 1984 that too many pacemakers were being implanted in the USA.[11] This resulted in the introduction of stricter criteria for pacemaker implantation and was followed by a reduction in the implantation rate in that country from 518 per million in 1981 to 359 per million in 1986. This figure is still over twice that of the UK for that year.

A reason for the lower implantation rate in the UK may be a different prevalence of conditions requiring pacing. Shaw and Kekwick[12] carried out an eight year survey in the Devon area in which they asked general practitioners to report patients who they thought had abnormal bradycardias. They included complete heart block and sinus node disease as indications for pacemaker implantation and found an incidence of complete heart block of 97 per million and symptomatic sinus node disease of 77 per million, a total rate of 174 patients per million requiring pacemaker implantation per year for these conditions. In Northern Ireland in 1988 the implantation rate was 20 per million for second degree heart block, 15 per million for atrial fibrillation and bradycardia, 10 per million for carotid sinus hypersensitivity, 3 per million for bundle branch block and 20 per million for other indications. Taken with the 174 per million estimated by Shaw and Kekwick for complete heart block and sinus node disease, this would suggest that a total implantation rate of 250 – 300 per million would be more appropriate.

Thus the discrepancy between the implantation rates in the UK and other countries may be due to a degree of overtreatment in those countries, but it also seems that there is undertreatment of patients with conditions requiring a permanent pacemaker in the UK. Examination of data from Northern Ireland may suggest reasons why this should be so. Prior to 1983 pacing in Northern Ireland was performed predominantly in one centre, the Royal Victoria Hospital, where 143 implants were performed in 1982. In that year only 35 implants were performed in the Belfast City Hospital. However, over the next five years the number of implantations in the latter hospital increased to 153 in 1988, but there has not been any reduction in the numbers implanted in the RVH. This would suggest that the implantation rates reflect the number of centres performing implantation, greater availability of a pacing service resulting in a greater referral for pacing. It may be that the UK practice of implanting pacemakers in regional centres specialising in pacing gives rise to lower referral rates for investigation and treatment of bradycardias and syncope than if pacing were to be performed in district general hospitals. It has been suggested that the higher implantation rate in the USA may be due to the widespread availability of a pacing centre in almost every community hospital.[13]

Fewer ventricular demand pacemakers are implanted in Northern Ireland than in the UK or any other country, but more atrial demand and dual chamber

pacemakers are used. The high implantation rate for these latter types of pacemakers probably reflects the fact that in Northern Ireland pacing is carried out in two specialised cardiology units with particular interests in pacing.

I would like to thank the cardiologists in both the Belfast City Hospital and Royal Victoria Hospital, in particular Dr D B O'Keeffe and Dr S Webb, for their help in compiling these data. This paper was presented to the Irish Cardiac Society in December 1989.

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Clinical Q fever in Northern Ireland 1962–1989

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SUMMARY

Q fever was diagnosed in 443 patients in Northern Ireland between 1962 and 1989. From 1986 onwards there was an increase, which peaked in 1989 with 107 cases of whom 47 were infected in Ballycastle, Co Antrim. There were three outbreaks and 21 clusters of patients with Q fever. Most cases were in April and May which correlated with the peak lambing and calving season. Q fever mainly affected males in the 40–49 year old age group. County Antrim had the highest prevalence rate of 40/100,000 population and also had the most sheep. The number of sheep in Northern Ireland has doubled in the past ten years.

Q fever was strongly associated with occupation and animal contact. Eighty-seven patients (19·6%) drank unpasteurised milk. The commonest presenting illnesses were pneumonia (62·8%), influenza-like illness (24·6%), involvement of the heart (9·0%) and hepatitis (1·6%). Thirty-two patients (7·2%) had endocarditis, 20 of whom had prosthetic valves and three of whom died. Coxiella burnetii *was present on valves removed from seven patients.*

INTRODUCTION

Unexplained fevers in Brisbane abattoir workers were investigated by Derrick in 1937.[1] An organism was isolated by guinea pig inoculation and serial passage, and later shown by Burnet to be a rickettsia. Derrick named the disease Q fever and the organism *Rickettsia burneti.*[2] It was subsequently renamed *Coxiella burnetii* because it had different properties from the rickettsia.[3] Q fever is now known to have a worldwide distribution. Investigation of Belfast abattoir workers in 1957 had shown that they lacked antibody indicating that the animals they slaughtered up to that time were not infected,[4] and the first patient known to have had Q fever in Northern Ireland was identified in 1962.[5] This paper reports the Northern Ireland experience since that date.

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MATERIALS AND METHODS

Serological methods. Complement fixation tests were performed using the method of Bradstreet and Taylor[6] and the Microtiter system. Q fever phase 2 antigen was used for diagnosing acute infections and in seroprevalence surveys. Phase 1 antigen was used for diagnosing chronic infections. Sera from chronic infections and inoculated guinea pigs were titrated to high titre to avoid prozone effects. All titres are expressed as reciprocals. A four-fold or greater rise of phase 2 antibody indicated recent infection. A titre of $\geqslant 160$ indicated infection in the recent past provided there was supporting clinical or epidemiological evidence indicative of recent infection. In seroprevalence surveys a titre of 8 or 10 indicated infection in the past. The presence of phase 1 antibody at a titre of > 200 indicated chronic infection.[7] Antigens and antisera were supplied by the Division of Microbiological Reagents and Quality Control, Public Health Laboratory Service, Central Public Health Laboratory, London NW9 5DF.

Isolation of C. burnetii. Natural heart valves were homogenised in 5 ml buffered saline. Prosthetic valves were rinsed in buffered saline and any material attached was homogenised. Groups of four guinea pigs were inoculated with each valve suspension using 1 ml intraperitoneally. The guinea pigs were bled before inoculation and again 24 days later.

A surveillance form was sent to the doctor caring for each clinical case asking for details of the age, sex, address, occupation, animal contact (especially if parturient), unpasteurised milk drinking, travel abroad in month before onset, chest X-ray and final clinical diagnosis.

RESULTS

There were 443 patients with Q fever diagnosed between February 1962 and December 1989. There were 6 deaths (1·4%) including one suicide after the illness. The annual incidence of Q fever is shown in Fig 1.

There was a moderate peak in 1976 and a greater increase from 1986 onwards peaking in 1989 with 107 cases. The month of onset of illness is shown in Fig 2. There was a marked rise in April with a peak in May.

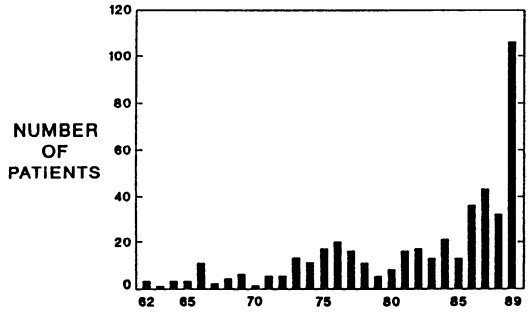

Fig 1. Annual incidence of Q fever.

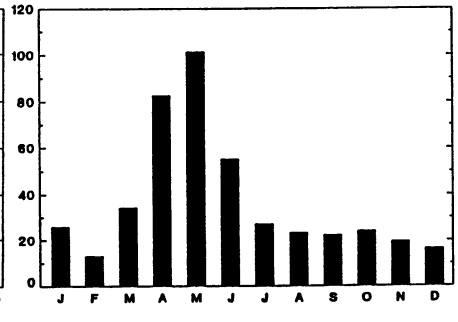

Fig 2. Monthly incidence of Q fever.

The age and sex of the patients is shown in the Table. There were 346 males and 97 females. Age ranged from 3 to 84 years and the peak incidence was in the 40–49 year old age group.

TABLE

Age and sex distribution of patients with Q fever

Age (Yrs)	*Male*	*Female*	*Total*
0 – 9	1	0	1
10 – 19	14	0	14
20 – 29	46	17	63
30 – 39	65	20	85
40 – 49	88	21	109
50 – 59	70	23	93
60 – 69	39	12	51
70 – 79	18	2	20
80 – 89	5	2	7
Total	346	97	443

Location

The number of patients in each county was:- Antrim 141, Down 93, Londonderry including the city 71, Armagh 51, Tyrone 33, and Fermanagh 4. There were 50 cases in Belfast city. The prevalence rate was 40 per 100,000 in Antrim, 39 per 100,000 in Londonderry including the city, 38 per 100,000 in Armagh, 30 per 100,000 in Down, 24 per 100,000 in Tyrone, 8 per 100,000 in Fermanagh, and 14 per 100,000 in Belfast city. Three patients were infected outside Northern Ireland, in Majorca, Cyprus and Sudan.

Outbreaks and clusters

The original outbreak on a farm on the eastern Ards peninsula in Co Down during 1965/66 has been described.[5] The second outbreak was in a Belfast animal research laboratory during March 1986. Two animal laboratory technicians each had pneumonia and an influenza-like illness and three others were found to have Q fever antibody out of 11 tested. The source was not proven, although sheep and calves were in the unit. The third outbreak was in Ballycastle, Co Antrim during April — June 1989 when 47 people became ill. The origin of the outbreak was not found although sheep were known to be lambing in fields around the town. Clustering of patients in time and place was present in Limavady during April — June 1988 (8 cases), Londonderry during April — June 1989 (5 cases), Kilkeel during April and May 1989 (5 cases) and Rostrevor during March and April 1976 (4 cases). In addition 17 towns or villages had clusters of two or three patients each. Belfast was not included in the study of clusters.

Occupation

Occupations where farm animal contact occurred and the numbers affected were farmers (72), farmers' wives (17), farmers' daughters (2), farm labourers (10), abattoir workers (14), butchers (3), a veterinary surgeon and a veterinary assistant. Eleven other people had visited farms in the course of their work, including animal feed and other salesmen, Tourist Board inspectors and milk tanker drivers. Others who had animal contact outside farms included cattle dealers, graders, herders and livestock truck drivers (9), animal laboratory technicians (2),

a pig dealer, a sheep skin assessor, a sheep skin painter, a fur factory worker, a taxidermist, a dog handler and a horserider. Seven doctors, eight nurses, two dentists and a dental technician developed Q fever.

Some occupations listed in our survey had no obvious animal contact yet this may have occurred indirectly such as house builders, customs officers, a docker's wife, a dustman's wife, an evangelist in a tent mission, forestry workers, housewives who visited farm shops, truck drivers, policemen, soldiers, postmen, telephone engineers, a tyre fitter, a farm vehicle mechanic and those who holidayed in caravans or farms.

In 1976, 90 workers in an Omagh Co Tyrone abattoir, and 73 workers in a Belfast abattoir were bled. Fifteen (17%) of the Omagh workers and 23 (31·5%) of the Belfast workers had Q fever antibody in their sera. In 1986, 406 farmers from all over Northern Ireland were tested and 114 (28%) had Q fever antibody in their sera.

Animal contact and milk consumption

The animal contact and number of patients in contact were as follows: – cattle (137), dogs (127), sheep (101), cats (25), horses (17), pigs (16), goats (9) and fowl (5). Eighteen patients had unspecified farm animal contact. One hundred and eleven patients (25%) had no animal contact recorded although 11 of these drank unpasteurised milk.

Pasteurised milk was consumed by 331 patients and unpasteurised milk by 87 patients, three of whom drank goat's milk. Among the unpasteurised milk drinkers were farmers, farmers' wives or farm labourers (42), abattoir workers (2), a cattle grader and a farm salesman. Unpasteurised milk was consumed from February 1962 through to November 1989. No information was available from 25 patients.

Clinical illness

The presenting illness with the number and percentage of patients affected were: – pneumonia 278, (62·8%); influenza-like illness/pyrexia of unknown origin 109, (24·6%); endocarditis 32, (7·2%); hepatitis 7, (1·6%); myocarditis 4, (0·9%); pericarditis 4, (0·9%); meningitis 2, (0·5%); others 7 (1·6%).

Additional complications of the presenting illness included hepatitis (23); chest pain (4); myocarditis (2) including one death; pericarditis (2); auricular fibrillation (3); arthritis (3); and one patient each with meningism, popliteal nerve palsy, parotid swelling, lymphadenitis, purpura, spontaneous rupture of the spleen, and unsuspected pneumonia.

Endocarditis

Thirty-two patients had endocarditis between 1974 and 1989. One patient had high phase 1 antibody only and the rest had high phase 1 and phase 2 Q fever antibody titres. One patient had high phase 1 and 2 Q fever antibody twelve weeks before developing symptoms and signs of endocarditis.[8] Eighteen were males and 14 were females. Their ages ranged from 23 to 74 years, but half the cases were in the 50 – 69 year old age group. The incidence of endocarditis in Q fever infected patients fell from a peak of 37·5% in 1981 to 1·9% in 1989. The valves infected were aortic (18), mitral (9) and both of these (5). Twenty of these valves were prosthetic, one was a homograft and eleven were natural. Two

female patients had immunological or neoplastic abnormalities: one a serum paraprotein band, the other a non-Hodgkins lymphoma and had progressed from an acute Q fever pneumonia to chronic endocarditis in seven months. No other patient had a previous history of acute Q fever. There were three deaths (9·4%) in this sub-group of patients aged 48, 67 and 71 years. Nineteen of the endocarditis patients had contact with cattle or sheep and four of these also drank unpasteurised milk. Three additional patients drank unpasteurised milk who had no animal contact. Valves removed at surgery from seven patients were available for isolation of *C. burnetii* and all were positive. Three of these valves were prosthetic, one was a homograft and three were natural.

DISCUSSION

The patients investigated in this study were sufficiently ill to consult their general practitioners or be admitted to hospital. Many other patients with minor illnesses or no illness must also have been infected with Q fever. This disease is now endemic in Northern Ireland, which has become second only to the south-western region of England in the number of cases reported in the United Kingdom.[9] The increasing yearly incidence of Q fever may be related to the number of farm animals. In 1990 there were 1,439,231 cattle and 2,073,111 sheep in Northern Ireland and the human population was 1·56 million. The number of sheep has doubled in the last ten years. Sixty-one per cent of all calving occurs between January and April with a peak in March, and 80% of all lambing takes place in March and April. Calving and lambing probably both contribute to the peak incidence of Q fever in April and May.

Q fever predominantly affects males with a peak incidence in the 40 – 49 year old age group. It is unexplained why younger age groups have a lower incidence. Geographically the highest prevalence rates for Q fever were in counties Antrim and Londonderry (including the city). County Antrim has 565,590 sheep which is the highest number in any county, while County Londonderry was second highest with 476,604 sheep.

Several outbreaks have been described on farms, including one in Northern Ireland.[5] The danger of infection with *Coxiella burnetii* in laboratory workers despite the use of safety precautions is well known[10] and includes personnel who worked with sheep.[11] The Ballycastle outbreak may have originated from surrounding fields where lambing was taking place. *Coxiella burnetii* is excreted in milk, urine, faeces and birth fluids of infected animals. The animal placenta may contain 10^9 infectious doses per gram of tissue. Humans are at greatest risk of exposure at parturition of livestock because primary aerosols containing large numbers of *Coxiella burnetii* are shed at that time.[12] *Coxiella burnetii* has been recovered from soil and surface water of fields used by parturient sheep and viable organisms were continuously present in soil for periods up to 150 days.[13] The concentration in the soil was such as to suggest that secondary dust aerosols may lead to infection of man and livestock in the absence of active shedding of the organisms by infected animal hosts. In guinea pigs, and probably also in humans a single inhaled organism is sufficient to initiate infection.[14]

The extreme resistance of the organism to drying and to physical and chemical agents means that it can survive for long periods in the environment.[15] All of these factors contribute to the successful transmission of Q fever infection. Since Northern Ireland has large rural areas the clustering of cases in towns and villages could be explained in the same way. Q fever has also been described in an urban

area where there was no direct contact with farm animals but farm vehicles had probably disseminated contaminated straw, manure or dust in the area.[16] A large outbreak of 136 cases in Birmingham occurred in 1989 although there was no obvious animal contact.[17] Occupation was strongly associated with Q fever: those who worked, lived on or visited farms or worked with farm animals were affected while indirect contact with farm animals probably played an important part in other occupations. Farm animal contact was predominantly with cattle and sheep while pigs, horses, goats and fowl were less often associated with Q fever. Dogs (domestic[5] or stray[18]) may carry *Coxiella burnetii* into the house. Parturient cats[19] and wild rabbits[20] have also been associated with Q fever infections. One quarter of all patients had no recorded animal contact. Unpasteurised milk was consumed by 87 patients up until 1989. Forty-two of these patients were farmers, farmers' wives or farm workers who presumably drank milk from their own cows or goats. There are now only four licensed farm producers of unpasteurised milk in Northern Ireland. Outbreaks of Q fever associated with drinking raw milk have been described.[21]

The seroprevalence studies in Co Tyrone abattoir workers showed an increase from 5·9% in 1966[5] to 17% in 1976. This may indicate a greater spread of Q fever in farm animals in that county. There was a fall in the number of Belfast abattoir workers with Q fever antibody from 71·7% in 1966[5] to 31·5% in 1976, but 33 of these workers in 1966 were aged 40–63 years and in 1976 only ten men were over the age of 30 years. The seroprevalence of farmers throughout Northern Ireland has increased slightly from 23·1% in 1965–67[5] to 28% in 1986 although past infection is now more widespread geographically.

Overall the heart was involved in 47 patients (10·6%) and there were four deaths in this group. In patients with endocarditis who have repeatedly negative blood cultures it is important to test for Q fever phase 1 and 2 antibody since the antibiotic treatment of Q fever endocarditis differs from other bacterial causes.[22] The incidence of endocarditis in Q fever infected patients has fallen in England, Wales and Ireland from 13% in 1976–78 to 4% in 1985–87.[9] Our endocarditis patients were on average at least 10 years older than the peak incidence of acute Q fever, and data from England and Wales also suggest that there is a 5–10 year delay between acute infection and the diagnosis of chronic Q fever endocarditis.[23] Only one of our patients had a previous history of acute Q fever but she also had a non-Hodgkins lymphoma. Only eleven endocarditis patients (34%) had natural valves. Two-thirds of the endocarditis patients had contact with cattle or sheep or drank unpasteurised milk, which are high risk factors for patients with prosthetic valves.[24] The presence of *Coxiella burnetii* on valves from seven patients confirms its causative role in their endocarditis, as chronic Q fever infection of the liver may also produce high phase 1 and phase 2 antibody titres.[25] Pericarditis[26] and myocarditis[27] associated with Q fever have been previously described.

Pneumonia was the commonest presenting illness, followed by an influenza-like illness or pyrexia of unknown origin. Hepatitis is a well known complication[28] and was present in 30 patients (6·8%). Meningitis, encephalitis, rashes, lymphadenitis,[29] and meningoencephalitis associated with nerve palsies[30] have been described. Spontaneous rupture of the spleen in a patient with Q fever pneumonia was first described in a patient in the Northern Ireland series.[31]

The control of Q fever presents difficult problems. The farm animals do not show any obvious signs of infection, so they cannot be segregated from people. Although *Coxiella burnetii* is present in milk, urine and faeces of an infected

animal the placenta is the most infectious product. The placenta should be disposed of properly and not left in the open air, which presents huge problems because of the large numbers involved. All milk for human consumption should be pasteurised. Ticks may transmit Q fever but there is no evidence that they do so in Northern Ireland. The commonest method of infection is by inhalation of infected aerosols or dust but dealing with this in the workplace is very difficult. There is at present no respiratory protective equipment approved specifically by the Health & Safety Executive for work involving microbiological risk.[32] The only effective protection would be a "moonsuit" or a high efficiency full face ventilated visor or blouse with an independent positive pressure air supply. The cost of purchase and maintenance, and the inability to do heavy work while wearing such equipment makes it impracticable. In an abattoir, infected material may be accidentally swallowed or contaminate the eyes. *Coxiella burnetii* may be inoculated through cuts in the skin or by thorns entangled in the wool of sheep. Control of infection should be by good general working methods, facilities, hygiene and education. In particular the workers should be made aware of the possibilities of infection and the need to seek prompt medical attention. An abattoir should provide proper washing facilities, protective clothing and suitable ventilation to help disperse aerosols which are generated. Segregation of pregnant animals to a separate area should diminish the risk of infection to other workers in the rest of the abattoir. Training workers in health and safety should help to reduce the risk of infection in abattoirs and in farms as well. A Q fever vaccine has been used in Australian abattoir workers[33] but it is not licensed for use in the United Kingdom. Research work on farm animals, particularly sheep, should be confined to laboratories dedicated solely for that purpose and all isolation procedures for *Coxiella burnetii* must use a containment level 3 laboratory.[32]

While it should be inappropriate to advise people to stay away from the countryside, it would be sensible to avoid farm animals at parturition, to stop drinking unpasteurised milk and to constrain household pets from running about in the fields. Pregnant women, immunosuppressed persons[28] and those with damaged or prosthetic heart valves[24] should take particular care to avoid at-risk work and leisure activities.

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Preoperative measurement of haemoglobin concentration

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SUMMARY

Haemoglobin concentrations were estimated in 1158 patients admitted to hospital for elective surgical procedures. Haemoglobin concentration below 10 g/dl was not seen in male patients who were fit and healthy, or had mild or severe non life threatening systemic diseases. In female patients haemoglobin concentration below 10 g/dl was seen in 0·3% of normal fit healthy patients, 2% of patients with mild systemic diseases, 6·4% of patients with severe, non life threatening diseases, and 18·7% of those with severe incapacitating diseases. Routine haemoglobin measurement is unnecessary in fit healthy patients, or in those with mild systemic disease (American Society of Anesthesiologists grades 1 or 2), but should be done in those with more severe systemic diseases (American Society of Anesthesiologists grades 3, 4 or 5).

INTRODUCTION

Measurement of the haemoglobin concentration in blood remains one of the most frequently performed preoperative investigations. It is of value for many reasons: it may aid diagnosis, especially when accompanied by an indication of red cell morphology; it may give an indication that intra or postoperative blood transfusion will be needed; it may screen for unexpected anaemia; and lastly, it may influence the anaesthetic technique.

In the anaemic patient, cellular oxygen demands may only be satisfied if cardiac output is increased (with an increase in stroke volume),[1] and hypoxaemia may accompany the fall in cardiac output associated with the induction of anaesthesia.[2] A fall in haemoglobin concentration is associated with an increase in cardiac output and conversely, any increase in haemoglobin concentration is associated with an increase in viscosity and a fall in tissue perfusion. A packed cell volume of 0·30, or a haemoglobin concentration of 10·0 g/dl probably gives optimal tissue perfusion and oxygen carriage. The majority of anaesthetists would consider a haemoglobin concentration of 10·0 g/dl as the lower acceptable limit prior to elective surgery, unless compensation has occurred as in the case of chronic renal failure. Some would accept values as low as 9·0 g/dl.[3] Carson and colleagues[4] demonstrated that in patients who declined transfusion, the operative mortality was 7% when preoperative haemoglobin concentration was above 10 g/dl, but 61% if the concentration was below 6 g/dl.

Dundee[5] has shown that the induction dose of sodium thiopentone is related to the preoperative haemoglobin concentration by the formula: Log dose of thiopentone = 0·995 + 0·019 × Hb. The patient with a low haemoglobin

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concentration is therefore at increased risk from anaesthesia. Routine measurement means testing many healthy patients unnecessarily, and has been suggested that age may be a useful criterion for requesting this investigation. The aim of this study was to evaluate the American Society of Anesthesiologists (ASA) grading system as a criterion for haemoglobin measurement prior to anaesthesia and surgery.

METHODS

1158 (704 female, 454 male) patients admitted for elective surgical procedures at this hospital over a 9 month period were studied. Those with known active bleeding were excluded. Each patient was allocated an ASA grade depending on the preoperative history and examination (Table I). A routine 2·5 ml sample of venous whole blood was obtained for laboratory haemoglobin measurement using an automated Coulter counter.

TABLE I

American Society of Anesthesiologists grading system (ASA)[9]

1. Normal, fit healthy patient.
2. Mild systemic disease.
3. Severe systemic disease, not a constant threat to life.
4. Severe, incapacitating systemic disease which constitutes a constant threat to life.
5. Moribund, not expected to live for 24 hours with or without surgery.

RESULTS

The number and mean age of patients allocated to each group are shown in Table II. No patients in grade 5 were seen as the surgery was elective in nature. Ages ranged from 6 months to 98 years. No male patient in ASA grade 1 to 3 had a haemoglobin concentration below 10 g/dl. In the female patients, many of whom were of reproductive age, haemoglobin concentrations of less than 10 g/dl were seen in 0·3% of those of ASA grade 1, 2% of grade 2, 6·4% of grade 3 and 18·7% of grade 4. The mean haemoglobin concentration in the 454 males was 14·2 (+/− 0·14) g/dl, and in the 704 females 13·0 (+/− 0·09) g/dl.

TABLE II

Mean age and haemoglobin concentration in 1158 patients admitted for elective surgery, classified by ASA grades of preoperative fitness. The percentage of patients with haemoglobin concentrations below 10 g/dl and above 17 g/dl is shown

ASA grade		*n*	*Age*	*Hb g/dl (SEM)*	*Range*	*<10 g/dl*	*>17 g/dl*
1	Male	174	35	14·6 (0·1)	11·1 – 17·9	0%	2·3%
	Female	366	33	13·1 (0·1)	9·9 – 17·5	0·3%	0·3%
2	Male	118	55	14·3 (0·1)	10·4 – 17·4	0%	2·5%
	Female	197	47	13·0 (0·1)	9·4 – 16·7	2·0%	0%
3	Male	133	59	13·8 (0·2)	10·3 – 18·2	0%	2·2%
	Female	125	61	12·7 (1·7)	8·7 – 17·9	6·4%	1·6%
4	Male	29	69	13·4 (0·4)	8·9 – 17·9	3·5%	3·4%
	Female	16	77	12·2 (0·6)	8·2 – 16·7	18·7%	0%

DISCUSSION

Less than 1% of patients in this study had haemoglobin concentrations below 10 g/dl, in agreement with the results of Walton [6] and of Gold and Wolfersberger.[7] The low incidence of anaemia implies that this investigation is performed unnecessarily in many preoperative patients. In the absence of bleeding, and prior to procedures in which blood loss is expected to be low, this test is unlikely to be of value in the fit and healthy (ASA grade 1) patient. Apparently healthy patients sometimes present for surgery and are found to be anaemic, but a careful history and examination will usually show symptoms such as lethargy or breathlessness, and signs of a hyperdynamic circulation such as tachycardia and bounding pulse at rest, which exclude them from ASA grade 1. In the absence of these signs, circulatory compensation for chronic anaemia (as in chronic renal failure) with increase in plasma volume and rightward shift of the oxygen haemoglobin dissociation curve means that anaesthesia is likely to be relatively well tolerated.

The higher incidence of low haemoglobin concentration in ASA grades 3 and 4 indicates that this investigation should be performed in all preoperative patients in these groups. In other words, all those with signs and symptoms of systemic disease. A raised haemoglobin concentration also leads to increase in perioperative risk from thromboembolism, and may be due to polycythaemia or to dehydration. A haemoglobin concentration above 17 g/dl may indicate the need for preoperative venesection or fluid replacement depending on the aetiological cause. This occurred in only two of 704 female patients in this study, but in from two to four percent of male patients regardless of ASA grade.

It is presently recommended that haemoglobin concentration should be measured preoperatively in those over 40 years,[8] or in the presence of cardiovascular or respiratory disease. Chronological age, however, gives no indication of physical health, and some young patients have severe systemic disease. The ASA guide is a logical, easily learnt patient classification system, and gives an accurate indication as to when measurement of haemoglobin concentration is likely to give results which will affect clinical practice. This classification is commonly performed by the anaesthetist, but could easily be performed as well by the house surgeon. It may still be necessary to measure the haemoglobin concentration for diagnosis or assessment of treatment, but the vast number of tests routinely requested as a preoperative check can safely be reduced.

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Visual analogue measurement of pain

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SUMMARY

Two separate studies were carried out to determine if three visual analogue scales for various feelings including pain could be marked consistently by patients, without reference to previously completed scales.

Sixty patients undergoing extraction of their lower third molars had measurements of acute preoperative anxiety, expected postoperative pain and postoperative perceived pain three times in quick succession. There was no significant difference between the three measurements for any of the feelings. Although a correlation was detected between expected pain and preoperative anxiety, there was no meaningful relationship between perceived postoperative pain and expected pain or preoperative anxiety.

Eighty patients suffering from a wide range of chronic painful states completed three identical scales for pain, anxiety, depression and mood during their first visit. These measurements were repeated at a later time following a treatment, with the addition of a visual analogue scale for pain relief. Mean scores for anxiety, mood and pain relief were consistent, but mean pain scores were more variable. There was a very close correlation between any two feelings expressed on these visual analogue scales during both the initial and second visits. Litigation or social problems were not associated with increased pain scores.

INTRODUCTION

It is widely accepted that subjective methods of determining the "pain experience" are the most valid.[1, 2] Various mood changes occur in the chronic pain situation, in particular depression.[3, 4] Anxiety, on the other hand, tends to be associated with acute pain.[5, 6] Visual analogue scales have been shown to be a simple, reliable means of allowing patients to express their feelings with a high degree of resolution, without resorting to cumbersome questionnaires.[1, 7] The primary aim of this study was to determine if these scales can be marked consistently by patients suffering pain in various situations and to determine if any relationship exists between the expressed emotions and pain.

METHODS — Study 1

Sixty consecutive patients requiring surgical extraction of their lower third molars were admitted to the study. Patients with mental or physical disabilities rendering them incapable of using a visual analogue scale were excluded. Informed verbal

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consent was obtained. They were not premedicated with a sedative. Immediately prior to the induction of anaesthesia the patients indicated their anxiety and their expected pain using a 10 cm visual analogue scale. Each patient completed these scales twice more without visual reference to the previous scores, so that a total of three independent measures of anxiety and expected pain was obtained in quick succession. Anaesthesia was induced with propofol followed by muscle relaxants to facilitate intubation. Maintenance of anaesthesia was with halothane in nitrous oxide and oxygen. In the recovery room, approximately 30 minutes after the operation, postoperative pain was measured three times, in quick succession, prior to any analgesia being given. The visual analogue scales were : –

Anxiety	: Totally relaxed	— Extremely anxious.
Expected pain	: No pain expected	— Worst pain possible expected.
Postoperative pain	: No pain	— Worst pain possible.

When the operation was over the surgical difficulty was graded — 1. Simple elevation without bone removal, 2. Simple elevation with minimal bone removal, 3. Wide bone removal or tooth section, 4. Wide bone removal and tooth section.

METHODS — Study 2

Eighty consecutive patients referred to a pain clinic were studied. All had suffered pain for four or more weeks but had received little or no benefit from their current therapy. Following interview and examination, the patients were asked to indicate their present pain intensity and psychological state on four 10 cm visual analogue scales. The scales were arranged so that all good experiences were to the same (left) side to avoid confusion during use. Each visual analogue scale was explained as a "thermometer of feeling" and a means of conveying these feelings to others. They were asked to mark the line between the two extremes of experience, to indicate how they felt at that moment. The scales were : –

Pain	: No pain	— Worst pain experienced.
Anxiety	: Totally relaxed	— Extremely anxious.
Depression	: Elated	— Extremely depressed.
Mood	: Feeling great	— Feeling rotten.

Immediately on completion of the four scales the procedure was repeated twice more in quick succession, without visual reference to previous scales. A treatment was then carried out and the three sets of four scales were again completed at a later time, with the addition of a pain relief scale which ranged from complete pain relief to no pain relief. A record was also kept of the diagnosis, pain duration, sleep disturbance, co-existing problems such as litigation, social and psychiatric factors and employment status.

The marking of visual analogue scales can lead to skewing of results at either end of the scale. To correct this and promote a normal distribution, arcsin transformation was carried out on all visual analogue data, permitting parametric statistics to be used.[7] Following transformation, the data was analysed using one-way analysis of variance. Pearson correlation coefficients for untransformed data was used to detect relationships between pairs of variables.

RESULTS

In Study 1 there were 39 female and 21 male patients aged 16 – 33 years (mean 22·4 ± 3·8 years). There were no significant differences on the first visual

analogue scale presentation between the sexes, between preoperative expected pain or anxiety or postoperative pain. In view of this, the data for males and females has been pooled for all subsequent analyses. Neither was there significant drifting in the marking of the visual analogue scales for any of the three feelings observed, so that the first measure could be used in each case in subsequent analyses.

For each of the four grades of surgical difficulty the mean scores for preoperative anxiety and expected pain and postoperative perceived pain varied from 41 to 68 but analysis of variance showed no significant differences (Table I). There was a trend, which did not reach significance, for the mean expected pain scores to be higher in the more difficult cases. The only significant correlation between the three variables was obtained between preoperative expected pain and preoperative anxiety ($r = 0 \cdot 27$, $p = 0 \cdot 02$), but this accounted for only 7% of the total variation.

TABLE I — Study 1

Mean results (± SD) on each visual analogue scale by the four grades of increasing surgical difficulty (n = 60). Untransformed scores; the analysis of variance was carried out on arcsin transformed scores

	Surgical difficulty				Analysis of variance	
	1	*2*	*3*	*4*	*F*	*p*
n	5	32	17	6		
Expected pain (± SD)	43 (± 3)	52 (± 4)	63 (± 7)	68 (± 10)	1·91	0·14
Preoperative anxiety (± SD)	50 (± 3)	58 (± 6)	41 (± 8)	45 (± 2)	1·91	0·14
Postoperative pain (± SD)	49 (± 4)	52 (± 7)	51 (± 7)	46 (± 10)	0·08	0·97

In Study 2, the chronic pain population aged 17 to 78 years, the 80 patients included 19 (24%) with neuralgias, 16 (20%) with back pain and 8 (10%) with carcinoma. The pain had been present for more than one year in 56 (70%) of patients. No obvious co-existing problems were detected using standard clinical interviews in 49 (61%) of the patients, but 12 (15%) were in the process of litigation related to their pain. A further 16 (20%) had social problems such as marital difficulties. Thirty-one (39%) reported a sleep disturbance and in nine (11%) sleep was grossly upset. Thirty-six were in employment (two self-employed), three were unemployed, 15 were housewives, 16 retired and 10 were receiving sickness benefit. The mean time interval between the first and second consultation was 32 days (range 1 hour — 260 days).

There were some significant differences between the first, second and third tests both at the first and second visits. The first result was lower than the other two for pain scored at the initial visit (Table II). Using transformed scores, analysis of variance showed $F = 4 \cdot 49$; $df = 2,158$; $p < 0 \cdot 05$ between the first, second and third tests. The mean depression scores showed a different pattern before and after treatment, $F = 5 \cdot 98$; $df = 2,158$; $p < 0 \cdot 005$, the first test result being raised in comparison with the two subsequent tests at the initial visit but not at the second visit. Anxiety, mood and estimated pain relief scores were stable throughout the three tests on both visits and the second set of tests has been used for subsequent analysis.

TABLE II — Study 2

Mean scores (± SD) for each feeling (n = 80). Tests 1 — 3 were carried out at the initial visit and tests 4 — 6 during the second visit. (Untransformed data)

Test	*Pain*	*Anxiety*	*Depression*	*Mood*	*Pain relief*
1	51 (±28)	49 (±26)	55 (±22)	52 (±26)	—
2	52 (±26)	49 (±23)	52 (±24)	51 (±23)	—
3	52 (±25)	49 (±23)	51 (±23)	52 (±23)	—
4	44 (±25)	45 (±24)	51 (±23)	49 (±23)	49 (±26)
5	47 (±24)	48 (±24)	51 (±23)	49 (±25)	49 (±25)
6	47 (±24)	46 (±23)	51 (±23)	51 (±25)	49 (±26)

A stepwise regression was carried out on the variables measured on the first visit with pain as the independent variable. Depression was eliminated as it did not contribute significantly to the regression ($p = 0{\cdot}65$). The final relationship was expressed as: Pain = 0·51 (Mood) + 0·32 (Anxiety) + 10·49. (t – test for significance, Mood $p = 0{\cdot}001$; Anxiety $p = 0{\cdot}007$; constant $p = 0{\cdot}105$). This regression accounted for 38% of the variance. Similarly, the relationship for pain relief was determined, eliminating depression ($p = 0{\cdot}49$); Pain relief = 0·45 (Mood) + 0·24 (Anxiety) + 15·35. (t – test for significance, Mood $p = 0{\cdot}001$; Anxiety $p = 0{\cdot}051$; constant $p = 0{\cdot}007$), this regression accounting for 37·1% of the variance. Pearson correlation coefficients for the untransformed data from these sets of tests indicated a very close relationship between all combinations of the feelings measured, ($r = 0{\cdot}48$ to $0{\cdot}79$, $p < 0{\cdot}001$ in each case).

The mean scores on each of the scales in relation to litigation and social problems are shown in Table III. Although there was a tendency for less pain and anxiety in the litigation group, none of the differences reached significance. Analysis of variance to consider differences due to age, duration of pain, employment status or sleep disturbance revealed no significant effect.

TABLE III — Study 2

Untransformed mean (± SD) scores of each visual analogue scale for legal or social coexisting factors. (n = 77, 3 unclassified). The analysis of variance was carried out on transformed data (df = 2,74)

	n	*Pain*	*Anxiety*	*Mood*	*Depression*
Nil	49	54 (±29)	49 (±26)	49 (±26)	48 (±26)
Legal	12	46 (±24)	45 (±16)	54 (±14)	55 (±14)
Social	16	52 (±20)	53 (±18)	51 (±18)	58 (±21)
Total	77	52 (±26)	49 (±23)	50 (±23)	51 (±24)
F		0·58	0·52	0·16	0·85
p		0·56	0·60	0·86	0·43

DISCUSSION

The study of acute pain patients indicated that the visual analogue scales for each feeling were marked consistently and that one scale for each variable should suffice. This was not the case in the study of patients suffering chronic pain, where anxiety, mood and pain relief were consistently marked but pain and depression were not. Although the change detected for the latter two modalities was small (approximately 2 – 3% in each case), this may be of practical importance in attempting to detect small but meaningful changes which may be important in assessing chronic pain.

It was surprising that the difficulty of the surgical procedure did not correlate with postoperative pain. This may be because psychological factors play the major role in pain perception, or because the surgical grading scale was too insensitive. Correlation between expected pain and anxiety was not significant, which differs from the results observed by Martinez-Urrutia,[6] possibly because our scales didn't measure what was intended. Although there was a significant correlation ($p = 0 \cdot 02$) between expected pain and anxiety, this relationship is not likely to be clinically significant as it only accounted for 7% of the variance. Postoperative pain did not correlate with either preoperative expected pain or preoperative anxiety, but might have correlated with postoperative anxiety had this been measured. These results do not necessarily contradict the well established relationship between pain and anxiety.[8] In the study of acute pain the patients all knew its cause. In many previous studies individuals have had to interpret the meaning of their pain, and their anxiety may have been related to the significance and meaning of the pain itself, whereas in this study anxiety was more likely to have been directed towards the procedure.

In the chronic pain study, a relationship was found between pain and anxiety. Here the patients did have to interpret the meaning of the pain, even though there may have been some explanation. The relationship obtained between pain, pain relief and depression would correspond with previous studies, where a depressive affect accounted for a third of pain or pain relief. All of the variables correlated in the chronic pain study and these were analysed to determine if a relationship existed between depression and pain during the first visit, and pain relief during the second visit. A depressive affect was eliminated in each case, perhaps due to patients preferring to express the depression associated with chronic pain as poor mood and not as frank depression.

It is often assumed that in the presence of litigation pain may be exaggerated, perhaps unconsciously.[9] There is increasing evidence that the persistence of pain symptoms is a function of the delay in the settlement of litigation.[10, 11] The present results would indicate that pain is expressed as no more severe by those awaiting a court case than in those with social problems, or by those with no other contemporary problems. It would be important to extend the study of the perception of pain to include the time period both before and after the court case.

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Incidence and site distribution of colorectal cancer in Northern Ireland

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SUMMARY

Death rates from colorectal cancer in Northern Ireland are higher than in most of the rest of the United Kingdom. Although local surgeons have recognised this problem for some time it has remained unclear whether this reflects a greater underlying incidence or a worse mortality. We have reviewed all histological diagnoses of colorectal cancer in the province over a three year period and we report the incidence and site distribution for this disease in this population of one and a half million.

With the exception of rectal cancer in females the incidence of colorectal cancer, whether histologically diagnosed or registered, is higher than in England, Wales or Scotland. The site distribution accords with that in other high risk countries. These results indicate that Northern Ireland has the highest underlying incidence of colorectal cancer in the United Kingdom.

INTRODUCTION

It is known that Northern Ireland has one of the highest death rates from colorectal cancer in the United Kingdom;[1] reliable province-wide incidence data has not previously been reported. There has been a local cancer registry since 1959, but its data-base has not been routinely collated by the International Association of Cancer Registries for inclusion in the World Health Organisation's "Cancer Incidence in Five Continents".[2]

Mortality trends for these two cancers in Northern Ireland have not followed the pattern of post-war decline in the rest of the United Kingdom.[3] Internationally, mortality and incidence for this disease show a high correlation[4] but there are known to be significant variations in incidence between relatively small areas within the UK.[5] As part of a larger study into the epidemiology of hereditary bowel cancer, we report the age and sex specific incidence, and site distribution, of all histologically confirmed bowel tumours diagnosed over a three year period in the 1·5 million population of Northern Ireland.

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METHODS

All new histological diagnoses of cancer of the colon and rectum were reviewed from the topographically indexed records of the three histopathology laboratories in the province between 1976 and 1978. From the operative request and the pathology report, details of the age and sex of the patient and the site of the primary tumour were obtained. Metachronous or recurrent cancers during the period were not included and only the site of the first growth (or the largest if synchronous tumours were present) was recorded. The cancers were assigned to five anatomical sites: caecum and ascending colon; transverse colon (including hepatic and splenic flexures); descending colon; sigmoid colon; rectum and anal canal. In accordance with the International Classification of Diseases, "recto-sigmoid" junction growths were routinely assigned to the rectal group. Mid-year population estimates (in five year age groups) for 1976–78 were obtained from the Northern Ireland Registrar General's Office, and age-standardised incidence rates were computed using a truncated world standard population.[2]

For comparison purposes, an age and sex breakdown of colorectal cancer registrations of new cases notified between 1976 and 1978 was requested from the local cancer register. Incidence was therefore calculated both for histologically confirmed cases and for registered cases. Comparisons were made with registration data from England, Wales and Scotland.

Analysis of variance was used to compare the mean age at diagnosis of cases at different bowel sites. A z statistic and a chi-square statistic were calculated to compare incidence rates and site distribution respectively between men and women.

RESULTS

There were 1241 histologically diagnosed cases and 1847 new cases registered over the period 1976–78. For two of the laboratory cases no age was given and they have been excluded from further analysis. The average age of patients with histologically confirmed colon and rectal cancer was 65·3 yrs (range 23–99) and 66·2 yrs (range 20–99) respectively. Although at relatively young ages the incidence of colon cancer in women appeared to exceed that in men, the difference in the 25–44 yrs age group was not significant. With the exception of the youngest age groups (for whom rates are based on small numbers of cases) the incidence of rectal cancer in men appeared higher than that in women. The difference between the sexes was not significant. (Fig 1).

Women have a greater proportion of proximal disease (ascending and transverse) than men (28·4% versus 19·1%). The differences in the proportions of proximal and distal disease were significant (chi-square = 13·56, df = 1, $p < 0·001$). The average age of females with proximal colon cancers (ascending and transverse) was some five years greater than those with distal disease and so the site distribution has been represented for each sex separately in two broad age categories (Fig 2). The proportion of proximal disease seems to increase with age for females but not for males. For males the distribution is dominated by the proportion of rectal cancer.

The age-standardised incidence (in age groups 35–64 yrs) of histologically confirmed cancer and of registered cancer is shown in the Table, with data from cancer registrations for England, Wales and Scotland shown for comparison.

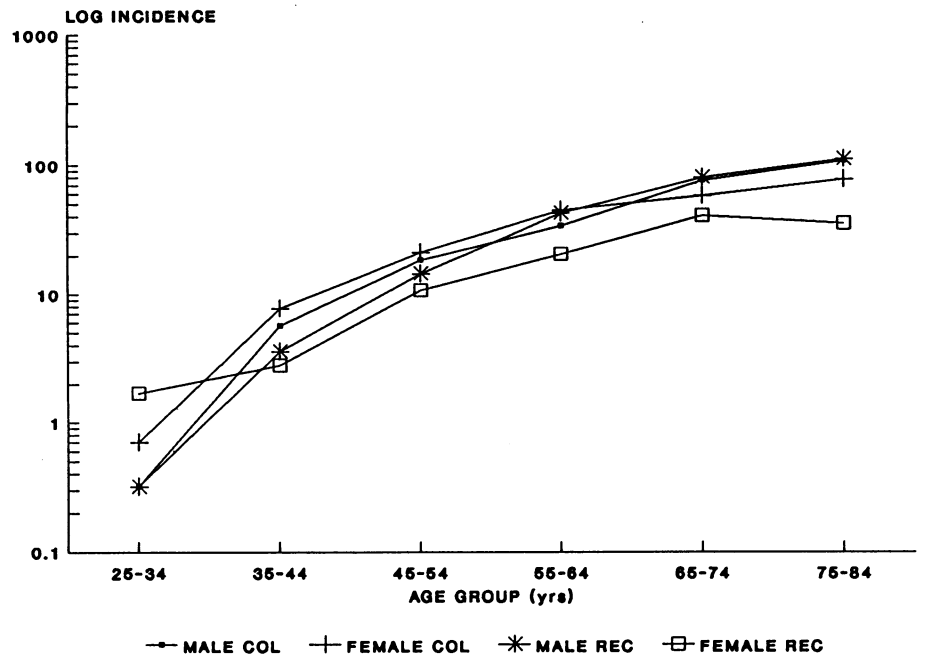

Fig 1. Age specific incidence of histologically confirmed colorectal cancer (per 100,000)

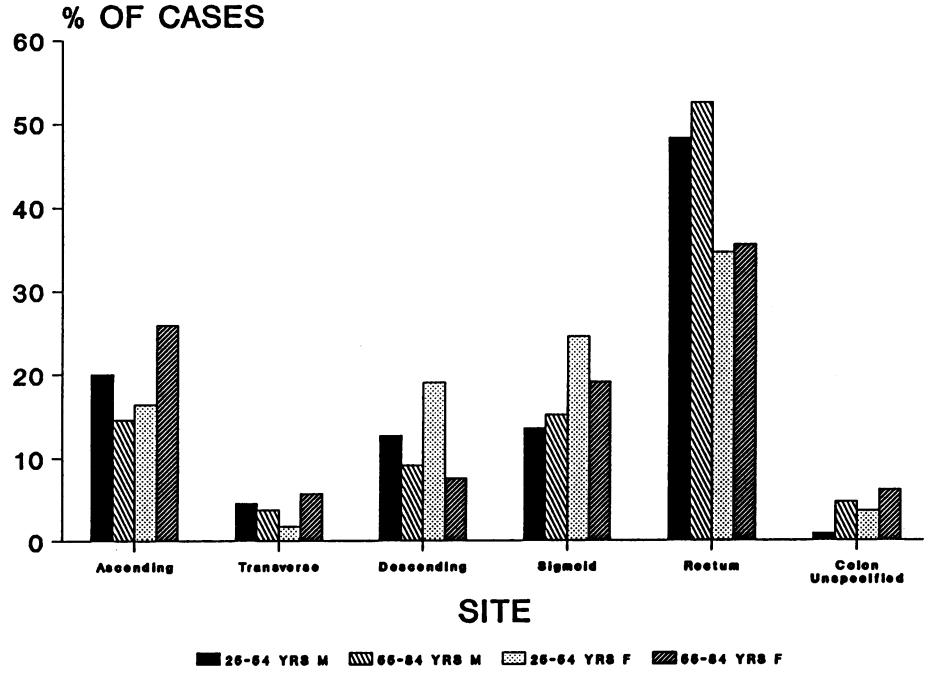

Fig 2. Site distribution of histologically confirmed bowel tumours in males and females (proportions calculated separately for each sex)

The incidence in Northern Ireland of laboratory confirmed colon cancer in females and of rectal cancer in males is higher that equivalent registered rates in the rest of the UK. With the exception of rectal cancer in females, the incidence of registered colorectal cancer in Northern Ireland is the highest in the UK.

TABLE

Age standardised incidence (per 100,000) of colorectal cancer in the United Kingdom (35–64 yrs)

	Colon		Rectum	
	M	*F*	*M*	*F*
Northern Ireland (histological)	17·5	22·2	19·1	10·2
Northern Ireland (registered)	26·1	26·6	16·6	9·8
Scotland* (period 1978–82)	22·2	21·3	14·8	10·8
England & Wales* (period 1979–82)	17·6	17·1	15·2	9·6

*Source: Cancer Incidence in Five Continents, Vol V. World Health Organisation 1987. Data based on pooled rates from contributing registries.

DISCUSSION

These observations have provided incidence data on histologically confirmed colorectal cancer in Northern Ireland, and also the opportunity to contrast these findings with the data recorded in the Northern Ireland Cancer Register. The concern that colorectal cancer is a particularly serious problem in Northern Ireland has been substantiated, and our findings come at an opportune time in view of the current interest in strategies for colorectal cancer prevention.[6] There is growing evidence that proximal and distal cancers may represent biologically distinct diseases,[7] and there has been renewed attention on regional variations in site distribution within the bowel.

It is not possible reliably to assess the site distribution of colorectal cancer from the Northern Ireland Cancer Register because over half of all colon registrations have site unspecified. However the findings of the histological case review and the pattern of age-specific incidence are consistent with the concept that the dominant aetiological influences may differ in the two sexes.[5,8] Women in Northern Ireland have higher rates of colon cancer at younger ages than men. Although the difference in rates is not significant the same pattern has been confirmed in many western developed countries in North America, Europe and Australia.[8] Local women have a greater proportion of proximal disease than men. This general distribution pattern is congruent with those described for medium and high risk countries such as in Great Britain, Scandinavia and New Zealand which has reputedly the highest mortality in the world.[9] Gender differences in the age-specific incidence rates have been attributed to physiological changes associated with the reproductive years which may modify risk status by altering bile acid secretion.[8]

It is harder to explain the proximal shift with age of colon cancer in females but not males. This is not to be confused with the apparently increasing proportion of proximal disease diagnosed in a number of countries in recent decades in Scandinavia and North America.[10, 11] Some have noted that this recently growing proportion of proximal disease appears to be more marked in the over 65 year age group[14] tending to support an aging gut hypothesis. However retrospective cohort and cross-sectional survey designs tend to be more prone to a number of biases. It is likely that proximal tumours have a longer sojourn time and become symptomatic at a later stage in their biological course.[12] Younger patients are therefore likely to have relatively fewer right sided lesions than their older fellows. Less than five percent of tumours in this histological series could not be sited due to lack of information on the operative request or pathology report. Their age distribution was no different to the anatomically sited cases. However a detection bias might still be contributing to the profile of laboratory cases as there were far more cases registered (over 65 yrs) than histologically diagnosed (1·75 cases registered per case histologically diagnosed). The shortfall in numbers (for those over 65 yrs) was least for males with rectal cancer (1·11 cases registered per case histologically diagnosed). Presumably rectal cancer requires less invasive investigation and is therefore more immediately detectable than more proximal disease.

The key finding in our study was the very high incidence of histologically confirmed colorectal cancer in Northern Ireland. How much credence can be placed on our estimates of incidence? Firstly, we have reported both histologically confirmed and registered cases of colorectal cancer using the age-standardised figure for age-groups 35–64 yrs as recommended by the World Health Organisation[2] to avoid the vagaries of diagnosis in the very old. Even so, the incidence of histologically confirmed tumours in Northern Ireland is as high as or higher than the registered rates elsewhere in the United Kingdom.

Secondly, the comparability of the incidence derived from the Northern Ireland Cancer Register with those of other United Kingdom registers can be judged against a number of indices of reliability which are used in the compilation of register statistics.[2] One such measure is the ratio of cancer deaths in a period to the number of registrations. From a recent statistical compilation of the Cancer Research Campaign[13] these ratios for colorectal cancer in the United Kingdom are: England 0·72, Wales 0·73, Scotland 0·65, and Northern Ireland 0·73. The proportion of cases which are registered by death certificate alone may reflect the extent of under-recording.[2] For gastrointestinal tumours in Northern Ireland this figure is 24%,[14] and for the regional registers elsewhere in the UK the variation is between 2% and 18% for colorectal cancer. Another index of reliability used by the World Health Organisation is the proportion of registrations which have been histologically confirmed. For the 35–64 yr age groups this varies from 66% to 99% of regional colorectal cancer registrations in the rest of the UK.[2] We have not attempted this form of validation and can only report that during our 3 year study period the ratio of histological diagnoses to registrations in this age-group was 521/603 or 0·86 to 1.

These observations suggest that the registered incidence of colorectal cancer in Northern Ireland gives a reasonable reflection of the excess of this disease above the rates in the rest of the United Kingdom. Although it has been generally recognised that mortality from colorectal cancer in Northern Ireland is high, our results indicate that this likely reflects a much higher underlying incidence.

Although the north and south of Ireland share a similar excess, the secular trends in the countries have not been identical.[3] Since it is conceivable that the risk factors for incidence may not exactly coincide with those for mortality,[15] further work is required to determine the important local determinants of this major public health problem.

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Audit in intensive care. The APACHE II classification of severity of disease

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SUMMARY

The Acute Physiology and Chronic Health Evaluation System (APACHE II) was used in 451 patients admitted to the intensive care unit, Belfast City Hospital, in 1988 and 1989. Mortality in the patients studied was 15·5% which is slightly less than that predicted for patients with equal severity of disease (18·8%). Within the limitations discussed the APACHE II system has valuable potential both in terms of predictive power and as a means of audit.

INTRODUCTION

The need for a reliable means of predicting outcome in intensive care units was pointed out in this unit 10 years ago.[1] The Acute Physiology and Chronic Health Evaluation (APACHE) system was introduced by Knaus and colleagues about that time but required the collection of large amounts of data, and was later simplified to APACHE II (pronounced Apache Two). This depends on 12 physiological variables derived from vital signs and standard laboratory blood tests to determine the acute severity of disease, and combines this with the patient age and the presence of severe chronic diseases to create a risk severity score that normally varies from 0 to 50. A high score at the time of admission to intensive care implies a higher acute risk of death. The undoubted popularity and widespread use of APACHE II are due to its relative simplicity in terms of data collection and the availability of computer software to simplify analysis of data. The aim of this study was to validate APACHE II for our patient population and to compare our overall mortality with that predicted by the computer programme.

METHODS

From 1st January 1988 to 31st December 1989 636 patients were admitted to the intensive care unit, of which 451 were included in this study. Those excluded were patients discharged in 24 hours or less (132), those who died within a few hours of admission (25), and those in whom data collection was inadequate (28). Biographic and administrative information was recorded, and the "worst over 24 hours" physiological data required for entry at the computer keyboard collected. The bulk of data collection and all keyboard entries were performed by the authors. Data collection in 1988 was restricted to the first day only (214 patients) and in 1989 daily or alternate day assessments were made where it was considered appropriate. (80 out of 237 patients). Day to day changes in scores

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are reputed to reflect the changing patterns of illness which affect patients in intensive care. The software was APACHE II Version 1.0 (1986) supplied by Knaus and colleagues, Intensive Care Research Unit, the George Washington Medical Center, Washington DC, and was run using an IBM compatible Hewlett Packard Vectra personal computer with a 20 Mbyte hard disc. Depending on the admission data, the programme divides patients into an active treatment group (which includes those who require artificial ventilation, vasoactive drug infusion, aggressive intravenous fluid or blood replacement, and many other interventions), or a monitor group. Monitor patients with a less than 10% predicted risk of active intervention are called low-risk monitor patients while those with a greater than 10% risk are labelled high-risk monitor patients.

RESULTS

Of the 451 patients studied, 158 (35%) were female; the age range was 16–91 years, mean 58·2 years. Details of the admission groups, first day APACHE II scores and number of deaths are shown in Table I. Seventy patients died, 66 in the unit, four after transfer to the ward; the interval between transfer and death was one day to three months. Four patients in the high-risk monitor group eventually required active therapy and two subsequently died. The mortality in the intensive care unit was 13·7% and the overall mortality 15·5%. This compares with a predicted hospital death rate of 18·8% using a computer derived multiple logistic regression analysis of mean APACHE II scores which requires a minimum number of 200 patients. Table II shows a general breakdown of admissions into broad diagnostic groups and some of these are considered in more detail.

TABLE I

Patients entered into the APACHE II study 1988–89. Mean first day APACHE II scores reflect group severity of illness

Admission group	*No*	*Mean first day APACHE II score (± SD)*	*Range*	*Deaths*
Active treatment	327	16·6 ± 6·6	2–41	68
High-risk monitor	36	14·0 ± 6·4	5–32	2
Low-risk monitor	88	8·4 ± 4·7	0–22	0
All patients	451	14·9 ± 4·7	0–41	70
Active treatment on admission — died	68	23·7 ± 6·6	9–41	68
Active treatment on admission — survived	259	14·7 ± 6·5	2–33	0

Vascular surgery. This comprised a major part of the work of the unit and of the 123 patients, 69 had surgery to repair abdominal aortic aneurysms. Thirty-two operations were elective and there were two deaths, one following graft infection (which led to a review of antibiotic policy in these patients), the other in a confused elderly man who removed his naso-gastric tube on return to the ward and

TABLE II

A breakdown of admissions into diagnostic groups with percent of total admissions in each group, mean APACHE II scores and number of deaths for each group

Admission group	*No (% of admissions)*	*Mean APACHE II score (± SD)*	*Deaths (% of group)*
Vascular surgery	123 (27)	13·3 ± 6·0	10 (8)
Abdominal and renal surgery	93 (21)	14·9 ± 5·8	15 (16)
Medical and self-overdose	88 (20)	18·3 ± 7·9	28 (32)
Trauma (multiple and head only)	49 (11)	14·7 ± 8·3	8 (16)
Thoracotomy (lung or oesophageal neoplasm)	37 (8)	8·7 ± 3·4	0
Renal failure (primary reason for admission)	17 (4)	18·4 ± 5·6	1 (6)
Sepsis (primary diagnosis)	9 (2)	24·7 ± 6·8	3 (33)
Postoperative respiratory failure	22 (5)	10·9 ± 3·3	2 (9)
Post cardiac arrest	6 (1·3)	22·2 ± 10·4	3 (50)
Others	7 (1·6)	9·3 ± 4·6	0

aspirated gastric contents. The figures for ruptured aortic aneurysm (Table III) do not truly reflect the disastrous consequences of this disease. Here, mean APACHE II scores were substantially higher than in elective cases; the patients spent much longer in intensive care, and while only four deaths are listed, six others occurred in the first few hours after admission and are therefore not included in the analysis, while six more died on the operating table. Aorto-femoral grafting was performed in 30 patients (3 deaths) and carotid endarterectomy in 16 (two suffered major stroke).

TABLE III

Numbers of patients undergoing certain procedures or satisfying selected diagnostic categories with mean APACHE II scores and the number of deaths

Diagnosis	*No*	*Mean APACHE II score (± SD)*	*Deaths*
Elective abdominal aortic aneurysm	37	12·7 ± 4·6	2
Emergency ruptured aortic aneurysm	32	17·4 ± 6·8	4
Respiratory infection	17	18·5 ± 7·3	6
Chronic obstructive airways disease	9	21·8 ± 8·0	1
Head trauma only	12	18·1 ± 11·1	6
Self-overdose	10	19·7 ± 8·6	3
Intracranial haemorrhage	7	21·4 ± 4·0	7

Abdominal and renal surgery. The 16% mortality in this group is an indication of the seriousness of intra-abdominal disease in elderly patients and the continuing high mortality in those with peritonitis and sepsis from perforation or rupture of bowel is to be noted. Sixty-six percent of these operations were emergency.

Medical and self-overdose. This is a heterogeneous group of patients suffering from such diverse complaints as respiratory infection, chronic obstructive airways disease, asthma, leukaemia, neurological problems including intracranial haemorrhage, and self overdose. APACHE II scores are high and the mortality in some of the sub-groups is also high (Table III). Some of these patients suffered from progressive pulmonary disease which proved to be irreversible; others developed infection due to immunosuppression. Remarkably, survival from acute infection in patients with chronic obstructive airways disease is good; this is presumably due to careful assessment of the likely outcome before agreeing to admission. Twenty percent of all admissions were patients with medical problems, and their illnesses which were often protracted occasionally put a considerable strain on the smooth running of the unit. Surgeons were unable to do elective operations and this led to frustration and criticism. One partially tetraplegic patient with severe respiratory problems occupied a bed for 174 days before being discharged home to continue a regimen of oxygen and suction.

Trauma. Of 37 patients admitted with multiple fractures, some associated with severe chest trauma and multiple rib fractures, only two elderly patients died. In contrast, when trauma was confined to the head, six out of twelve died.

Renal failure. This hospital is the Regional Centre for nephrology. Seventeen patients were admitted with a primary diagnosis of renal failure, many being referrals from other hospitals. A total of thirty-one patients required haemodialysis or ultrafiltration, while a further seven patients were managed with peritoneal dialysis only.

General management. Almost all patients received additional oxygen. Mechanical ventilation was employed in 240 patients (53%) for periods varying from hours to many days. An additional 30 patients required airway support using continuous positive airway pressure by mask, or in a few cases endotracheal intubation or formal tracheostomy, so that a total of 270 patients (60%) received some form of airway management. This figure would be even higher but for the liberal use of thoracic epidural analgesia to provide post-operative pain relief in many patients who had had either major abdominal surgery, thoracotomy, or were suffering from multiple fractured ribs. In the three final categories in Table III a number of patients were declared brain dead; six became kidney donors and one a multiple organ donor.

DISCUSSION

The aims of this study were to assess our performance and to validate the APACHE II system. We had initial problems with the software which were resolved (Appendix). The programme required us to convert SI units to traditional units for blood gas tensions and serum creatinine. The APACHE II system was originally developed for quality assurance on the day of admission using "worst over 24 hours" values. It was based on a study of treatment and outcome of 5030 patients

in 13 intensive care units in the United States, and from that study the predicted death rate according to the mean overall APACHE II score was calculated for a wide range of mean scores.[2] Our predicted death rate was only 15·5%. Although a mathematical prediction cannot be entirely precise, Knaus and colleagues did comment on the wide difference in mortality rates between hospitals. In one well staffed unit the senior nurse on duty had the power to cancel elective major surgery if adequate unit nursing was not available, and the mortality there was 41% less than predicted. We have adopted this principle. In another hospital where there was chronic under staffing and poor communication between physicians and nurses, mortality was 58% greater than predicted. The number of non-operation (medical) cases may also have an effect on prediction since the same authors noted that when medical and surgical patients with similar scores were compared, mortality in the former was twice as high. The numbers in our groups were too small and the mean scores too varied for us to confirm this assertion. Overall, using these criteria, we judge our performance as slightly better than the USA average.[2] First-day APACHE II scores cannot be used to make individual outcome predictions although they may be of value in making group analyses according to the particular disease process being studied. Trend analysis on a day-to-day basis is much more useful, and it was evident in our 80 patients subjected to trend analysis that a rapid rise in the daily score to levels greater than 30 was usually a poor prognostic sign, while a quick fall in score was most likely to be reflected in a satisfactory outcome.

The Riyadh group[3,4] have claimed relative success in using computerised trend analysis of daily scores corrected for organ system failure to identify with some certainty those patients least likely to survive. Unfortunately, such systems are fallible, and the Figure shows a plot of daily scores in two successive patients (uncorrected for organ failure) where those criteria would have predicted one

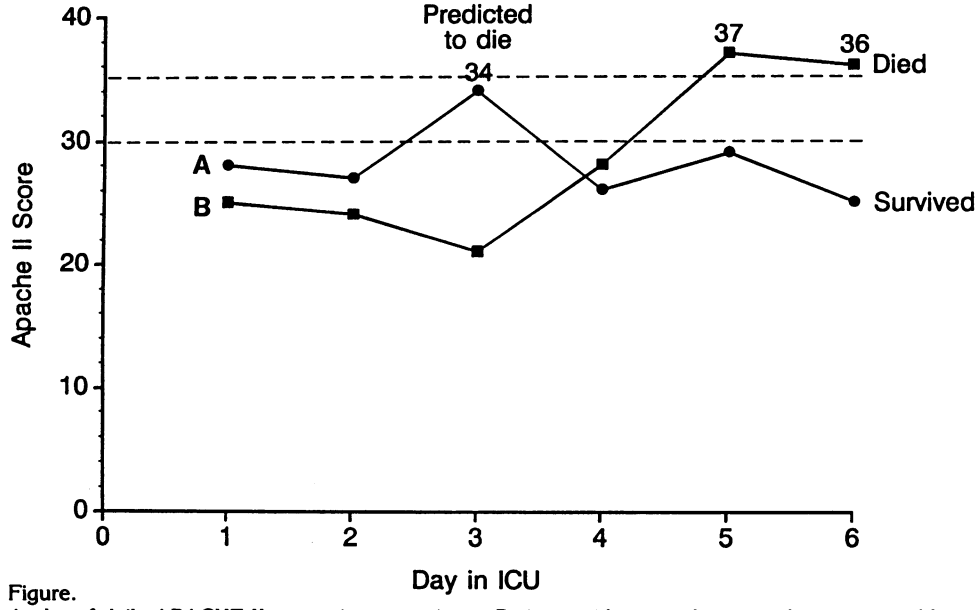

Figure.
A plot of daily APACHE II scores in two patients. Patients with scores between the interrupted lines (30–35) are likely to die while patients with scores greater than 35 are almost certain to die. Patient A deteriorated initially but ultimately survived. Patient B appeared to improve but subsequently died.

patient to die, the other to survive; in fact the converse was true. No patient admitted with a first day score greater than 33 survived, but the dangers of computer-based predictions which influence therapy are well recognised, since they may lead to therapeutic nihilism and therefore be self-fulfilling. The alternative argument is that needless and futile prolongation of life in patients with end stage disease is just as undesirable. One group of workers have indicated that clinical observation is as accurate as machine-based figures in deciding whether the patient will survive his illness.[5]

Interpretation of the Glasgow coma score is of critical importance in obtaining accurate figures for APACHE II. The Glasgow coma score can vary from 3 (worst possible) to 15 (normal), and the difference from normal is added to the APACHE II score. The lowest score given to a fatal outcome (9) was in a patient admitted with intracranial bleeding who died within 48 hours; it seems evident that an over-optimistic Glasgow coma score was given to this patient which resulted in a misleadingly low APACHE II score. We have noted that young patients who have suffered catastrophic intracranial bleeding score relatively low on the APACHE II system simply because the Glasgow coma score is the only abnormal parameter when cardiovascular, respiratory and renal systems are being supported as effectively as possible while brain-stem function is being tested with a view to seeking permission for organ retrieval (Table III). Heavily sedated patients or those curarised with muscle relaxants may also be given an inappropriately low Glasgow coma score when underlying brain function is near normal. It is our practice in these cases, where neurological assessment is difficult, to award a normal or near normal Glasgow coma score to reduce the chance of producing a misleadingly high APACHE II score.[6] The recent report on intensive care units by the King's Fund panel received general approval.[7] They correctly identified the major problem areas of costs and benefits, the need for individual and collective responsibility, and the requirement for proper audit procedures and prospective research. They have had a major input into the Intensive Care Society's UK APACHE II study which was completed in 1990 with 11,000 patient data sets. The American intensive care physicians have also expanded their data base (Knaus and colleagues, personal communication) which should give ample opportunity for comparison of the American and United Kingdom intensive care populations. Published reports[6] indicate a distinct difference between the United States and Saudi Arabia in terms of reasons for admission to an intensive care unit and the presence and type of severe chronic disease which may be observed. Whatever the outcome of these studies, we can expect to see a fine tuning of the APACHE system in the future. Modified systems are available for developing countries using clinical data only, where biochemical analyses are either unreliable or unavailable.[8]

Finally, consideration should be given to the long-term future of those patients who survive their experience in an intensive care unit. The expectation of life to one year seems to lie between 60–75%,[9, 10] with further survival after one year being almost the same as for the general population, and the quality of life determined by the health status prior to the acute illness. Other workers have reported a much higher mortality associated with intensive medical care in a predominantly geriatric patient population, but again emphasise that quality of life before admission is an important predictor of survival from critical illness and of the subsequent life-style.[11]

We have looked at one method of monitoring the quality of care, but have not touched on the question of "production costs", or the financial implications of intensive care. In a recent editorial [12] Knaus made the point that few if any doctors received training in outcome prediction, cost efficiency and quality of life assessment, and yet these issues may be among the most important considerations for meeting the challenges of tomorrow.

APPENDIX

Initial problems with the software were due to computer illiteracy on the part of the operators. In addition, the 'Backup' function on the 5¼" disc was unreliable so that information was not stored on hard disc and copies could not be made. These problems were resolved by writing two short programmes in MS–DOS which allow 'backup' and 'restore' functions to operate. These difficulties may well be historical, since an updated version is now available, and APACHE III will be available in late 1990.

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Psychiatric rehabilitation — does it work? A three year retrospective survey

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SUMMARY

To evaluate the effectiveness of a rehabilitation unit in returning psychiatric patients to community settings, a survey was undertaken of all patients referred to the rehabilitation unit at Holywell Hospital (Bush House) from 1985 to 1987. The location of 96 patients at the end of 1987 was recorded; 38 were living in the community and 38 had either failed to make this transition or had attempted a community placement but were back in hospital. Comparison of these two sub-groups showed those living successfully in the community to be older and to have had fewer hospital admissions, although the total number of years in hospital was similar. A number of other clinical findings have been helpful in planning future services and in modifying rehabilitation programmes at this unit. The deficiencies of this quantitative evaluation process were identified and there is a need for further qualitative investigations.

INTRODUCTION

One of the Regional Planning Guidelines for services for mentally ill people is that "effective rehabilitation programmes should be established in long-stay units".[1] The purpose of this is to accelerate in a planned and co-ordinated fashion the reduction in the resident population of large psychiatric hospitals. This decline in numbers has taken place over many years, most notably after 1960, since when the number of patients in psychiatric hospitals in Northern Ireland has fallen from 5500 to under 4000 in 1983. A continuation of this trend in the planning cycle 1987–1992 would result in a further reduction of 10%, but the four Health and Social Services Boards in the Province are expected, through the development of new community based services and the active work of rehabilitation units, to achieve a 20% reduction in hospital residents.

One of the problems of such extrapolation is that it does not take into account the disability level of those patients who remain in long-term care. It has been shown[2] that trends in discharge patterns are exponential rather than linear and that there is a core figure of those needing long-term care which is constant. This point is highlighted by a comparison of mental hospital populations over the years which reveals that patients who now become long-stay are less likely to be easily rehabilitated, and place greater demands on nursing time.[3] At present the

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rehabilitation units find themselves caught up between the clinical reality of a more dependent and handicapped population, and the government policy of accelerated reduction in psychiatric hospital population. In these circumstances it is essential that rehabilitation services are evaluated critically to ensure the most effective use of resources. In human terms this means that relevant programmes of rehabilitation practice are directed to appropriate individuals and that adequate systems of support and aftercare in the community are developed.

A variety of evaluation techniques have been used in other studies which can usually be classified in terms of four different perspectives — structure or input, process, output and outcome.[4] Each approach has its respective strengths and limitations. Outcome measures at first glance appear the most objective, where the effectiveness of a service is measured by the degree to which the patient has improved as the result of intervention. Using this criterion, a simple evaluation of a rehabilitation unit is the degree to which patients are enabled to live in the community. In this study the progress of each patient referred to the rehabilitation unit at Holywell Hospital (Bush House) over the three year period 1985–1987 was examined. A profile of patients was drawn up and comparisons made between those who are presently in the community and those who remain in hospital after taking part in the rehabilitation programme.

METHODS

The case-notes of 96 patients referred to the rehabilitation unit over the three year period 1985–1987 were examined using a standardized check list. Demographic details together with psychiatric diagnosis and history were recorded. The location and type of accommodation being used by each patient at the end of 1987 was noted. The sample was split into a group who had been discharged from hospital and were living in the community, and another group who had not been successfully resettled. The latter group was made up of patients who were readmitted after discharge and remained in hospital, and those who returned to their former long-stay wards from either day-patient or in-patient status at the rehabilitation unit. These groups were compared with respect to age, sex, length and number of admissions, ward of origin and diagnosis. A third group of patients who were resident in Bush House at the time of the survey were not included in these comparisons.

RESULTS

Background information on the 96 patients referred to the rehabilitation unit from 1985–1987 is shown in Table I. There were roughly equal numbers of males and females, the majority (70%) were single, and over 60% were diagnosed as suffering from schizophrenia. At the end of 1987 these 96 individuals were located as indicated in Table II. Of these 44 patients who had been discharged, ten had gone to a social services hostel, ten to private residential accommodation, eight to live with their own family, seven each to social services residential accommodation or a new single home, and one each to a group home or a Fold Housing Association.

By the end of 1987 thirty-eight patients were living in the community after planned discharge from hospital. An identical number had been in the rehabilitation unit but had either been returned to their original wards (32) or had returned to hospital after discharge and remained there (6). The majority of the remaining patients were still either day or in-patients at the unit (18).

TABLE I

Background data on patients referred to the rehabilitation unit 1985–1987 (Mean ± SD)

	Mean	Range
Age (yr)	48·7 ± 17·0	23 — 87
Length of present admission (yr)	8·3 ± 10·5	1 — 42
Length of all admissions (yr)	10·5 ± 11·7	1 — 49
Number of previous admissions	3·9 ± 4·0	0 — 18
Age at onset of illness (yr)	31·6 ± 13·3	15 — 72

TABLE II

Location of 96 patients at the end of 1987

Outcome	*Number*
Discharged without subsequent readmission	33
Discharged and subsequently readmitted	11*
Returned to long-stay wards from in-patient care at rehabilitation unit	10
Returned to wards from day-patient attendance at rehabilitation unit	22
In-patients at rehabilitation unit	18
Left the rehabilitation unit against medical advice	2
Died whilst in unit	2
Total	96

*Five of these patients were discharged again and remained in community. Six remain in hospital including two in the rehabilitation unit.

To investigate possible distinguishing characteristics between those who were in the community and those who had not successfully made this transition, a number of variables were compared between the first and second sub-groups (Table III). Resettled patients were significantly older and had had fewer psychiatric admissions than those non-resettled. Although the resettled patients had had fewer admissions, there was no difference between the two groups for the total amount of time spent in hospital.

TABLE III

Comparisons between resettled and non-resettled groups

Variable	*Resettled (Mean ± SD)*	*Non-resettled (Mean ± SD)*	*P*
Age	56·3 ± 16·3	42·1 ± 16·4	0·009
Length of present admission	10·3 ± 10·9	6·3 ± 7·3	0·11
Length of all admissions	12·7 ± 13·2	8·4 ± 7·5	0·37
Number of previous admissions	2·8 ± 3·1	5·3 ± 4·5	0·008
Age at onset of illness	37·2 ± 14·3	28·9 ± 11·8	0·014

The Mann-Whitney U test was used to assess significance.

There was no sex difference between the groups ($chi^2 = 1 \cdot 6$, NS) although there is a relatively large group of 25 males who were not resettled. Those with schizophrenia were as successful as non-schizophrenic patients in community placements ($chi^2 = 0 \cdot 22$, NS). No-one who had come to the rehabilitation unit directly from the intensive care wards had been successfully resettled, but no other ward of origin was identified as affecting the outcome.

DISCUSSION

During the three year period under investigation almost half the patients referred to Bush House were discharged into the community, and 38 remained there. This is an important reduction in hospital numbers given both the high proportion of seriously ill people referred to the unit and the duration of their psychiatric conditions. However, a further 32 patients referred to the unit had to be transferred back to their ward of origin. As all of these patients were originally assessed by staff at the rehabilitation unit it can be assumed that the referrals were appropriate. Therefore it is important to discover why such large numbers of patients failed to progress to any form of community accommodation after their involvement with the rehabilitation service.

The results indicate that older patients have been relocated more successfully than younger ones although they have not spent significantly longer in hospital. Older patients tend to move to residential accommodation and it might be speculated that this relative "success" is the result of transferring from one type of institutional care to another which has many similarities. Failure in the other group does not necessarily relate to problems in adjusting to a particular type of community facility, as the majority of this group (about 88%) returned to their ward of origin without having been discharged into the community. They usually returned to their original wards because of a deterioration in mental state, lack of interest in the programmes, no motivation to leave hospital or personality difficulties, which produced unrealistic expectations and/or interpersonal friction. Younger patients usually had a desire to live in the community, and it could be that rehabilitation services as currently delivered are not meeting the needs of younger people with chronic illnesses, resulting in their repeated admissions to hospital. If effective community based alternatives are to be found for these patients, then ward policies and practices may need to change and those who plan community services must ensure that sufficient support services are available to deal with the difficulties thrown up by the younger patients.

Although sex did not significantly affect the outcome between the groups there was a tendency for more males than females to fail to make the transition to community living. There was a great deal of emphasis on domestic chores, which many of the men resented and indeed may not have been relevant to them in their eventual location. It may be more realistic to provide community based accommodation for men, including appropriate domestic services, and to relocate patients directly with care staff who are known to them. The data relating to ward of origin could not be analysed because of inadequate cell numbers, but the fact that no-one who had come from the intensive care wards had been successfully relocated may have some clinical significance. In making the move from intensive care to rehabilitation the patient moves from a highly structured and intensively supervised régime to a fairly relaxed and more domestic setting, which must cause difficulty in adjusting.

Some comments are needed on the type of evaluation employed. The single criterion used was location at the end of 1987, which is based on the questionable

assumption that living in the community is synonymous with a successful rehabilitation outcome. Clearly the primary aim of psychiatric rehabilitation is not merely resettlement, and no statement about positive outcome can be justified without employing subjective or objective quality measures. Likewise, it is misleading to assign individuals to "successful" and "non-successful" groups merely on the basis of where they are living on a particular day. Such a simplistic view fails to reflect the nature of psychiatric conditions as a chronic illness where relapse and recovery, and occasional acute admissions, are to be expected. Just as such an admission does not necessarily represent failure on the part of a rehabilitation programme, neither does someone who is highly distressed by acute symptoms, but living in the community, give cause for congratulation.

It is clear that many factors interact to determine whether an individual with a psychiatric illness can be maintained in the community, is frequently readmitted to hospital or tends to remain in a ward setting for long periods. It also seems reasonable to assume that the factors which combine to enable an individual to cope in their own flat are different from those which might maintain them in residential accommodation. In order to make authoritative statements about the effectiveness of a rehabilitation service a very broad sweep of variables requires to be examined, increasing the chances of producing spurious findings. Such drawbacks spell out the need for caution when interpreting these results, but even such a rudimentary approach can point towards strengths and weaknesses in rehabilitation practice and may prove clinically useful.

The purpose of any evaluation exercise is critically to examine an intervention or service. Where deficiencies are found some working hypotheses can be generated, appropriate clinical changes made and further evaluation carried out. In addition, the results of such an exercise provide valuable information for planning purposes. Despite the reservations expressed at the outset of this discussion a number of significant findings, trends and clinically interesting issues were uncovered. Younger people and those with more admissions were less likely to be successfully placed in community settings. As stated previously most young patients wished to leave hospital and many have the practical skills to look after themselves in a flat. However, for many, previous admissions had been precipitated by lack of emotional support. Therefore, in planning services for these individuals both their needs and skills have been taken into account, and self-contained flatlets with built-in support from both the voluntary sector and the Health and Social Services Board are being provided. The relatively large numbers of males identified as not progressing to community living have very different needs from those provided by such a residential project. Their lack of motivation to engage in domestic chores has meant that two small community based facilities have been planned which will provide basic "hotel" services, although staff will still encourage and promote as much self-care as possible. These facilities will be centrally located in towns so that more extensive use can be made of community resources. In addition to these large scale projects smaller changes have been brought about within the hospital as a result of this review. Programmes have become more personalised and compatible with the anticipated lifestyle which patients will lead in the community. A new referral procedure between the intensive care wards and Bush House has been adopted which will hopefully overcome the transition difficulties experienced by patients from these wards.

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Medical treatment of impotence with papaverine and phentolamine intracavernosal injection

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SUMMARY

Intracavernosal injection of papaverine and phentolamine was used in 20 patients with impotence. After six months 14 patients were still using the self-injection treatment. Two patients failed to respond and two patients developed priapism which responded to venesection and metaraminol injection.

INTRODUCTION

Intracavernosal injection of the penis is the most effective medical treatment for impotence. While some of the original studies used phenoxybenzamine,[1] the most common treatment at present is with papaverine or phentolamine, either alone or in combination.[2-7] Papaverine (a smooth muscle relaxant) or phentolamine (an alpha-blocker) can increase penile tumescence and produce an erection after injection into the corpus cavernosum; unilateral injection produces bilateral engorgement due to cross circulation. When these drugs are injected arterial inflow increases and venous outflow decreases, probably due to relaxation of the corporeal smooth muscle with occlusion of the venules.

Previous studies have advocated the use of this treatment in impotence both of an organic and a psychological nature. This paper presents the results of the first 20 patients treated at the Department of Genitourinary Medicine, and it aims to assess the effect of these drugs, complications, acceptability and practical problems associated with this therapy.

PATIENTS AND METHODS

Patients presenting to this clinic with impotence, either total or partial, are encouraged to attend with their regular partner and are seen by appointment. After a sexual and medical history and examination the treatment options are explained. These include referral for psychotherapy, intra-cavernosal injection, mechanical aids to erection and surgery. All patients have routine tests for haemoglobin concentration, plasma glucose, testosterone, prolactin, liver function and hepatitis antigen.

Patients electing for intracavernosal injection have the possible complications explained and give signed consent for therapy. In addition they are given a list of potential complications and written instructions for the self-injection technique.

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An initial test dose of papaverine 30 mg and phentolamine 1 mg is given in the lateral aspect of the corpus cavernosum using a 2 ml syringe with a fine bore needle (26 gauge, 0·45 × 13 mm dental needle) at a site 1–2 cm proximal to the coronal sulcus of the penis with constriction of the base of the penis either manually or using a rubber band as a tourniquet. The drugs are combined in a single ampoule by the pharmaceutical department of the Royal Victoria Hospital and have an expiry date of approximately six months (note: phentolamine normally is available in vials containing 10 mg in 1 ml and the most convenient method of measurement is to withdraw 0·1 ml of phentolamine using an insulin syringe and transfer to a 2 ml syringe which is then used to draw the papaverine 30 mg in 2 ml). Response with erection is normally in 20–30 minutes and during this period patients have regular pulse rate and blood pressure measurements recorded at 10 minute intervals. Patients who fail to respond are given higher doses at later consultations, up to 90 mg papaverine and 3 mg phentolamine. Patients with a prolonged response, eg three to four hours duration, will be given a reduced dose at subsequent injections. The lowest effective dose is always used. After erection patients are allowed home. They are always given the home phone number of the supervising doctor, warned about the potential risk of priapism and instructed to present themselves early the following morning should this complication develop. Once the effective dose is attained patients inject themselves under medical supervision and, if this is successful, they are given a supply of needles, syringes and prepared ampoules containing phentolamine and papaverine for three occasions and are reviewed two weeks later. If there are no complications they are reviewed at two to three monthly intervals for a check-up and renewal of drug supplies.

RESULTS

The average age of the first 20 patients treated was 47·6 years (range 32–72). Seven had psychogenic or anxiety symptoms, 10 physical or mixed psychogenic and physical symptoms, and in three the impotence was judged to be of idiopathic origin. Fourteen patients were still using injection therapy at the end of six months. A total of six patients discontinued treatment, two due to priapism, one due to bruising, two due to incomplete injection response and one who gave no reason. The duration of erection in the patients who continued treatment was between 30 minutes and three hours with a median of two hours. The dosages used were papaverine 30 mg and phentolamine 1 mg in 18 patients, one patient used papaverine 15 mg and phentolamine 0·5 mg. In one patient treatment was unsuccessful at a dose level of papaverine 75 mg and phentolamine 2·5 mg.

The treatment of priapism is as follows: a 19 gauge cannula is inserted into the corpus cavernosum of the penis and blood allowed to flow into a dish for 10–15 minutes. One mg metaraminol is drawn up in an insulin syringe and diluted to 1 ml with normal saline. A further 10 ml of normal saline is drawn into a separate syringe. When the penis is compressible the metaraminol is injected and followed with 10 ml normal saline, with simultaneous penile massage. Up to 60–80 ml of blood is aspirated, and the penis should at this stage be flaccid. In one patient this procedure was repeated twice before relief.

DISCUSSION

Injection with phentolamine and papaverine produced an erection sufficient for intercourse in 18 of the 20 subjects. In two there was only a partial response,

reported by others.[3-7] Although priapism developed in two it was easily relieved with metaraminol.[8,9] It is essential that the doctor prescribing therapy makes himself available directly to the patient especially following the first and second doses as this is when priapism is most likely to occur. One patient felt a small fibrous nodule in the penis which resolved when treatment was stopped. Similar nodules have been described by Abozeid[10] after 100 intracavernosal injections in each of six Macaca monkeys and Hu[11] has confirmed in humans that this may be related to injection into the tunica albuginea. Patients are instructed to insert the needle until they feel a characteristic "give" as the needle penetrates the tunica of the corpus cavernosum, but this did not prevent the complication in one patient. Only 14 of the original 20 subjects were still on therapy at the end of six months, but this compares favourably with the 60–80% of patients who continue therapy reported from other centres.[2-7] These patients (most of whom are not diabetic and not used to injections) found the technique of self-injection easy to master, and no patient discontinued therapy because of injection discomfort.

This new method of therapy seems suitable for patients with an organic cause of impotence, but in elderly patients there is a less certain result and this may be due to related vascular disease. The method may be helpful in some cases of psychogenic impotence. However, in these cases it does not supplant the role of psychosexual therapy but may be used as an adjunct in unresponsive cases. In those considering penile implants intracavernosal injections clearly should be discussed before surgery is undertaken.

I am grateful to Dr Eithne O'Gorman and her staff for instruction in psychosexual therapy at Windsor House, Belfast City Hospital, and for her encouragement of my efforts. I am also grateful to the pharmacy staff of the Royal Victoria Hospital for preparing the papaverine and phentolamine injections. I thank Mrs Marie Loughran for typing the manuscript.

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Validation of two methods of long-term epidemiological follow-up

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SUMMARY

Two methods of long-term epidemiological follow-up were compared by using each to study the survival of 1622 myocardial infarction patients registered by the Belfast MONICA Project. Length of follow-up ranged between 3 and 5 years during which time 277 deaths were recorded.

A computer-based method for linking MONICA Project registration records with the Registrar General's death certification data identified 273 of the 277 deaths. Follow-up supplied by the Northern Ireland Central Services Agency through the flagging of patients in their master patient index identified 271 deaths; four of the six deaths which were missed occurred before computerisation of the index was complete. The study illustrates the value of computer-based linkage with death certification data and of flagging in the Central Services Agency master patient index.

INTRODUCTION

Obtaining long-term epidemiological follow-up on large numbers of patients can present substantial logistical problems. Nevertheless, Northern Ireland provides a good location for follow-up studies because of its position and its relatively low levels of migration in the older age-groups.

This paper compares the results obtained using two different methods to follow up patients registered by the Belfast MONICA Project. The first relied on a computerised search of death registration information collected by the Registrar General's Office, using personal identifying information recorded at death registration. The second was provided by the Central Services Agency through the flagging of patient records in the master patient index. Although the convenience of this approach is appealing, it is important that the method should be independently validated for completeness.

The WHO MONICA Project: Multinational MONItoring of trends and determinants in CArdiovascular diseases.

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MATERIALS AND METHODS

The Belfast MONICA Project

Since January 1983 the Belfast MONICA Project has sought to register every myocardial infarction occurring among individuals aged between 25 and 64 years and resident in the Belfast, Castlereagh, North Down or Ards district council areas. During the period 1983–85 a total of 2,727 events which fulfilled the Project criteria for definite or possible infarction[1] were registered. These events occurred among 2,512 residents, 1,622 of whom survived at least 28 days after onset of the infarction. Long-term follow-up of these 1,622 individuals was obtained to 30th June 1988.

Record Linkage

At the start of the Belfast MONICA Project it was appreciated that the follow-up of such large numbers of patients would be a major undertaking. It was therefore decided to automate follow-up by routinely linking computerised MONICA records with the Registrar General's computerised death registration records. Deaths registered in all age-groups throughout Northern Ireland during the years 1983–88 were considered. Patients who left the Project area to live in another part of Northern Ireland were therefore not lost to follow-up.

As with all large-scale data collection exercises, errors in the information recorded may result for a variety of reasons. As well as errors in data coding and preparation, misreporting errors can occur. Much of the information recorded at death registration is supplied by an informant who may not necessarily have been related to the deceased. Special steps were taken to ensure that errors in recorded data did not result in a failure of the search procedure to link MONICA records with matching death registrations. The approach built on the experience of the Northern Ireland Record Linkage Research Unit,[2] but used a rather different methodology.[3]

Ideally the personal identifying information available for such a linkage exercise should be permanent and have high discriminating power. Although the National Health Service number comes closest to this ideal, it is seldom known and is not recorded either by the MONICA Project or at death registration. Surname, marital status and occupation all lack permanence. Although forenames are less likely to change, they are often reported inaccurately with the use of abbreviated forms or inversion of order common. Surnames are sometimes misspelt with similar versions of the same surname confused. To minimise the effects of this problem the "Russell Soundex code" was used. Similar versions of the same surname all have the same Soundex code (eg Smith, Smyth, Smythe), so use of the code in place of the surname in the search procedure can deal with the majority of discrepancies in the spelling of surnames.[4]

Whether or not the search was successful was decided on the basis of "weights of comparison" derived from the Soundex code, the forenames and initials, the day, month and year of birth and the district council area of residence. The computer used these weights to mimic the intuition of the human mind. For example, one would be more likely to match two records which agree on a rare surname such as "Gravenitz" than two records which agree on a common surname such as "Smith". An illustration of the calculation of weights is shown in Fig 1. Although the first pair of records receive positive weights for agreement on surname and near-agreement on the forename and year of birth, the disagreement on the day

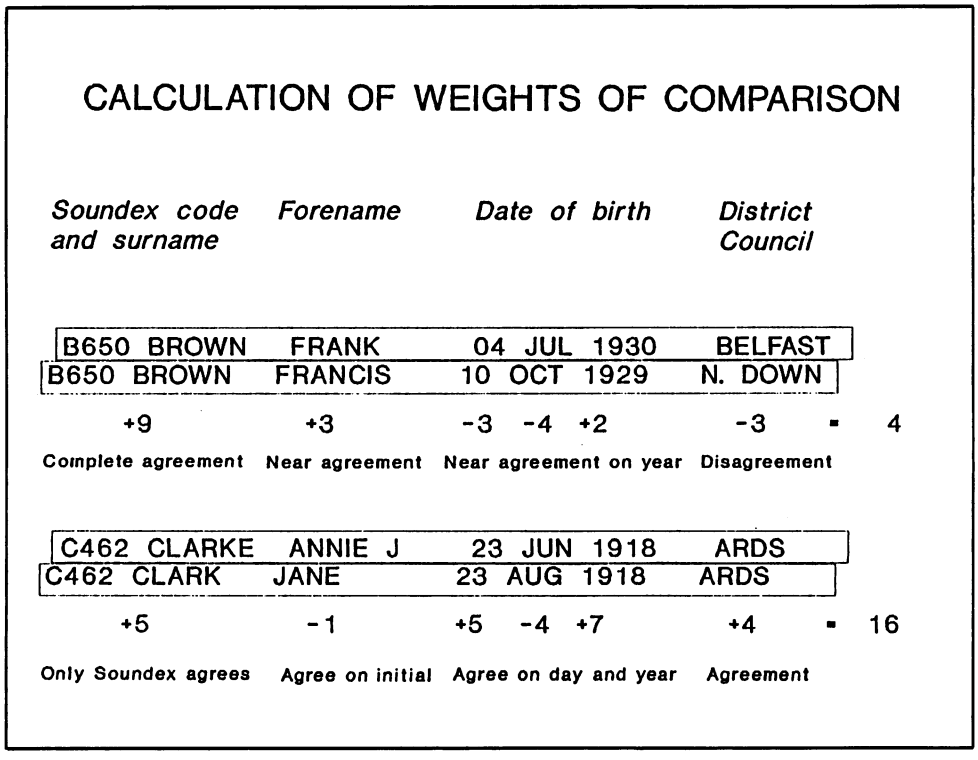

Fig 1. Use of identifying information in patient records to derive a weight of comparison for assessing the likelihood that two records relate to the same individual.

and month of birth and the district council are sufficient to give a low total weight of 4. In contrast the second pair of records give a high total weight of 16 despite discrepancies in forename, surname and month of birth. The derivation of the weights has been fully described elsewhere.[5]

Weights for a sample of 116 matching MONICA and death registration records and for a sample of 5,345 non-matching records are shown in Fig 2. The complete separation of the two distributions by the selected cut-off of 10 units illustrates the ability of the search procedure to distinguish between matching and non-matching records. The patient's sex, marital status, occupation and address were used to verify manually all matches generated by the search procedure.

Central Services Agency flagging

In 1983 the Northern Ireland Central Services Agency began to computerise its master patient index, so permitting the flagging of records of individual patients. The index is updated primarily by using data from death certification, but also by using information received from general practitioners and from relatives returning medical cards to the Agency. Patient transfers to health authorities in Great Britain are recorded. By 1986 the Agency was in a position to supply follow-up information on patients from mid 1983. A similar system has operated successfully in Great Britain for many years through the National Health Service Central Register.[6]

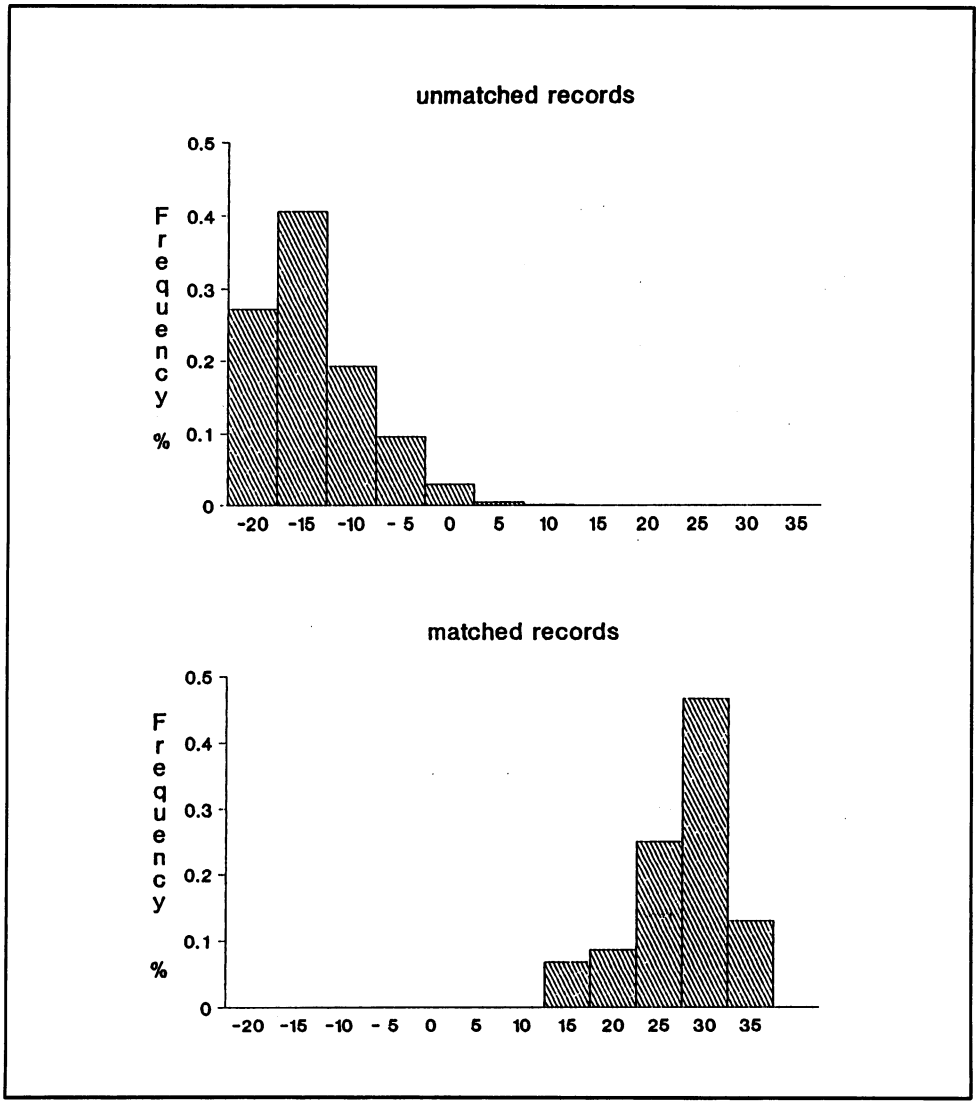

Fig 2. The distribution of weights of comparison in 116 pairs of records from the same individual (matched) and in 5,345 pairs of records from different individuals (unmatched).

RESULTS

A comparison of follow-up results obtained by the two methods is shown in the Table. A total of 22 patients could not be flagged because no entry with matching details could be found in the master patient index. Ten patients were notified as having transferred to health authorities in Great Britain, and were considered lost to follow-up.

Four deaths were missed by record linkage. One occurred in England and was registered there. Another produced a weight of only 8 because of a difference in forename (Eithne instead of Ann), a missing day and month of birth on the

TABLE

Comparison of Central Services Agency and Record Linkage follow-up for 1622 myocardial infarction survivors

Central Services Agency follow-up	*Record Linkage follow-up*	
	Dead (matched)	*Alive (unmatched)*
Dead	267	4
Alive	2	1317
Lost to follow-up	0	10
Not flagged	4	18

MONICA record and a discrepancy in the year of birth (1928 instead of 1929). The remaining two deaths were confirmed by the patient's general practitioner, but death certifications could not be traced despite extensive searches. Two deaths identified by record linkage were overlooked by Central Services Agency flagging, and further checks suggested that these were probably clerical errors by the Agency. The four deaths occurring among individuals who could not be flagged occurred in early 1983 before computerisation of the Agency's index was completed.

As a further check on the completeness of follow-up, 277 general practitioners were mailed in July 1988 with a request for follow-up information on the 1622 patients. Follow-up information was obtained for 1,385 (85%) of the patients. Nine (4%) of the 223 deaths notified had apparently been missed by record linkage and flagging. Two auxiliary sources were used to check the status of these nine patients. The first was hospital records, and these confirmed that three of the patients had been reviewed after 30th June 1988. The second was the Northern Ireland 1988 electoral register, the qualifying date for which was 15th September 1988. Of the remaining six patients, four were still on the 1988 register and were therefore assumed to be alive at the end of follow-up. One of the final two patients had previously been reported by his general practitioner as being alive in July 1987, but the other could not be traced at all.

DISCUSSION

The flagging of patients in the Central Services Agency master patient index performed very satisfactorily missing only six deaths in the follow-up period, four of which occurred before computerisation of the index was complete. Any chance of clerical error has been reduced by the recent introduction of computer-generated follow-up reports. The attractions of flagging are its convenience and completeness, and its ability to identify some patients who emigrate. The flagging facility will prove extremely valuable to those involved in epidemiological research in the Province. Additionally, at a time of increasing interest in clinical audit, there will be an important role for this facility in the evaluation of the long-term outcome of medical care. The introduction of this service by the Central Services Agency is therefore both welcome and timely.

The computerised search procedure developed to link MONICA Project records with death certification data was also found to perform well, ascertaining all but

four of the 277 deaths identified in the follow-up period. This procedure does retain some advantage over flagging. It may be performed retrospectively, while the flagging has only a limited retrospective capability. Record linkage also directly identifies the Registrar General's serial number, thus simplifying ascertainment of the registered cause of death.

The broader issue of the difficulty of linking computerised records from different sources (general practice sessions, inpatient admissions, outpatient attendances, screening and immunisation clinics etc) has yet to be resolved. Only when a unique identification number is in widespread general use can the information technology revolution be expected to make its fullest contribution to research in the health service.

The authors acknowledge the kind co-operation of the staff of the Registrar General's Office, the Central Services Agency and the general practitioners of the study area without whose assistance this work would not have been possible.

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Radical hysterectomy for stage 1 cervical carcinoma in Northern Ireland. A five year review

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SUMMARY

Forty-four radical hysterectomy operations were performed between 1981 and 1986 for stage 1 cervical carcinoma. The five year survival rate, (actuarial) was 84%. All deaths were directly attributable to disease recurrence. Five year mortality was higher in those under age 45 years, and who had more than two pregnancies. Histology and node status did not show significant correlation with outcome. In comparison with results of radiotherapy for this condition in Northern Ireland, (five-year survival rate 67·6%), radical hysterectomy produced better survival for stage 1 cervical carcinoma.

INTRODUCTION

In 1898 Wertheim[1] performed the first radical hysterectomy in Vienna whilst Forsell[2] first treated carcinoma of the cervix with radium. Since then the debate as to which is the more appropriate treatment for cervical carcinoma has remained unresolved. Both methods have been refined, modified and employed either separately or in combination (Rampone[3]). Houston[4] has recently reviewed the results of radiotherapy for all stages of cervical carcinoma in Northern Ireland. In this paper the results of radical hysterectomy for the treatment of stage 1 cervical carcinoma are presented and the two therapeutic regimens compared.

MATERIALS AND METHODS

Forty-four radical hysterectomies were performed between June 1981 and June 1986 in the Belfast City Hospital on patients clinically assessed to have stage 1 cervical carcinoma. Neither pre- nor post-operative radiotherapy was employed. The operation consisted of removal of the uterus, cervix, a vaginal cuff of one to two centimetres, and dissection of the parametrial tissues and pelvic nodes. Removal of the ovaries was optional and depended largely on the age of the patient: those under 45 years generally having one or both ovaries conserved, those over 45 years generally having both ovaries removed. All specimens were examined histologically at the pathology department of this hospital.

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The patients were reviewed at a joint gynaecological/oncological outpatients' clinic. Any patients in whom recurrence was suspected were investigated appropriately and, if necessary, treated with radiation therapy.

RESULTS

There was no operative mortality. In acute operative morbidity there were two cases each of urinary tract infection and chest complications, five wound abscesses and one pelvic abscess. Three instances of ureteric damage and one of urinary retention were recorded. There were no cases of deep vein thrombosis. No patient suffered long-term sequelae from the acute infections. The three cases of ureteric damage were detected during operation, repaired immediately, and no long-term complications developed.

Chronic operative morbidity included two cases of leg oedema, one of urinary incontinence and one of coital dysfunction. No vaginal fistulae developed. Incontinence of urine, (urgency in type), and coital dysfunction occurred in women aged over 60 years and may not necessarily have been related to surgery.

TABLE

Percentage survival after radical hysterectomy, using various potential predicting variables

	Survived (n = 37)	*Dead (n = 7)*
Age at treatment:		
< 45 years.	21 (78%)	6 (22%)
⩾45 years.	16 (94%)	1 (6%)
Parity:		
⩽2	16 (94%)	1 (6%)
> 2	21 (78%)	6 (22%)
Clinical stage:		
1a	11 (100%)	0
1b	26 (78%)	7 (22%)
Node status:		
Positive	3 (75%)	1 (25%)
Negative	34 (85%)	6 (15%)
Histology:		
Squamous:		
Differentiation		
Well	1	0
Moderate	22 (82%)	5 (18%)
Poor	8 (80%)	2 (20%)
Adenocarcinoma:	5 (83%)	1 (27%)
Recurrence:		
Nil	36 (100%)	0
Pelvis	1 (17%)	5 (83%)
Abdomen	0	1
Distal	0	1

A variety of prognostic factors is assessed against outcome in the Table. The only clearly significant predictor is the presence or absence of recurrence at follow-up. Only one patient survived more than 24 months after detection of recurrence; the majority died within one year. Only two recurrences occurred over two years after primary surgery, and only one patient died more than three years after primary surgery. The mean recurrence interval was 12 months and the mean mortality interval from primary surgery 22 months. The actuarial five year survival is 84%.

DISCUSSION

Five year survival rates for stage 1 cervical carcinoma treated by radical hysterectomy have been reported by Benedet[5] (81·8% of 88 patients), Powell[6] (90·3% of 238 patients), and Artman[7] (84·0% of 153 patients). These results compare favourably with those reported for radiotherapy of stage 1 carcinomas by Bygdeman[8] (88·0% of 60 patients) and Hanks[9] (92% of patients from a multicentre review). Houston[4] reported a 67·6% five year survival of 71 patients with stage 1 disease treated at the Northern Ireland Radiotherapy Centre.

No cases of deep vein thrombosis occurred even though prophylactic anticoagulants were not employed routinely in this series, prevention being dependent on early mobilization. No case of vaginal fistula occurred in this series and preoperative radiotherapy was not used. Benedet[5] showed the incidence of vaginal fistula to be much higher in the group which received preoperative radiation therapy than in the group which did not. The five year survival rates were not statistically different.

In terms of prognostic factors higher age and lower parity seem to confer a slightly better prognosis which is the international experience. However nodal status and histological grading were of little value, as also observed by Rommel.[10] Crissman[11] has suggested that lymphatic and vascular space involvement in the primary tumour is actually of greater prognostic value than confirmation of nodal metastasis, but we did not record such involvement. The single most important prognostic factor is recurrence: even after palliative radiotherapy only one patient is alive more than two years later.

We wish to thank those consultants who referred patients to this regional unit for treatment, our oncological colleagues with whom the combined review clinic is run, and the hospital pathology services. Mrs M Simms, who typed the manuscript in each of its many forms, also deserves special thanks.

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authors recommend an aggressive approach including nutritional assessment, biopsy, contrast radiography and endoscopy, to confirm the diagnosis in chronic granulomatous conditions of the mouth and to detect coincidental gastrointestinal involvement in cases of Crohn's disease.[4, 14] This case, where clinical assessment was equivocal, confirms the traditional difficulties in distinguishing between the two conditions. The evidence of malnutrition encouraged us to recommend careful follow up and nutritional assessment. We feel it is reasonable to reserve contrast radiology, endoscopy and biopsy for cases where specific symptoms or nutritional assessment suggest that they are indicated. Careful clinical examination, particularly of the perianal area, is necessary in all patients with chronic orofacial swelling.

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The harmony of disability

Michael Swallow

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Based on the Jim Egar Memorial Lecture delivered to the Northern Ireland Council for Orthopaedic Development (NICOD) on 29 November 1989.

At first sight the word harmony does not seem to be one most people would associate with disability. Harmony has a very specific meaning in musical terms, the combination of notes to form sequences of chords which follow accepted patterns and rules. The New Oxford Companion to Music defines harmony as the element of agreement in music, as opposed to counterpoint, in which parts move against each other and thus represent the element of disagreement. In a more general sense the word harmony is used to mean the combination or adaptation of parts, elements or related things, so as to form a consistent and orderly whole. Synonymous words are peaceableness and concord (Concise Oxford English Dictionary). In this sense disability might be thought of as the antithesis of harmony — the incongruity and disagreements of human impairment being positively dysharmonious. It will be my purpose in this lecture to look at ways in which we can reduce or reverse this assumption and help to restore the harmony to disability. I will be looking at the limitations which society imposes upon disabled people and seeking specific strategies to reduce this handicap. Much of my own work has been concerned with music and the arts as instruments for this purpose, but there are other aspects of what we may broadly term the rehabilitative process which I would also like to explore.

The other word in my title — disability — also needs some comment. The World Health Organisation has carefully defined the words impairment, disability and handicap and these distinctions are widely used and agreed by those working in the field. My own clinical experience is mostly of physical impairment and the disability that results, but much of what I have to say applies equally well to those with mental illness and handicap, to the frail elderly and to those with the sensory impairments of blindness and deafness.

The concept of restoring harmony to people with disabilities is made more difficult because of the diverse nature of disability and the many different ways of looking at the problem. From a practical point of view physical disability falls into two broad groups. First congenital impairments such as cerebral palsy and spina bifida, most commonly with non-progressive disabilities which have been part of the perception of life from infancy and often do not limit life expectancy. Secondly acquired disability, also of two types — the acute disability of trauma

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and stroke and the progressive disability of chronic arthritis or chest disease, multiple sclerosis, motor neurone disease or muscular dystrophy in younger people. The onset of disability in these cases is unexpected and inexplicable and results in profound changes in perception of life which has to be recognised by all who are involved in the problem. These two groups pose somewhat different problems in terms of the timing and type of intervention, but both have similar difficulties in trying to maintain a sense of worth and self-esteem, which is a common theme running through much of what I have to say today. We must also remember that disability is a family problem and that our strategies for helping people with disabilities must include major considerations of individual choice and support for, and involvement of, the family and carers at all times.

I would define rehabilitation as a process by which one seeks to maximise an individual's potential in terms of physical, mental, emotional and social capacity. In this sense rehabilitation never ceases because one can always do something to help. The diagnosis and management of impairment is a medical responsibility, combined with the use of specific therapeutic techniques. Disability can usually be minimised by the provision of suitable aids and equipment and handicap demands manipulation of the environment to restore or achieve a place in society for the individual. These concepts can be applied equally well to a person with a progressive disabling condition as to someone recovering from a head injury, and in this sense rehabilitation is a continuing process.

There is no doubt that the achievements of many disabled people are much more limited than they might be, quite apart from obvious physical limitations. I suspect that lack of expectation and lack of opportunity are two of the most important. The potential of disabled people is often unrecognised. Relatives may feel that they should not be "stretched", that stress is bad for them and this results in over-protection and an emphasis on disability rather than ability. Sometimes this is still compounded by the unjustified assumption that physical disability is necessarily associated with reduced intellectual and cognitive function. Another factor is the severe difficulty in communication because of speech or language disorder which affects many disabled people and which causes immense frustration. Alternative methods of communication include not only new computer technology, but also some aspects of arts involvement. It is up to us to provide the opportunities for disabled people to find the most appropriate and rewarding ways of achieving some of these new-found expectations.

There are many activities in which disabled people have been outstandingly successful. The Paraplegic Games attract large numbers of people in international competition and those competing in the Special Olympics have shown a diversity of athletic potential that, once accepted, has shown us how meaningless the word handicapped can be. Many people's interests and talents lie more in the field of the creative arts and these also have much to offer to those with the more severe types of physical disability. Here too, many have overcome their disabilities to achieve wide recognition. One thinks of Izak Perlman, a victim of poliomyelitis, Jeffrey Tate (Principal Conductor at the Royal Opera House) who has spina bifida and had just been made President of the Association for Spina Bifida and Hydrocephalus in England, and Evelyn Glennie, a virtuoso percussion player in spite of being profoundly deafened at the age of twelve. In the world of literature we record the achievements of Christopher Nolan and Christy Brown, both

profoundly disabled by cerebral palsy, Stephen Hawking and Ivan Vaughan with chronic progressive neurological disease, and Feodor Dostoievsky and Gustave Flaubert, both incorporating their disability of epilepsy into novels.

It will not be given to many to achieve at this level, but the important point is that artistic talent is not the prerogative of the able-bodied, that creativity is not related to intelligence and that specific disabilities may not be as limiting as they appear. Sometimes this leads us to strange concepts — for instance, the belief that deafness is no bar to music. I have recently been working with a professional musician who was born with profound hearing impairment and has a sister similarly disabled. However, Paul was brought up in a musical family and he makes the point that being born with musical interest and ability is nothing to do with hearing — perhaps more a question of how the brain is wired up. Paul started to play the piano at an early age and had no difficulty in learning to read music (which again as a process does not depend on hearing). He began to learn the organ at age 14 and in due course applied to read music at University level. Four departments of music turned him down without interview, but Oxford had the foresight to realise that if a deaf person wanted to read music he must be given a chance to prove himself, and they found him to be one of the best students of his year. Paul has virtually no useful hearing, and hears practically nothing of what he plays, but is now an organist and choirmaster in Huddersfield and spends much of his time promoting the idea of music with deaf people. I have dwelt at some length on this story to emphasise again that we should be looking at people's ability, and not allow disability to carry with it unjustified assumptions about inability.

This is part of the philosophy behind the *SHARE-MUSIC* courses which we have been developing over the last few years. *SHARE-MUSIC* aims to give physically disabled young people an opportunity to share a wide variety of musical activities with able-bodied colleagues and helpers under the guidance of expert tutors. We share not only our skills and talents and enthusiasm but also our resources and social activities by making all the courses residential. We insist on having at least an equal number of helpers (many of whom are skilled musicians or students) and disabled people, and the courses have an important educational component for the helpers, who learn about the problems of disability in practical terms. We would always have a mixture of people with profound and mild disability and we do not look for any particular musical expertise although playing an instrument even at a simple level is a help. The emphasis of the courses is on creative music-making in groups and we usually put together a piece of music theatre which is entirely created by the participants themselves and leads to a final performance to a small public audience. We provide an enormous range of orchestral and school percussion instruments as well as pianos, electronic keyboards and computers.

In Northern Ireland we have also had the opportunity to use the Gamelan, a collection of instruments from Bali, owned by Queen's University and have found these particularly suitable for disabled people to play. With such a variety of resources we can always find some way in which people can contribute, no matter what their physical difficulties. In this sort of environment we see disabled people exploring ways in which they can express themselves and making discoveries, finding talents and abilities that they were not aware of, and developing ways of communicating with people in musical terms, when more conventional means of communication are sometimes denied them. For those with very severe

disability the introduction of computers with MIDI-linked keyboards has opened a new world of music-making. In the last two courses we have provided a fully equipped workshop running a system called Midigrid developed in the department of electronics at York University. This system requires no knowledge of conventional music notation and is activated by moving a "mouse". For those who cannot even do that, there is always an alternative way and two of our most disabled young people have become skilled at programming a keyboard or a drum machine with one foot and are in this way able to provide backing for a whole musical sequence.

An interesting feature of these courses has been the tendency for disability issues to feature prominently in the creative projects of the students. In 1988 a lady with severe limb deformities produced a simple song she had written about a young friend who was a ballet dancer and suffered a sudden paralysis from the waist downwards, putting an end to her career. The poem compared the threads of life to those of the weaver and emphasised how tenuous they are and how easily broken. A whole music theatre project lasting 25 minutes was built round this idea. Another student who had lost both legs from progressive disease wrote a song entitled "The incredible shrinking man". People's ability and desire to express their problems in artistic ways (musical, dramatic, visual or even in poetry) is part of the enrichment which the creative arts can bring into the lives of disabled people. Some might even regard it as therapeutic although we make a point that *SHARE-MUSIC* is not a course in or of music therapy.

The special relationship and potential of the arts for and with disabled people was recognised by the Attenborough Committee, set up by the Carnegie Trust, which produced a far-reaching report in 1985. This recognised that the arts are for everyone, not just the privileged few . . . . "To some degree it is within everyone's capacity to reinterpret their own experience and to share the experience of others by involvement in the world of art". The Committee took evidence from all parts of the UK and made some specific recommendations with regard to Northern Ireland, among which was that a pilot project should be set up to make recommendations as to how the arts and disability issue should be developed and co-ordinated in the province. With the help of the Northern Ireland Council for Disability and financial support from the Carnegie Trust, we appointed a part-time project worker about a year ago. As Chairman of the Steering Group, I have been specially concerned with this work and we have collected a large amount of data about what is going on in Northern Ireland. The brief of the project includes the setting up of a number of workshops in related topics, the first of which was held recently, concerned with access to arts venues. A lot of interest was shown in this, and we hope in due course to produce an access guide to arts centres, theatres, museums and cinemas in Northern Ireland. We are also interested in the potential of the community arts in relation to disabled people, and the Project Officer has been able to introduce artists with an interest in this work to local groups who are seeking help with specific projects.

Another particular interest of mine has been in the hospital arts. The Hospital Arts Movement started in Manchester some 15 years ago, and the Health Authority there now have several full-time artists working in health care settings. A department called Arts for Health was set up at Manchester Polytechnic last year and is in touch with over 230 hospital arts projects in Great Britain. One of these, I am

pleased to say, is in Belfast where we have now appointed an Artist in Residence to the Royal Group of Hospitals. The terms of reference of this post will be to enrich the total environment of the hospital by the introduction of arts activities of all sorts, to involve staff at all levels, visitors and patients as far as possible, and to liaise with local community groups and schools to bring their skills into the hospital environment. The underlying philosophy is that the arts can make a very positive contribution to health care and can encourage and hasten the healing process. The project at the Royal is being developed in association with the Arts Council of Northern Ireland, with major funding from the Gulbenkian Foundation. The DHSS has also contributed to the scheme and is itself setting up an advisory service to encourage other hospitals to explore the potentials of arts programmes. The performing arts are, of course, included in this programme and we have already held a number of lunchtime musical events in various parts of the hospital.

Music in hospitals is not, of course, new. Edward Elgar was appointed Music Director at the Worcester County and District Lunatic Asylum in January 1879. He was required to spend one day a week at the hospital and to direct the resident band which was mostly made up of hospital staff. He received a salary of £30 per annum, five shillings for each quadrille he composed and one and sixpence for every Christy minstrel accompaniment he arranged. He held the post for five years. Since 1947 the Council for Music in Hospitals has been promoting live concerts by professional musicians in hospitals, hospices and other institutions. In 1988 over 2000 such concerts were given in England and Scotland, and we have presented a number of tours by these musicians in Northern Ireland. The last was in August of last year when I myself joined two singers from the Council as accompanist and we gave eight concerts in five days in hospitals all over the Province. Another similar organisation is Live Music Now, which employs young professionals at the start of their career, and we have been exploring the possibility of extending their work to Northern Ireland. All this is in addition to the contribution which our own musicians, especially the groups from the Ulster Orchestra, make towards bringing live music into the hospital environment. There is abundant written evidence of the power that music has to stimulate the withdrawn, to awaken memories in the confused, to calm the anxious and to enrich the experience of people who are in threatening and sometimes uncongenial circumstances.

I have made little mention of the arts as therapy — another area in which I have been particularly interested, specially in relation to music therapy. I have been a member of the British Society of Music Therapy for many years and have recently been invited to be a Vice President. For the last two years I have been an examiner for the Music Therapy Diploma Course held at the Guildhall School of Music in London and from personal knowledge I have great regard for the dedicated work of Music Therapists. While traditionally much of their work has been with disabled children and with long-term mental illness and handicap, there is now an interest in exploring how music can help in other fields. One of the problems in relation to the creative arts as therapy is the question of evaluation. In the past there has been an emphasis on anecdotal evidence of benefit, but there is now a need for more objective evidence of effectiveness if scarce resources are to be used in this way. The need for research in music therapy has been recognised, particularly by the City University in London where a research fellow is appointed each year.

The present holder of the post is investigating the use of music therapy with Aids victims. Another research music therapist is working in the brain injury unit at the Royal Hospital & Home in Putney, investigating the part that music can play in the coma arousal programme in head injured patients and whether music can help the physiotherapist with the restoration of function to stiff and paralysed limbs.

Music therapy has been very slow to make its mark in Northern Ireland. One music therapist has been working in the Eastern Health Board for many years and another has recently been given a research grant to support her work with speech and language delayed children at Thornfield School. The Parkinsons Disease Society has supported some work to investigate how music can help people with this condition and some of the encouraging results might well be extended to other progressive disabling diseases. One or two students from Northern Ireland go to train in music therapy each year in London, but unfortunately we have not had the posts here for them to return to. However, a proposal to develop a Music Therapy Trust in Northern Ireland is now under way and we hope this will provide a focus for education and research, for the appointment of music therapists and for raising funds to support this work.

This sort of approach to disability does not easily fall within the thinking of doctors working in clinical medicine, who are trained to evaluate disability — its cause, diagnosis and treatment. The hidden ability, which everyone possesses no matter how severely incapacitated, may elude us. Only recently has the concept of looking objectively at the quality of life emerged as an important element in an overall view of health care. Oliver Sacks expresses it with characteristic insight " . . . . our tests, our approaches . . . . only show us deficits, they do not show us power; they only show us puzzles and schemata, when we need to see music, narrative, play, a being conducting itself spontaneously in its own natural way". Sacks speaks of the power of music in many of his writings, of its capacity for making people whole — a concept which has been with us at least since the time of the Greek philosophers. An important principal contained within the government paper "Caring for People" is the prominence given to a flexible system in which client choice and satisfaction are an integral part of the delivery of care. The Arts can make a positive and enriching contribution to restoring this harmony to disability.

In concluding I would sound a word of caution. Some people with severe and sometimes progressive disability seem to have within them a spiritual strength and tranquility which sustains them in the face of insuperable difficulties. The phrase "The harmony of heaven and earth" embraces the concept of bodily and spiritual unity and for some disabled people this inner resource may be sufficient to give them a purposeful and fulfilling life. We should consider whether we are justified in imposing our own ideas about quality of life upon them. We should recognise that some people achieve their own secret harmonies without our intervention — perhaps it is given to them to hear the Music of the Spheres, or the Song of the Angels — and we should remember that these issues should be handled with sensitivity. Our role is to encourage and to enable disabled people to lead as full and as meaningful a life as they wish within the constraints imposed by our present social customs.

The World Health Organisation in its manifesto "Health for All by the Year 2000" sets 38 targets for the European Region. The third of these reads " . . . . by the year 2000 disabled people should have the physical, social and economic opportunities that allow for at least a socially and economically fulfilling and mentally creative life". This is a message with which all of us undoubtedly agree.

The Jim Egar Memorial Lecture is given at the invitation of NICOD and commemorates the life and work of Mr J J Egar who died in 1964. He was founder, secretary and treasurer of the Northern Ireland Spina Bifida and Hydrocephalus Association, and Vice Chairman of NICOD for 10 years. He was awarded the MBE for his services to the community.

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Historical Note

The Anatomy Museum at The Queen's University of Belfast

D J A Heylings

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The one hundredth anniversary of the British Museums Association, the association of museum curators, took place in 1989, and was designated Museums Year. Throughout the country museums mounted special exhibitions to publicise their role. As I am the current curator now is a good time to give a brief overview of the history of the anatomy museum at Queen's and to assess its role now and in the future.

Until relatively recently the anatomy museum was part of the medical museum and therefore could be said to have originated when a museum room was formally established alongside the dissection room in 1835.[1] This was associated with the formation of the Belfast Medical School in the Collegiate Department of the Belfast Academical Institution, which in 1831 had become the Royal Belfast Academical Institution. Anatomy and physiology had been taught previously in the Institution from 1819 when James Lawson Drummond was appointed to the chair.[2] In 1849, after the retirement of James Drummond and the formation of Queen's College, within which the medical school formed a faculty, Hugh Carlile was appointed to the chair and with him came the entire contents, purchased for £500, of the medical museum of the Park Street School of Medicine in Dublin. This greatly increased the collection, commenced by Drummond, which was then temporarily housed in the general College museum located in the north wing of the original main building of Queen's College.

In 1863 the first medical building was completed on the east side of the quadrangle of Queen's College, providing a new dissecting room with a lecture theatre attached (Fig 1). Then in 1866 the second and larger section containing the museum room was completed, allowing the museum to move to this permanent home. There was still only a medical museum, part of which comprised the anatomical collection. In 1910 the museum roof, having been found to be in a dangerous condition, was supported by steel pillars. At the same time it was observed that the floor of the museum was a fire risk, being situated over the heating furnaces for the building.[3] Between 1911 – 13 the floor of the museum was fireproofed and a new extension to the building to house the anatomy collection was added — the Bone Room with a small lecture theatre beneath, later used for biochemistry. This occurred during the curatorship of Professor Johnson Symington, which was the heyday for the museum. It also appears to have been the norm that the head of the anatomy department was

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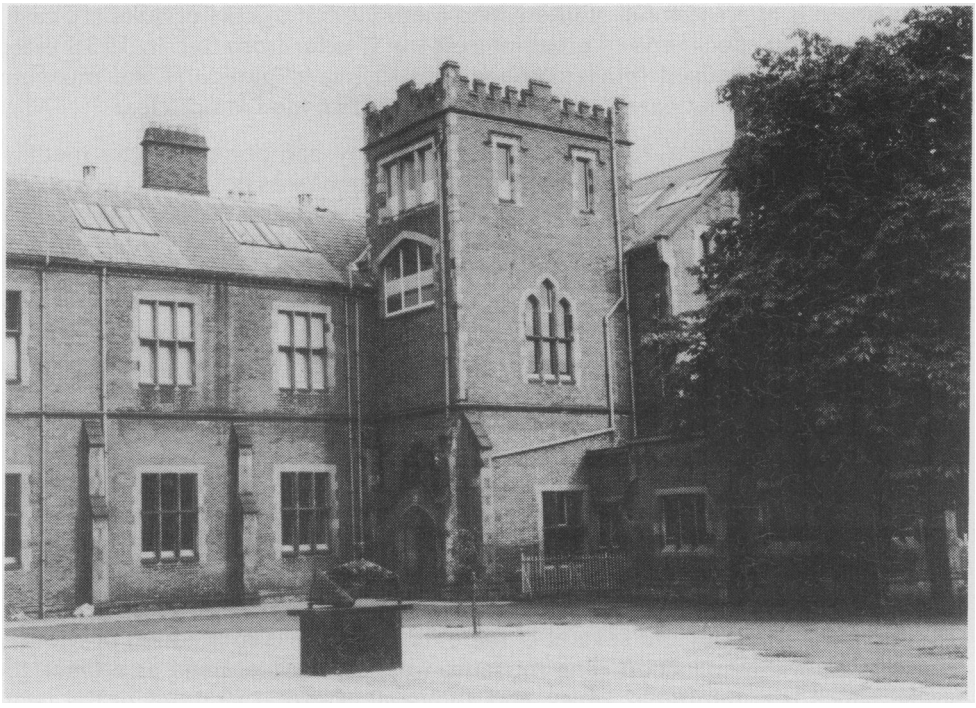

Fig 1. The original section of the medical buildings comprising the main door in the tower with the dissection room on the first floor to the left. *Reproduced with permission of Mr G Bryan.*

curator of the medical museum. This remained the custom until the pathological component was moved from the original museum room to new premises on the Royal Victoria Hospital site in 1933. The original museum room was then used for physiology lectures. The anatomy collection continued, housed mainly in the Bone Room, until it moved in 1969 to its present premises in the Medical Biology Centre.

The Medical Museum was one of four museums within the University at the turn of the century, the others being museums of Materia Medica, of Sanitary Science and of Classical Archaeology. All museums at this time provided regular summaries of recent activity for the Vice-Chancellor's annual report, but this stopped in the academic year 1924–25. In 1910 the Vice-Chancellor's report stated that the medical museum housed "collections of exceptional value and rarity", and in 1912–13 they were described as a "very valuable collection of specimens". It would appear that in its heyday there was also a large anthropological collection, to which in 1914 was added a "number of valuable specimens consisting principally of native Australian skulls and teeth ... Tasmanian skulls ... three well-preserved skulls belonging to the tribes inhabiting Northern Nigeria". Then in 1918–19 a Miss Bamford presented an Egyptian mummy, the embalmed body of a young female. In 1921–22 Dr R H Hunter made a large addition to the collection exhibiting ossification of the human skeleton. In 1924–25 there is a report of a benefaction:- "the University has to thank the Belfast Natural History Society for the permanent loan to the Medical Museums of a series of about 100 casts of an anthropometric interest". From these reports one would imagine that

the museum is at present full of interesting material, but this is not quite the case. There are many specimens of great interest but it is far from full. In 1938 there was a fire which caused much damage, destroying in particular the museum catalogues but to what extent the collection was damaged is not clear.

On its current site from 1968–1985 the anatomy component of the medical museum along with a few stray pathological specimens was housed in two rooms, the main teaching museum being on the first floor with open access for medical and dental students. This room housed specimens which illustrated normal osteology, human development and wet specimens preserved in bottles. The comparative, anthropological and archaeological collections were housed in a small room on the seventh floor with access for staff and research students only. In 1986, with reorganisation of space within the anatomy department, this latter collection was moved down to rooms adjoining the main museum, but still with restricted access. Over the past two years the museum contents have been sorted and a catalogue is in process of being prepared to replace the one lost in the fire. The current collection is divided into three main sections: comparative anatomy, embryological anatomy and gross anatomy. Each section includes osteological material alongside specimens preserved in jars, and to varying degrees histological material. The embryological section is greatly enhanced by a large collection of microscope slides, mainly produced by the late Dr W R M Morton. The Egyptian mummy and several of the other specimens mentioned previously are still within the collection. The museum was, and still is, used as a place for study, which is comfortable, quiet, and above all, warm at all times of the year.

What is the future for the Anatomy Museum? Within the Medical Biology Centre, with restructuring of departments in the university and a shortage of accommodation locally, its continued existence appeared to be doubtful. However, over the next decade it has been secured as a study area for undergraduate students and has been enlarged to double the seating capacity for at least 100 students. Accommodation now is in three rooms. The first is an osteological room, in which students will be able to study the development of bone along with all the normal osteology that is still expected of medical students. Within this room one wall will house the portraits of the past professors and a semi-permanent display illustrating their work, and the study of anatomy in Belfast. The second room is dedicated to displaying X-rays with at least forty-eight square feet of illuminated screens: these displays will be semi-permanent, relating to the current teaching programme. The main museum room will house various specimens in jars and models, all displayed to greater advantage than was previously possible. There will be several special cases to display the unusual and more interesting specimens from the reserve collection. It is proposed that an interactive video/computer system will be housed within this museum room (after the video packages have been developed). This new system will allow students to study anatomical specimens recorded on video tape through a programme running on a computer. The computer provides the options of either a tutorial study programme or a testing programme which will match the current teaching programme, both of which are tailored to each student, depending on his/her level of knowledge. The museum therefore will eventually be a place for private study, display of unique historical material, and will provide an interactive environment between students and the display material.

Fig 2. Detail from the lifesize portrait of Peter Redfern, Professor of Anatomy 1860–1893, painted by Ernest E Taylor (1863–1907).

In building towards this vision of the museum, the new catalogue is the first phase and is nearing completion. The second phase of restoration has already commenced with cleaning and restoration of the lifesized portrait of Peter Redfern (Fig 2), painted by Ernest E Taylor in 1894. It has been suggested that the portrait contains a second portrait in the mirror of the microscope, possibly of the artist. Close inspection does not indicate so and the painter has attempted to record the reflections of the instrument itself. This was an important beginning, as Peter Redfern was perhaps one of the earliest teachers of histology in these islands and as professor saw the number of medical students increase to a peak of 364 in 1881. It is therefore appropriate that we are able to study more clearly the detail of the microscope and the drawing draped over the table in the background. Is the drawing an early illustration of cardiac muscle, or what type of tissue does it represent? The drawing is in a style similar to that of other paintings within the museum collection. This was the first portrait to be restored and though expensive it has been rewarding. This year restoration of the portrait of Hugh Carlile has followed. The museum houses a very large collection of anatomical paintings, all in watercolour, which vary considerably in detail and quality. All have one common factor, the need for cleaning, paper restoration and conservation. Perhaps of greatest interest are the three-times-life-size paintings of transverse sections of Man 50, various sections of Man 24, Girl 18 and others produced during the time of Symington. Many paintings show evidence of smoke damage and perhaps some were lost in the fire of 1938. The paintings were used to illustrate points during teaching, thus avoiding the need to use the lantern lamp, and so perhaps will be fondly remembered by old graduates of Queen's. The oldest paintings, including a set of four paintings of the perineum, are signed by Hugh Carlile and dated October 1853. They are outstanding in quality and clarity, and the colours are as new. There is no indication, though, that Hugh Carlile was the painter and his signature could simply indicate that on this date he accepted the paintings into the department. They are in urgent need of paper conservation and cleaning before they can be displayed. Within this part of the collection also are the proof prints of the famous cross-sections of Man 50 published by Professor Symington in 1917. In association with these is the wooden reconstruction of Man 50, sadly lacking the thorax but of great value as a teaching aid as well as being an excellent example of hot wire work.

Many of the bottled specimens require attention to the topping up of fluid and resealing. New key diagrams are needed to explain the structures demonstrated

in each specimen. Renewal of the museum will take time due to the expense of conservation and restoration, and the shortage of skilled technicians and illustrators. It is fortunate that the Egyptian mummy (Fig 3) has survived the passage of years in excellent condition; it was recently examined with staff from the Ulster Museum conservation department.[4] X-rays were carried out and the examination confirmed that the mummy was a female, 5 to 6 years old at the time of death. The full mummification rites had been performed using the oral route through the basilar portion of the occipital bone to remove the brain, and evisceration of the body cavity through an incision of the left flank. Linen was used to pack the body cavity and only remains in the thorax. Within the linen there is a shadow, possibly the heart, which was normally left *in situ*. An unusual finding was the total removal of psoas major from both sides of the lumbar spine. The left innominate bone had been disarticulated at the sacroiliac joint and the pubic symphysis. This occurred during life and was associated with the swelling seen in the left thigh. These changes are not normally associated with artifacts produced by the mummification process. This gives a possible primary cause of death, pelvic fracture, there being no other signs of chronic illness as would be indicated by the presence of Harris lines within the bones. Recently sponsorship has been obtained which will allow the mummy to be displayed alongside the X-rays within the main museum in a secure environmentally controlled display case.

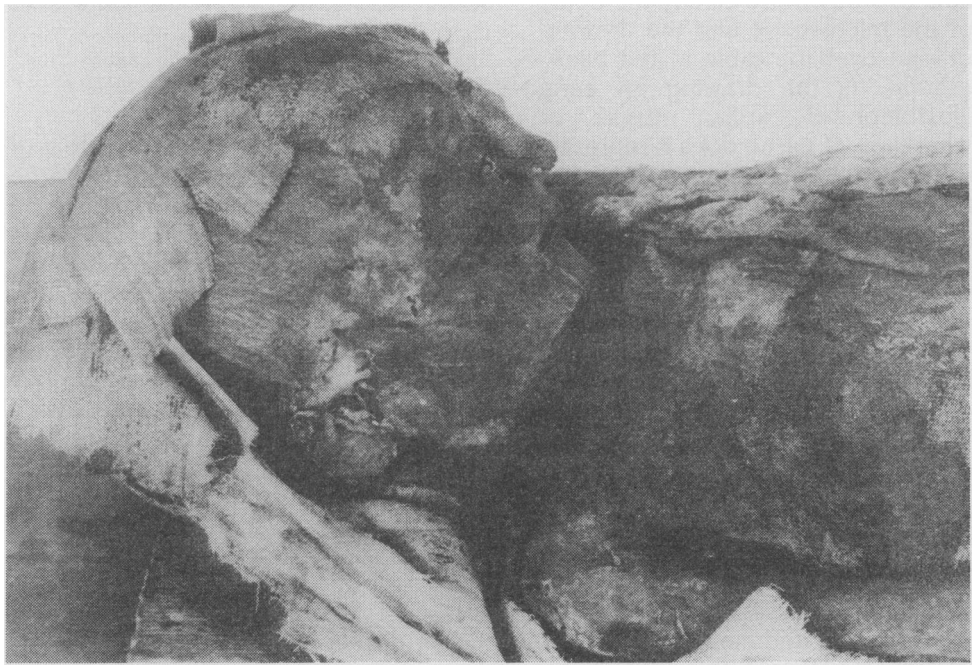

Fig 3. The mummy of an Egyptian child, presented to the medical museum by Miss Bamford, 1918.

Unfortunately, due to the change in the undergraduate course and the reduced time to study human anatomy compared to earlier days, there is no longer the opportunity for students to study comparative anatomy. This part of the collection will be housed separately in a store and made available on request.

The role of an anatomy museum in a modern department was aired recently at the Anatomical Society of Great Britain and Ireland, anatomists being asked if the anatomy museum was "dead or alive".[5] From this meeting contacts are being formed with curators of other anatomy museums to facilitate the exchange of experience in various conservation problems. Museums are traditionally places of conservation, storage, and display of any item of interest: in more recent times they have placed the role of education more highly in their activities. It is not sufficient today for a museum to place objects on a shelf to be admired and preserved for posterity; it must educate as well. Conservation is expensive and must be justified, generally by the rarity of an item globally, and more specifically, within the collection of which it forms a part.

With these points in mind, I wish to develop the role of the Anatomy Museum in Belfast along the following lines: to educate undergraduate students not only in normal anatomy but also in the history of the department and medical school to which they belong; to provide a resource for research workers, both local and national; and to offer space to any material that is of interest to students of anatomy or the history of the medical school in Belfast. There will be no strict interpretation of the term anatomy. The museum will have sections covering the main aspects of anatomy within the current curriculum, with special areas displaying various aspects of the history of Queen's in pictures, specimens and equipment. An old electron microscope from the Physiology department has already been obtained, which will be the central exhibit in the display charting the development of histology. This will demonstrate equipment used locally from the time of Peter Redfern, the Cambridge rocker microtomes of 1890 and 1894, through to more modern microtomes, displayed with microscopes used in the department up to recent times. For this revitalisation I need to collate information on the history of the museum and I would be grateful for any photographs of the old medical museums or their contents, or recollections that may help in the identification of those specimens which sadly have become separated from their labels.

In summary, it is my intention that the anatomy museum will continue to be a place of private study for Queen's students. It will allow access to all specimens of interest, displayed to the best educational advantage. The atmosphere I hope will at the same time immerse the student in the history, not only of anatomy, but also of medicine at Queen's University.

I wish to thank Dr George Cowie for his invaluable assistance in researching old University records, and all authors who have researched the history of Queen's, in particular Sir Peter Froggatt for his very useful papers on the history of the medical school.

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Historical Note

The centenary of the admission of women students to the Belfast Medical School

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At a dinner in the Great Hall of the Queen's University of Belfast on the 12th December 1989 the Northern Ireland Branch of the Medical Women's Federation celebrated the centenary of the first admission of women to the classes of the Faculty of Medicine of the Queen's College, Belfast, and to the clinical classes of the Belfast Royal Hospital. In September 1889 a meeting of medical professors approved their admission to the College and in November 1889 the College Council authorised it.[1] On 10th September 1889 the medical staff of the Belfast Royal Hospital recommended that lady students should be admitted to hospital classes[2] and on the 14th September the management committee accepted their advice.[3]

The movement of women's education began in England in the 1860's. The whole basis of women's advancement in life was education. Without it only the lowest and meanest work was open to them. In 1870 the question came before the Council of the Queen's College Belfast.[1] Thomas Andrews, Vice-President proposed that they should be permitted to attend particular courses of lectures "if the Professors considered it expedient, and were satisfied that the discipline and instruction of the classes would not suffer". 'Females' thus attending would be required to pay a special registration fee in addition to the ordinary class fee and to sign an engagement to conform to the discipline of the class. They would not be eligible for scholarships or prizes or enjoy any of the privileges of the male students. Professor Redfern, President of the Biology Section objected to these restrictions and wanted women to be admitted on equal terms with men, if the charter permitted. However, the matter was deferred indefinitely.

In 1873 the Belfast Ladies' Institute requested the Senate of the Queen's University in Ireland to admit women to its degrees. "It would be a source of the keenest satisfaction to us if an Irish University should be the first to do an act of justice to the half of the community which most needs it". At the same time a Miss Mary Edith Pechey who had studied medicine at Edinburgh applied for permission to graduate MD after keeping one session in a Queen's College. This was made a test case and the law officers of the Crown gave a negative judgement. In 1876 there was passed an amendment to the Medical Act, which provided that any licensing body wishing to do so could grant medical qualifications without distinction of sex. Again Miss Pechey requested permission to attend 4 courses at the College for one session. The Senate of the Queen's University in Ireland decided by 6 votes to 4 to grant her request but the Council of the Belfast Queen's College refused to admit her.

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In 1882 the Belfast Ladies' Institute again approached the College in an attempt to get women admitted. The Royal University of Ireland which had now replaced the Queen's University in Ireland was open to women, and the President of the Queen's College Belfast, JL Porter, supported the application. It was perhaps relevant that he had three clever daughters. The Council agreed, rather cautiously, to admit women to Arts classes and a dozen attended the College during the session 1882–83, reportedly "without serious mishap". That must have been a relief to the authorities. Their relief was short-lived. There was astonishment and dismay when the women, though few, proved to be serious competitors and took top places in college and university examination. The question of eligibility of women for college prizes and scholarships was referred to the law officers of the Crown who said they were not eligible, as the law was. JL Porter, the President, called for an amendment to the College Statutes but it was not until 1896 that the necessary steps were taken and equality was achieved. Though women were admitted to the Arts faculty of the University it was another 7 years before they were able to enter the Medical faculty. The establishment of the Royal Colleges and of the universities had excluded women from medical classes.

The General Medical Council had been established after the Medical Act of 1858, which required that persons requiring medical treatment should be able to distinguish between qualified and unqualified medical practitioners. A Register had been commenced of qualified doctors who alone were entitled to sign death certificates or claim legal protection in their work. In theory there was no bar to a woman getting on the Register. In practice there was no way in which women could attend medical classes or become qualified. The first woman to get on the British Medical Register was Elizabeth Blackwell.[4, 5] Born in Bristol she had gone to America in childhood. After a very long and hard struggle against prejudice she became MD of the Geneva School of Medicine in New York State, in 1849. She got on the British Medical Register in 1859. The second was Elizabeth Garrett[5, 6] from Suffolk who became a Licentiate of the Society of Apothecaries in 1865 and got on to the Register in 1866. These two remarkable women had broken down the barriers and opened the way for women doctors in the future. Sadly many of these barriers were hastily re-erected but they were never again so hard to overcome.

In the Queen's College Belfast in 1889 three women who had completed one year's study in the Arts faculty applied to transfer to the faculty of Medicine and were permitted to do so. They were Elizabeth Gould Bell and her sister Margaret Smith Bell, from Newry, and Harriette Rosetta Neill from Belfast.[7]

In and after 1889 it was usual for medical students to enrol for hospital classes as soon as the first college year was completed. Until 1889 hospital classes were at the Belfast Royal Hospital only, but from then on the wards of the Belfast Union Hospital were also open to students. The Royal Hospital was in Frederick Street until 1903 when it moved to its present site on the Grosvenor Road, having become the Royal Victoria Hospital a few years before its move. The Belfast Union Hospital became the Belfast City Hospital, still on the same site. There was no opposition to women medical students from the staff of the Royal Hospital. When the question was brought up at a meeting attended by Drs Wheeler, Sinclair, Byers and Whitla, with John Fagan in the chair, the feeling of the members was that "there should be no restrictions or objections made to their admission".[2] This was very different from the plight of women students in Edinburgh who were refused admission to the Royal Infirmary until they had

raised £700 (a huge sum in those days) to furnish two wards in which and in which alone they could work.[8] They were excluded from the classes of the medical faculty of Edinburgh University but did their studies at the Edinburgh School of Medicine for women which was founded in 1877 by Dr Sophia Jex-Blake.[9]

Elizabeth Bell graduated MB, BCh, BAO, RUI in 1893. She published details of *"A Curious Condition of Placenta and Membranes"* in the annual report of the Northern Ireland branch of the British Medical Association for 1895–96. In the medical register of 1910 she was living at 83 Great Victoria Street, at that time a fashionable place to live. In 1925 she was at 4 College Gardens. The University Common Room is now on the site. The last entry was in 1934. She was well known and respected. Margaret Bell became LRCPI & LM and LRCSI & LM in 1894. In the medical directory of 1905 she was in Manchester but there is no subsequent entry for her. Harriette Neill graduated MB, BCh, BAO, RUI in 1894. Over the years she had various addresses in Bangor. The last found in the medical register was in 1941.

The medical faculty in 1889 did not need to fear that they would be swamped with women students because for the next four years there was only one woman in each year on the books of the Royal Hospital. There were five in 1895–96, three in 1896–97, three in 1897–98, and one in 1898–99. The Council of the Queen's College was in no way a pioneer in allowing women their rightful place in the academic field. In 1889 the Women's Medical College of Pennsylvania, which had been in existence for 37 years, had graduated 36 women that session including an Indian and a Japanese.[10] That same year the total number of women doctors in the USA was estimated to be 3000.[10] In Europe 700 Russian women took the medical degree in the 10 year period 1872–82, mostly at the University of Berne.[10] They were now (1889) to be allowed to practise medicine in Russia but could only attend adults of their own sex and children of both sexes.[11] In India there were 200 women studying medicine in the medical schools of Bombay, Calcutta, Lahore, Madras, Hyderabad and Agra.[12] Many of these women students carried off prizes and honours in every branch of medicine.[13] Compared with these impressive statistics three women in Belfast in 1889 was meagre.

Although women could become doctors it took very many years to overcome the prejudice against them. They were excluded from learned societies.[14] In 1874 Elizabeth Garrett had been elected a member of the British Medical Association. In 1875 she travelled to Edinburgh to read a paper at the annual conference. The President, Professor Christison, did his utmost to prevent her from giving her paper but could not.[6] During the next three years the BMA circularized all its 6000 members on the admission of women. About 4000 replied and of these three to one voted against having women members. Sir William Jenner threatened to resign if they were admitted. Punch enquired "what can the Council do to please Sir W Jenner? Only turn the *young* women out of their Society? The BMA will always contain a certain number of irremovable *old* women". The decision by the BMA in 1878 was not made retrospective, so for the next 19 years, until 1897, Elizabeth Garrett Anderson (as she had become) was its solitary woman member. The Physiological Society founded in 1886 elected its first 6 women members in 1915, Professor Winifred Cullis being one of them. The Anatomical Society of Great Britain and Ireland and the Royal Society of Medicine were also very slow to admit women. Pay was unequal between men and women for the same post.

Able and active women doctors founded the Association of Registered Medical Women in 1878 for scientific discussion and dining. Local associations gradually

developed in other parts of the country as many problems arose in relation to the status of women, to employment and to medico-legal social and political problems. By the time of the first world war the need for a single representative body was apparent. In 1916 representatives of local associations met and pooled ideas for the constitution of this body. Articles of Association of the Medical Women's Federation were signed in February 1917 and the first meeting was held in Manchester in June 1917. Since then it has done invaluable work in many spheres. It has produced reports on a large range of subjects.[14] It has given valuable information to the Royal Colleges and acts as a channel for opinions on matters affecting the whole community. It is part of a larger, world-wide organisation, the Medical Women's International Association of which it was a founder member in 1922. It is still working to gain equal treatment, for example in superannuation and tax allowances. It has achieved much. In World War II women doctors had equal rank, pay and conditions with their male colleagues. Here in Northern Ireland we have a good record of having women as heads of departments, as consultants, as holders of senior administrative posts and as senior partners in general practice.

We have had a branch of the Federation in Northern Ireland since 1941. Before that, individual doctors belonged to the Great Britain association and travelled over to attend meetings. In summer of 1941 a meeting was held at Dr Mary Erskine's house at Whitehouse. It was a lovely summer day. We walked in the garden and had afternoon tea in the long drawing room, while Dr Violet Reilly played the piano. Then we had the aims and objects of the Medical Women's Federation set out for us by Lieutenant-Colonel Letitia Fairfield, in army uniform. We then voted in favour of forming a Northern Ireland Branch, which held its first meeting in November of that year. The first President to be elected was Dr Beatrice Lynn, an ophthalmic surgeon. This was almost half a century ago, and 52 years after the events of 1889. Many younger doctors believe that the need for a watchdog for women's rights no longer exists. Those of us who have seen inequality and exploitation believe that there is still a need to be eternally vigilant and to have a channel through which our views can be expressed.

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Historical Note

Vincent Ketelaer did *not* describe tropical or non-tropical sprue or coeliac disease in his book *De Aphthis Nostratibus seu Belgarum sprouw*

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It has been said repeatedly [1, 2, 3] but erroneously that Vincent Ketelaer described tropical or non-tropical sprue in his book *De Aphthis Nostratibus.*[4] The error arises because it has not been understood that the Dutch word sprouw, as Ketelaer used it, meant and meant only stomatitis. In particular, as can be seen from Ketelaer's description, it meant mainly thrush, the common monilial infection of the mouth. Although for the most part his description is that of thrush, he may also have included some exudative inflammations of the fauces, perhaps diphtheria, glandular fever or Vincent's angina. Ketelaer expressly says that his aphthae are not the same as those of Hippocrates which were ulcerated and inflamed and occurred elsewhere as well as in the mouth. That thrush was one kind of aphthae is confirmed by the entry under that term in the Medical Lexicon of S Blancardius[5] (Blankaart) which describes "the whiteness as if the whole mouth had been sprinkled with white flour". In English, sprue continued with some authors to mean oral thrush, until the latter part of the nineteenth century. Doctor John McCaw,[6] the Belfast paediatrician, in his book *Aids to the Diseases of Children,* published in 1899, gives sprue as a synonym for oral thrush. This usage has died out in the English language and sprue is now only used of the tropical or non-tropical malabsorbtive disease.

The Belfast Medical Library possesses a copy of the 1715 edition of Ketelaer's book, and also a typescript copy of Doctor Hugh Calwell's translation of it into English. Scrutiny of these two texts makes it clear that in this edition there is no description of tropical or non-tropical malabsorbtion, nor any of coeliac disease. Sheehy's[1] reference is to the 1672 edition. Bartholomew's[2] is to the 1715 edition. Manson-Bahr[3] correctly says that "an aphthous stomatitis, which is frequently seen in badly nourished children and women, and which was popularly known in Holland as sprouw, has no apparent relation to tropical sprue". Curiously, inconsistently, and wrongly, on the same page he asserts that "the first accurate description (of tropical sprue) is undoubtedly that given by Vincent Ketelaer in 1669". Curiously again the reference he gives is to the edition of 1672.

Ketelaer was born in Vlissingen in 1627 and died in Zierikzee in 1679.[7] He began his medical studies at Utrecht and Leiden in 1647. His studies included literary subjects and he had some reputation in Latin poetry. He began practice as town

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doctor in Zierikzee, and in 1664, presumably because of his classical accomplishments, he became rector of the Latin school in that place. But one must say that the Latin of his book is neo-Latin of the 17th century and is not Ciceronian. He published his book *De Aphthis Nostratibus* in 1669, and there was a reprint, possibly a new edition, in 1672. These were the only editions published in Ketelaer's lifetime. I have not been able to consult them because the British Library will not lend books dated earlier than 1801. It is a presumption, but perhaps a reasonable one, that the printers of the 1715 edition, the first of the Latin text after Ketelaer's death, did not excise a description of tropical or other malabsorbtion. A list of all known editions of *De Aphthis* is included in Doctor Hugh Calwell's translation. His biographical details [7] do not record his ever having travelled outside Holland. Moreover *"nostratibus"* means specifically "of our country". He married in 1657 and had four children.

It is beyond the purpose of this note to trace in detail the transfer of the popular Dutch name for stomatitis, sprouw, to the tropical malabsorbtive disease now known as sprue. The sore mouth of tropical malnutrition is sometimes so very sore as to be the most distressing symptom. It would not be difficult for Dutch physicians in their East Indies to transfer their popular name for a sore mouth to a disease they eventually recognised as a major cause of it. That one old name for sprue was aphthae tropicae seems to show an evolution of thought and nomenclature from 17th century Holland through the practice of the Dutch East Indies to modern tropical medicine.

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Case report

Beçhet's disease presenting with mononeuritis multiplex

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Beçhet's disease was probably first described by Hippocrates,[1] "Many had their mouths affected with aphthous ulcerations. There were also many defluxations about the genital parts, boils externally and internally about the groins". In 1937, Hulusu Beçhet described the clinical triad of oral ulceration, genital ulceration and uveitis, now known to us by his name.[2] The presence of multiple mononeuropathies in a patient with Beçhet's disease is rare, and has not previously been reported outside Japan. We describe a patient with Beçhet's disease who presented with skin folliculitis and a rapidly progressive clinical picture of mononeuritis multiplex and died from brain stem involvement.

CASE REPORT

A 51-year-old construction worker was admitted to a district general hospital with weakness of the left hand and mild unsteadiness of gait. Four months earlier he had attended a skin clinic with a persistent rash on his lower legs: skin biopsy had shown folliculitis, but no evidence of vasculitis. The weakness in his left arm progressed, his gait deteriorated and he developed sore red eyes. When transferred to the neurological department he was thin and depressed. He was apyrexial. There were a few small aphthous ulcers on his tongue, but no genital ulceration, and a pustular erythematous rash on both legs below the knees, with marked bilateral ankle oedema (Fig 1). Both conjunctivae were congested and the right pupil was dilated and reacted sluggishly to light. Visual acuity in the right eye was restricted to appreciation of hand movement only, but was normal in the left eye. Slit-lamp examination revealed the presence of cells, flare and keratic precipitates in keeping with bilateral anterior uveitis. Fundoscopy showed retinal vasculitis in the right eye. There was severe weakness of the left hand and of wrist extension, and moderate weakness of elbow extension. Power in the right arm was normal. There was severe weakness distally in both legs. Reflexes were normal in the arms, but diminished in the legs. Sensation was intact. He could walk with assistance. He required urinary catheterisation shortly after admission.

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The ESR was 120 mm/hr, C-reactive protein 22·5 mg/l (normal < 6 mg/l), aspartate transaminase 50 U/l (normal range 10–40 U/l), alanine transaminase 156 U/l (normal range 10–45 U/l), total haemolytic complement 763 units/ml (normal range 250–700 units/ml and immune complexes 263 microgram/ml (normal range 0–49 microgram/ml). Motor nerve conduction was slow in the left upper limb. Sensory potentials were not recordable. Repeat skin biopsy showed changes consistent with folliculitis, with no evidence of vasculitis.

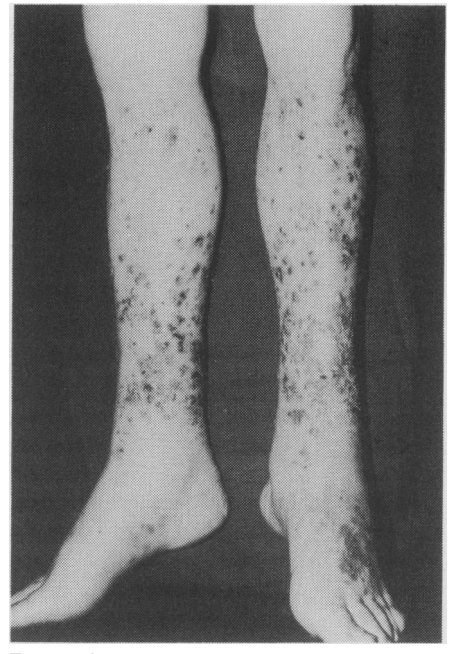

Fig 1. Rash on lower legs due to folliculitis.

The neurological presentation was of mononeuritis multiplex, but the associated uveitis led us to consider Beçhet's disease, sarcoidosis or the acquired immune deficiency syndrome. The additional features of aphthous ulceration and folliculitis suggested that Beçhet's disease was the most likely diagnosis. In spite of treatment with prednisolone 80 mg daily and azathioprine 50 mg twice daily, he developed progressive distal weakness in the arms and legs, and his general condition deteriorated. Azathioprine therapy was discontinued after 10 days, due to thrombocytopenia.

Six weeks following admission he developed slurred speech, right-sided facial weakness and a gaze palsy to the right. CT scan of the head showed multiple low attenuation non-enhancing areas in the brain-stem and periventricular areas. CSF protein was 1·5 g/l, glucose 3·3 mmol/l (plasma glucose 6·8 mmol/l), and there were no white cells. Cyclophosphamide 50 mg tid was added to the steroid therapy, but his condition deteriorated with the development of bilateral bronchopneumonia and he died seven weeks after admission.

At autopsy, sections of the cerebral hemispheres and brain stem revealed multiple foci of perivascular lymphocytic cuffing with fibrinoid necrosis of the vessel walls, in keeping with vasculitis. Small areas of infarction were also seen, although the neurones were well preserved, and also areas of meningeal reaction with lymphocytic infiltration and of demyelination. The overall appearances were of an intense vasculitic inflammation and encephalitis, but the degree of inflammation was out of proportion to the vascular involvement.

Sections from the spinal cord showed prominent demyelination particularly in the posterior and lateral columns of the cord, and vasculitis with fibrinoid necrosis of the walls of the blood vessels. Sections from several peripheral nerves showed foci of lymphocytic infiltration within the nerve bundles (Fig 2), amounting to nerve infarction, and the perineural vessels also showed evidence of vasculitis. Sections from skeletal muscle showed mild variation in fibre size with evidence of fibre grouping, in keeping with denervation. There were also vasculitic lesions elsewhere including the liver, spleen and small nerve plexuses in the urinary bladder.

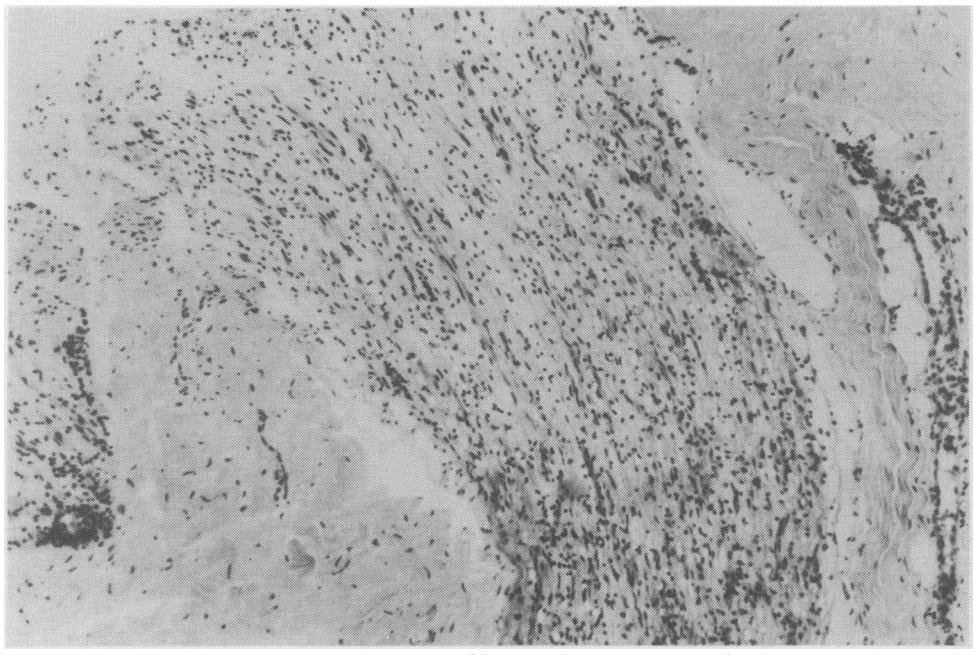

Fig 2. Peripheral nerve (H&E stain) × 60 magnification. Nerve infarct and vasculitis.

DISCUSSION

The clinical triad of relapsing iritis and ulcers of the mouth and genitalia named after Beçhet[2] is part of a systemic disorder of unknown aetiology. Other manifestations include erythema nodosum, folliculitis, pyoderma, thrombophlebitis, pericarditis, arthritis, epididymitis and ulcers of the gastrointestinal tract.[3] The diagnosis of Beçhet's disease may be missed or delayed if only the classic triad is sought. In the absence of any single specific diagnostic test for Beçhet's disease, various sets of diagnostic criteria have evolved. Major features of the disease include orogenital ulceration, eye lesions and skin lesions.[4] Minor features include central nervous system (especially brain stem) involvement, arthralgia or arthritis, intestinal ulcers and epididymitis. A definite diagnosis can be made if three major features, or if two major and two minor features are present.[4] In our patient, the diagnosis of Beçhet's disease is supported by the coexistence of aphthous ulceration, uveitis, retinal vasculitis, folliculitis, brain stem involvement and the histopathological findings.

The association of neurological symptoms with Beçhet's disease was first reported by Knapp in 1941.[5] Later, Berlin reported the first autopsy case of Beçhet's disease with involvement of the central nervous system.[6] Approximately 25% of patients with Beçhet's disease have neurological manifestations, although these antedate the more diagnostic criteria of aphthous stomatitis, genital ulceration and uveitis in only 5% of cases.[7] Neuropathological criteria for neuro-Beçhet's disease have not yet been fully established. An early classification divided the neurological manifestations of Beçhet's disease into three groups of brain stem syndrome, organic confusional state and meningomyelitic illness. Since then, many other neurological manifestations have been reported, including cranial nerve palsies, meningoencephalitis, hemiparesis, pseudobulbar palsy, epilepsy,

spinal cord involvement with Brown-Séquard syndrome and benign intracranial hypertension.[8, 9] Peripheral nervous system involvement in Beçhet's disease is rare: we could find reports of only eleven cases, of which three had mononeuritis multiplex.[10, 11, 12]

The clinical evidence of brain stem involvement in the final stages of our patient's illness is in keeping with previous pathological observations, which have shown this to be the commonest site for neurological involvement in Beçhet's disease. Computerised tomography of patients with neuro-Beçhet's disease has no pathognomonic features. Our patient's first computerised tomographic scan showed no abnormality, the second showed multiple low attenuation areas in the brain-stem and periventricular areas compatible with the post mortem finding of infarction. It has been suggested that magnetic resonance imaging may be more sensitive than CT scanning for detecting brain stem involvement in patients with neuro-Beçhet's syndrome.[13, 14] Therapeutic options in severe Beçhet's disease include steroid and azathioprine therapy, chlorambucil, cyclophosphamide or acyclovir therapy.[15] Assessment of treatment is difficult due to the rarity of the disease and its unpredictable course. The absence of response to prednisolone therapy is not unusual: in one series of 75 patients with Beçhet's disease with neurological involvement, steroids were ineffective in 50%.[16] Clinical remission and disappearance of magnetic resonance imaging changes have been observed following high dose steroid therapy, albeit in small numbers of patients.[14, 17] Whilst the effect of prednisolone therapy on the neurological manifestations of Beçhet's disease is unpredictable, the treatment of retinal vasculitis has been clarified: urgent therapy with local and systemic steroids is indicated to limit visual loss.[1]

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Case report

Pneumothorax following breast aspiration

L H Stewart

Accepted 17 May 1990.

Breast cyst aspiration is a minor procedure commonly performed in surgical outpatient clinics, and should be free from complication. The occurrence of pneumothorax after aspiration prompted a literature review. A simple method by which this serious complication can be avoided is described.

CASE REPORT

A 46-year-old lady returned to surgical outpatients with a recurrent left breast cyst which had been aspirated six weeks previously. She was thin and nervous, on treatment with diazepam and tri-cyclic anti-depressants. She was distressed that the cyst had recurred but lay quietly as aspiration was performed. Multiple cysts were present, and when the first one had been aspirated the 21FG needle was gently repositioned for the next. During this procedure the patient suddenly sat upright on the couch thereby impaling herself on the needle. She appeared to be none the worse as a result and the operation was completed. She then left the clinic. Moments later, she was brought back into the clinical room complaining of left-sided chest pain, clutching her chest and exclaiming that she was having a heart attack. Clinically she was not cyanosed nor short of breath but she had decreased breath sounds over her left chest consistent with a pneumothorax. X-ray confirmed a partial pneumothorax. A chest drain was inserted with immediate re-expansion of the lung and she made an uneventful recovery.

COMMENT

In 1978 Orr and Magarey reported three similar cases, two of which required intercostal chest drainage; the third developed only a small apical pneumothorax which was treated expectantly. All three made satisfactory recoveries.

The diagnosis and treatment of breast cysts by aspiration is a well-established and widespread practice, and is considered a safe procedure.[1,2] The only complications reported in the literature are pneumothorax,[3] breast haematoma,[4] and epidermal inclusion cysts of the breast.[5] It seems reasonable to suggest that pneumothorax would be most likely to occur in thin, nervous ladies with small breasts and thick walled or multiple cysts, where cyst penetration is difficult, necessitating repeated repositioning of the needle. Production of a pneumothorax must involve penetration of the lung by the aspirating needle, either due to sudden penetration of a thick walled cyst, or to sudden unexpected movement

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by the patient. In either event this serious complication may be avoided altogether by the simple precaution of aspirating all breast cysts with the needle held tangentially to the chest wall, and not at right angles, as is implied in some major surgical texts.[6]

I would like to thank Mr T K Day FRCS, Consultant Surgeon for his permission to report this case.

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Case report

Self-mutilating behaviour and deliberate ingestion of foreign bodies

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Accepted 26 April 1990.

Ingestion of foreign bodies and self-mutilation by individuals with personality disorders and low intelligence are well documented in the literature. We describe a patient with these behaviour patterns and discuss aspects of diagnosis, surgical management and cost of treatment over a six year period.

CASE HISTORY. The patient is a thirty-seven year old single man of below normal intelligence (IQ = 74, Wechsler Adult Intelligence Scale). He has always lived at home with his parents and has never formed any lasting relationships outside his immediate family. He left school at the age of sixteen, and worked in unskilled jobs until six years later when he started to abuse alcohol, after which his work record deteriorated. He first presented to hospital aged 25 years in 1979 having taken an overdose of sleeping tablets. Not until 1982 did he begin coming regularly to the acute general hospitals in Belfast with ingestion of various foreign bodies, overdoses and superficial cutaneous injuries (Figs 1 and 2). Typically these episodes occurred in the context of an episode of family friction and alcohol consumption. He has been admitted to psychiatric hospitals on twenty-five occasions, and has been diagnosed as having a personality disorder with no evidence of mental illness. Throughout this time he has generally been uncooperative with any attempts at therapy or rehabilitation. During the period 1982 – 1988 he required admission to acute medical or surgical beds on more than sixty occasions following episodes of self harm or foreign-body ingestion (Table).

During this time he has had approximately 250 X-rays performed and has spent more than 170 days in general hospital beds. Abdominal surgery has been required twice, endoscopy on at least five occasions and a few minor operative procedures for removal of needles stuck in various parts of his body. The estimated cost of his acute general hospital admissions alone exceeds £26,000 based on the estimate that an NHS acute bed costs on average £130.00 for 24 hours and an X-ray £12.00 per unit, although costs in different hospitals vary (quotation for cost year 1988 – 89). This excludes theatre time, admissions to psychiatric units and attendances at accident and emergency departments.

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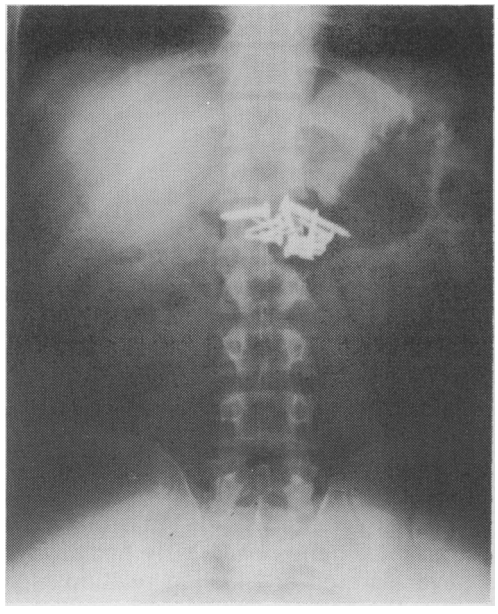

Fig 1. Screws ingested.

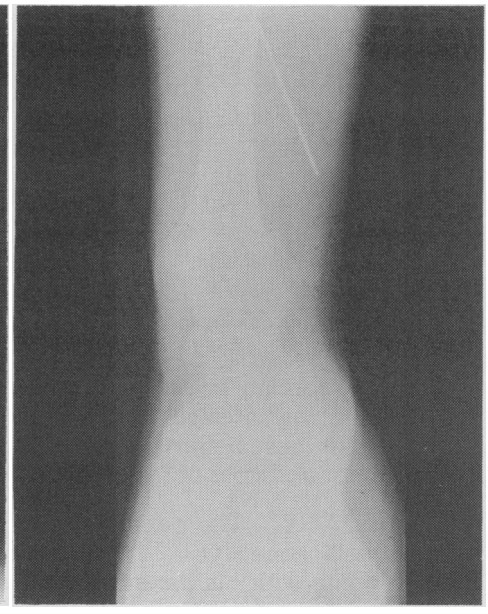

Fig 2. Needle inserted at wrist.

TABLE

Details of admissions to individual hospitals

	Number of admissions for			*Total*	*Days in hospital*	*X-rays taken*
	Overdose	*Self-mutilation*	*Foreign body ingestion*			
Belfast City Hospital	5	5	6	16	34	70
Royal Victoria Hospital	2	2	10	14	68	60
Mater Infirmorum Hospital	9	3	18	30	68	100
Ulster Hospital	2	—	—	2	2	10
Whiteabbey Hospital	1	—	—	1	1	3
Total				63	173	243

Despite the large number of foreign objects ingested over the six year period there have been few complications. His first operation was performed when he presented with generalised peritonitis after ingesting a cocktail fork. The fork was found at laporatomy to have perforated his caecum. The second operation was to retrieve a ten centimetre knife blade (Figs 3 and 4). Oseophagoscopy was performed on three occasions for removal of objects and once to assess the mucosal status after he had ingested ammonium liquid. Three small nails were removed from his lung by bronchoscopy on another occasion. Admissions for overdosage occurred on 19 occasions: these mostly followed the ingestion of household cleaning liquids but also included drug overdosage with antihistamines and hypnotics.

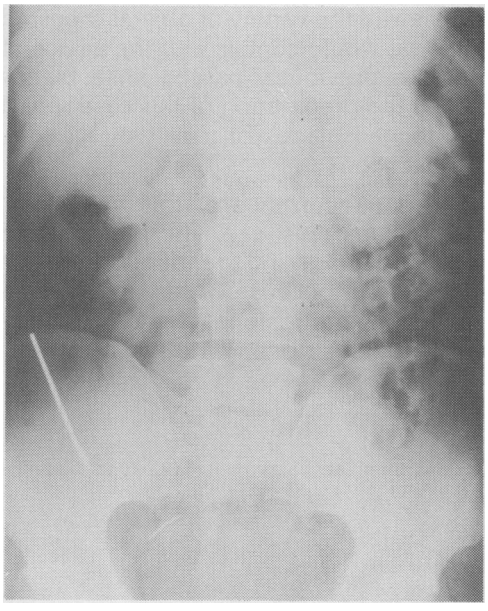

Fig 3. Cocktail fork which perforated the caecum.

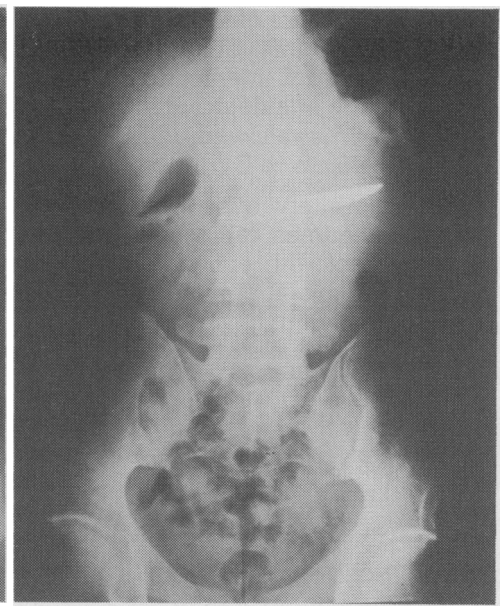

Fig 4. Knife blade in the stomach.

DISCUSSION

This patient has a personality disorder characterised by dependence, attention-seeking behaviour, and poor tolerance for stress in addition to low intelligence. The association between personality disorder, ingesting foreign objects and self-mutilation was first described by Carp.[1] Although this case has many similarities to Munchausen's syndrome, in that the patient presents himself frequently at hospitals, closer analysis reveals fundamental differences. Baron von Munchausen (1720–1797) was a retired German soldier who became well known as a raconteur of extraordinary tales about his life. It was this feature of "story-telling" which led Asher to ascribe the name to a group of patients who frequently presented at hospital with fictitious or simulated symptoms of illness.[2] Typical examples of such symptoms include haematuria, haematemesis and pyrexia of unknown origin, most of which usually require extensive investigations. When confronted, the patients often discharge themselves only to present at another hospital. Our patient's behaviour on the other hand, is impulsive and occurs during emotional crisis. He presents himself at hospital giving a true and accurate account of what he has done, and has no qualms about repeated presentations to the same hospital.

Conservative management is recommended for foreign body ingestion as more than 80 per cent of those reaching the stomach will pass through the bowel without complications.[1,3] Endoscopic removal is recommended for foreign bodies above the cricopharyngeal sphincter and in the upper oesophagus.[3,4,5] Previous abdominal surgery may predispose to impaction and perforation because of kinks in the bowel secondary to adhesions,[4] and in these cases endoscopic removal should be considered while the foreign object remains in the upper gastrointestinal tract. Surgery is indicated if definite signs of complications such as perforation, obstruction or haemorrhage occur or appear imminent in view of

the shape and size of the objects.[5] Having reviewed the variety of objects ingested and passed by this young man without harm we would concur with the advice of minimal surgical intervention. The patient has previously passed knife blades similar to the one depicted in Fig 4, and since the preparation of this case report he has even swallowed and passed the detached blades of a pair of scissors, measuring 8 cm × 3 cm each, without complications.

Psychiatric intervention does not often prevent recurrence of this behaviour, which sometimes can recur years after a previous episode.[6] The main aim of management with our patient has been to encourage independence from his family, but this approach has not been successful because of his own lack of commitment and the internal family dynamics. It is probable that he will continue to pose a surgical and psychiatric problem as well as running the risk of serious injury to himself.

We acknowledge the assistance of the photographic department at Craigavon Area Hospital, and Miss L Ross for typing the manuscript.

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Case report

Idiopathic acute fatty liver of pregnancy: three cases including a subsequent normal pregnancy

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Acute fatty liver of pregnancy is a rare medical complication of late pregnancy and is associated with a high rate of maternal and fetal mortality. Prompt accurate diagnosis and institution of appropriate management should improve the prognosis. We report three cases and outline practical guidelines for diagnosis and management.

CASE 1

A 39-year-old patient in her eleventh pregnancy was admitted to the obstetric unit of another hospital at 34 weeks gestation with nausea, vomiting and malaise for two weeks, and jaundice for two days before admission. Although there was no previous history of pre-eclampsia, hypertension (170/100 mmHg) and mild oedema had been noted at 30 weeks for which she was treated with labetolol, chlorthalidone and diazepam. The day following her admission she became confused and unresponsive. Intrauterine death occurred and she was transferred to this hospital.

On arrival she was in circulatory collapse and made only unco-ordinated responses to painful stimuli. She was icteric but had no other stigmata of liver disease. She was profoundly hypoglycaemic, and following 50 ml of 50% dextrose intravenously her level of consciousness improved so that she mumbled responses to questions but remained drowsy (grade 3 hepatic encephalopathy). Normoglycaemia was maintained by continuous infusion of dextrose and the blood pressure was maintained by a dobutamine infusion. Initial investigations are shown in the Table. An electroencephalogram showed 2–3 Hz slow wave and triphasic activity consistent with hepatic encephalopathy. Examination of peripheral blood revealed giant platelets and normoblasts. A computerised tomogram (CT) showed diffuse abnormality of the liver with reduced attenuation over the liver of 19 Hounsfield units (normal 50–70), indicating fat infiltration. Eighteen hours after admission spontaneous labour began and progressed to a normal delivery of a stillborn male infant. Following delivery she rapidly improved and within an hour was able to answer rationally. Liver biopsy on the tenth

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TABLE

Laboratory results in three cases of acute fatty liver of pregnancy (normal range in brackets)

	Case 1	*Case 2*	*Case 3*
Serum values:			
Bilirubin umol/l (3–18)	140	185	224
Aspartate transaminase U/l (10–40)	357	139	373
Alanine transaminase U/l (10–45)	435	108	405
Alkaline phosphatase U/l (35–120)	681	375	533
Gamma glutamyl transferase U/l (7–46)	89	47	109
Urate mmol/l (0·15–0·50)	0·76	0·54	0·68
Ammonia U/l (13–52)	53	64	24
Plasma glucose mmol/l	0·2	0·7	4·0
Prothrombin time — seconds (15–19)	19	36	40
White cell count 10^3/ul (411)	18·4	14·4	28
Platelet count 10^3/ul (150–400)	250	60	98
Coagulopathy (Disseminated intravascular coagulation)	No	Yes	Yes

Serology for hepatitis A and B and autoantibody screens were negative in all cases.

postpartum day was consistent with resolving acute fatty liver of pregnancy with moderate accumulations of fat within hepatocytes, marked cholestasis and small foci of liver cell necrosis and regeneration. Serial CT scans showed progressive normalisation of the liver attenuation value which had risen to 48 Hounsfield units twenty days after delivery. She was discharged three weeks after admission and remains well.

CASE 2

A 25-year-old primagravida was admitted to the obstetric unit of another hospital at 38+4 weeks gestation. The first half of her pregnancy was complicated by severe hyperemesis and she had remained in hospital until 20 weeks, when the vomiting settled. On admission she was taking chlorpromazine 25 mg daily. Five days prior to admission vomiting had recurred and she had become jaundiced but had no abdominal pain or itch and was fully alert. The next day spontaneous labour began but emergency caesarean section was required for fetal distress. A live, heavily meconium-stained female infant was delivered: she required artificial ventilation for a period but then thrived. Postoperatively, the mother became drowsy and then unresponsive and was found to have a blood sugar of 0·7 mmol/l. Her level of consciousness responded to infusion of dextrose. There was bleeding from the surgical wound and disseminated intravascular coagulation was confirmed.

On transfer to this hospital, she was drowsy (grade 3 hepatic encephalopathy) and very icteric with a liver flap and mild foetor hepaticus. She was treated with fresh frozen plasma and cryoprecipitate along with a dextrose infusion. A CT scan showed diminished attenuation over the liver of 38 Hounsfield units (normal 50–70), consistent with fatty change. A transjugular liver biopsy performed five

days postpartum showed centrizonal fat accumulation which was mostly micro-vesicular, some focal cholestasis but no widespread necrosis or inflammation, the appearances being consistent with acute fatty liver of pregnancy. The bilirubin eventually peaked at 300umol/l fourteen days postpartum and all liver function tests subsequently returned to normal. She made an uneventful recovery and has remained well.

CASE 3

A 26-year-old primigravida was admitted to the obstetric unit of another hospital at 34 weeks gestation with a three week history of fatigue, nausea, vomiting and headaches. She was found to have hypertension, proteinuria and oedema, and was treated for pre-eclampsia with bed rest, hydralazine and diazepam. Her condition deteriorated and she became jaundiced. Six days after admission intrauterine death was diagnosed and she was transferred to this hospital.

She was deeply icteric but not initially encephalopathic and there were no other stigmata of liver disease. Coagulation screen indicated disseminated intravascular coagulation. CT scanning showed decreased liver attenuation of 29 Hounsfield units (normal 50–70) consistent with fatty infiltration. Labour was induced and a small-for-dates male stillbirth was delivered. Within hours of delivery the patient became drowsy and confused (grade 3 hepatic encephalopathy) and a liver flap was present. She was oliguric with a low urinary sodium consistent with functional renal failure of liver disease. She was treated with a continuous dextrose infusion, neomycin and lactulose via a nasogastric tube and four units of blood and fresh frozen plasma for postpartum haemorrhage associated with coagulopathy. She steadily improved. Percutaneous liver biopsy performed on the sixth postpartum day showed cholestasis and widespread microvesicular fatty change typical of acute fatty liver of pregnancy. She was discharged three weeks after admission but the serum bilirubin did not return to normal until nine months later. Two years later she again became pregnant. The pregnancy was without complication and her liver function tests were monitored at regular intervals and remained normal. Labour was induced at term and she had a normal delivery of a healthy baby girl. A CT scan on the day after delivery showed normal liver attenuation values.

DISCUSSION

Since first described in 1857 about one hundred cases of acute fatty liver of pregnancy have been reported.[1] The incidence of reported cases has risen in the last decade, probably as a result of improved antenatal surveillance, greater awareness of the diagnosis, and recognition that it is not an invariably fatal disease.[2] It is more common in twin and in male pregnancies. Historically, both maternal and fetal mortality were very high in this condition. In a series of twelve cases from the Royal Free Hospital in 1982 maternal mortality was 33% and fetal mortality was 67%.[3]

Although still a rare disease, it must always be considered in the differential diagnosis of jaundice or impaired level of consciousness in late pregnancy. Further improvement in prognosis depends on prompt accurate diagnosis and decisive management. In acute fatty liver of pregnancy there is usually a history of a prodromal malaise followed by rapid onset of nausea, vomiting and jaundice. Diminished level of consciousness can occur due to hepatic failure or hypoglycaemia when there is only slight jaundice. Prolongation of the prothrombin time in these circumstances indicates severe liver disease. Whilst abdominal pain

may occur, cholelithiasis can be excluded by ultrasound scan. Viral hepatitis can present similarly but the serum transaminases are usually very high in this condition compared to the nonspecific or cholestatic pattern seen in acute fatty liver of pregnancy. Cholestasis of pregnancy is characterised by itch and does not cause hepatic failure. Preceding pre-eclampsia is common (as in Cases 1 and 3), and it may be difficult to distinguish the liver disease of severe pre-eclampsia from acute fatty liver. Computerised tomography appears to provide a rapid non-invasive means of establishing the diagnosis and in all of our patients CT scans on the day of admission strongly indicated fatty infiltration of the liver. Use of CT scanning in Case 1 has previously been reported elsewhere.[4] Neutrophilia, thrombocytopenia and abnormalities of the peripheral blood film including giant platelets, normoblasts and basophilic stippling are useful diagnostic indicators in acute fatty liver of pregnancy. Elevation of serum uric acid is usual.

The first principle of management should be to stabilise the patient and aim for delivery as soon as possible.[5] Secondly, since hypoglycaemia is typical and may contribute to intrauterine fetal death, careful attention should be paid to maintaining normoglycaemia and large volumes of 10% or 50% intravenous dextrose may be required. As in Cases 2 and 3, bleeding may occur in association with disseminated intravascular coagulation but delivery should not be delayed on this account. However, bleeding should be treated in the usual way with fresh frozen plasma and platelets in addition to blood transfusion. Fresh frozen plasma and platelets should also be used to cover delivery where coagulopathy is present. Neonatal hypoglycaemia should be anticipated and promptly treated.

The aetiology of acute fatty liver of pregnancy remains unknown. It is one of a group of conditions with similar histological appearances of microvesicular fat accumulation within hepatocytes. These include Reye's syndrome in children, Jamaican vomiting sickness which is associated with a toxin in ackee fruit, and the fatty liver disease occasionally precipitated by sodium valproate and high doses of tetracyclines. Affected patients may have an underlying defect in fatty acid metabolising enzymes, and viral, toxic or nutritional factors may initiate the fatty change in susceptible patients.[1]

In Case 3, pregnancy complicated by acute fatty liver resulting in stillbirth was followed by an uneventful pregnancy and normal delivery of a healthy baby. Burroughs et al reported seven subsequent normal pregnancies in four of their twelve patients.[3] It appears that subsequent pregnancies carry no added risk of recurrence. Both liver function and histology rapidly return to normal in survivors.

We wish to thank the obstetricians who referred patients and gave us access to their records and Dr JM Sloan for reviewing the liver histology of the three patients.

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Case report

Pseudo-tetanus following trifluoperazine

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The risk of mistaking a dystonic reaction following trifluoperazine for tetanus is well recognised.[1,2] In 1979, Stoddart[3] reported six cases of pseudo-tetanus, "a syndrome which is produced by a variety of non-clostridial factors". Trifluoperazine was implicated in one of these cases. We report a similar instance in which circumstantial evidence supported such a misdiagnosis.

CASE HISTORY

A 22-year-old man presented to the casualty department with "locking" of his right great toe in dorsiflexion, and of his jaw. One week previously, he had complained of nausea and a sore throat for which his general practitioner had prescribed a course of oral penicillin. He also suffered from intermittent pain arising from a wisdom tooth, and he had been involved in a fight four days previously in which he had sustained an abrasion to his right foot and a human bite to his right hand. He had received no anti-tetanus immunisation for more than ten years. It was felt that tetanus (albeit atypical or local) might account for these muscular spasms at presentation. Tetanus immune globulin (250iu) was administered intramuscularly and benzylpenicillin (600mg six hourly) intravenously. He was transferred to the intensive care unit and sedated using midazolam (2·5mg intravenously) and chlorpromazine (25mg intramuscularly). Within eight hours all muscular spasm had disappeared. Only then did he volunteer that he had taken six doses of trifluoperazine 5mg during the previous 36 hours, which had been prescribed by his general practitioner for nausea. There was no recurrence of his symptoms and he was discharged from hospital two days later.

COMMENT

Acute dystonic reactions are usually characterised by sudden intermittent episodes of uncontrolled movement of the head and upper body. Trismus is less common but well recognised. Trifluoperazine is a phenothiazine derivative with a piperazine side chain, a group of drugs well known to produce such reactions.[4] In this case several features were worthy of note. The history of a recent abrasion in the absence of tetanus immunisation supported the misdiagnosis of atypical

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tetanus. Children and young adults are most susceptible to drug induced acute dystonia; the higher risk may be explained by an age related fall in the number of dopamine 2 (D2) receptors in the substantia gelatinosa.[5] Thirdly, and typically, the reaction took place within 36 hours of taking the first dose of the responsible drug.[6] Midazolam and chlorpromazine were used as sedatives, and symptoms resolved within eight hours. Benztropine is recommended as the initial pharmacological treatment of such reactions, but Stoddart[3] pointed out that diazepam may be a satisfactory alternative. A non-specific benzodiazepine effect (of midazolam) may have been beneficial in this case. In view of its antidopaminergic properties chlorpromazine must be contraindicated in such circumstances, and it has been implicated as a precipitator of a tetanus-like syndrome. Chlorpromazine has been used in the treatment of true tetanus but it is not ideal in those with autonomic manifestations of the disease.[7] The potential danger of misdiagnosing a drug induced acute dystonia as tetanus exists, but a complete drug history and an awareness of the danger should prevent such an error.

We thank Dr F A O'Connor, Consultant Physician, Altnagelvin Area Hospital, for permission to report this case history.

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Case report

Oral Crohn's disease: the distinction from the Melkersson-Rosenthal syndrome

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Orofacial swelling, particularly of the lips, tongue, buccal mucosa and gingiva is a feature of the localised granulomatous condition known as the Melkersson-Rosenthal syndrome,[1] but may also represent oral manifestations of chronic inflammatory conditions, including Crohn's disease[2] or sarcoidosis.[3] Oral manifestations of these systemic conditions may precede involvement in other areas by several years[4] and clinical, biochemical or even histological differentiation is often extremely difficult. Considerable debate exists in the general surgical and dental literature regarding the extent to which a patient presenting with granulomatous inflammation of the orofacial region should be investigated for evidence of chronic granulomatous disease elsewhere. This case demonstrates the diagnostic difficulties, and illustrates that Crohn's disease may present in the mouth before it becomes symptomatic in other parts of the gastrointestinal tract.

CASE REPORT. A six-year-old girl presented with a four year history of recurrent episodes of non-tender swelling of the upper lip, and angular chelitis. The swelling usually lasted from a few days to a few weeks, but during the previous six months exacerbations had become more frequent and persistent. Otherwise, her mother had not noticed any specific problems. In view of the long history and lack of other symptoms, this was thought to be an example of the Melkersson-Rosenthal syndrome although the possibility of an oral presentation of Crohn's disease was also considered. The condition remained quiescent over the next few months until she re-attended with a flare-up of her oral lesions and episodes of severe crampy abdominal pain and constipation. The swelling of the lower lip had increased with marked thickening of the buccal and gingival mucosa, and associated deep fissures and ulcers producing a "cobblestone" appearance. The perianal area was inflamed and excoriated, with fissuring, ulceration and skin tags. Her weight was below the fifth centile. Haemoglobin was 9·8 g/dl, ESR 36 mm/hr and serum iron 6·1 umol/l (normal 13 – 30 umol/l). Serum vitamin B_{12} and folate levels were normal

Biopsy of both the oral and perianal lesions was performed. Histology of the oral lesions revealed a diffuse infiltrate of chronic inflammatory cells but it was not

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possible on the basis of this alone to make a definitive diagnosis. Histology of the perianal lesions revealed the presence of non-caseating granulomata with lymphocytes, histiocytes and multi-nucleated giant cells, confirming the diagnosis of Crohn's disease. Treatment with sulphasalazine and metronidazole was commenced, but progress was initially slow and complicated by the development of two perianal abscesses which required drainage. Over the next few months her general condition gradually improved, weight increased to above the tenth centile, the thickening of her lip and oral mucosa decreased and the inflammation of her perianal area gradually settled. Haemoglobin, ESR and serum iron returned to normal. She remained well at her last review, three years after Crohn's disease was diagnosed.

COMMENT

The Melkersson-Rosenthal syndrome is a rare chronic granulomatous condition of unknown aetiology. The full clinical picture consists of swelling of both lips, a fissured tongue (*lingua plicata*), and facial paresis, which may be unilateral or bilateral. These features may appear together or at intervals, and the clinical course is often episodic, with exacerbations or symptom-free periods lasting from months to years. Swelling of the lips occurs in all patients,[1] but *lingua plicata* and facial paresis occur in only about 30% of cases.[5] Oligosymptomatic cases, in which all three features are not present include variants such as *cheilitis granulomatosa* (orofacial swelling only[6]), oral swelling with facial nerve paresis[7] and oral swelling with *lingua plicata*.[8] Rarely, the condition may be familial.[9] In addition to the three main features, some patients show other features such as gingival hypertrophy, attacks of migraine, herpes simplex infection, decreased salivation and lacrimation, and involvement of the chin, nose, eyelids and forehead.[8]

Oral lesions of Crohn's disease are characterised by induration and swelling of the lips, hyperplastic folds and fissures of the buccal and gingival mucosa, with aphthous-like ulceration, producing a "cobblestone" appearance.[10] These may occur at any time during the course of the disease and isolated Crohn's disease of the mouth may be diagnosed and treated without evidence of other gastro-intestinal involvement.[11] In typical cases of either condition differentiation on clinical grounds can be made with reasonable accuracy. The most useful different-iating factors are the characteristic hyperplastic folds, fissures and mucosal ulceration of Crohn's disease, and the presence of *lingua plicata* or facial paresis, which points towards the Melkersson-Rosenthal syndrome. The difficulty in diagnosis arises when Crohn's disease presents without intestinal involvement, as in this case, or in oligosymptomatic forms of the Melkersson-Rosenthal syndrome, perhaps with some of the additional features described above. In such situations, clinical differentiation is impossible and further investigation necessary.

The extent to which investigation, especially invasive procedures, should be undertaken is not universally agreed. It is generally accepted that there is no effective therapy for the Melkersson-Rosenthal syndrome,[8] although there are isolated reports of improvement following intra-mucosal steroid injection.[7] In contrast, local steroid therapy is often effective in oral Crohn's disease[12, 13] and if these patients are carefully observed for evidence of other gastrointestinal involvement, therapy with sulphasalazine or steroids may arrest the disease at an early stage and prevent more serious deterioration. For this reason, several

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authors recommend an aggressive approach including nutritional assessment, biopsy, contrast radiography and endoscopy, to confirm the diagnosis in chronic granulomatous conditions of the mouth and to detect coincidental gastrointestinal involvement in cases of Crohn's disease.[4, 14] This case, where clinical assessment was equivocal, confirms the traditional difficulties in distinguishing between the two conditions. The evidence of malnutrition encouraged us to recommend careful follow up and nutritional assessment. We feel it is reasonable to reserve contrast radiology, endoscopy and biopsy for cases where specific symptoms or nutritional assessment suggest that they are indicated. Careful clinical examination, particularly of the perianal area, is necessary in all patients with chronic orofacial swelling.

We acknowledge the assistance of the Departments of Pathology of Altnagelvin Hospital, Londonderry and The Royal Victoria Hospital, Belfast.

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