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THE ULSTER MEDICAL JOURNAL

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The Ulster Medical Journal, Volume 67, No. 1, May 1998.

The Ulster Medical Journal

The Journal of the Ulster Medical Society. First published in 1932.
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The Ulster Medical Society was founded in 1862
by the amalgamation of the Belfast Medical Society (founded 1806)
and the Belfast Clinical and Pathological Society (founded 1853)

THE ULSTER MEDICAL JOURNAL

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THE ULSTER MEDICAL SOCIETY

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The Ulster Medical Journal, Volume 67, No. 1, May 1998.

Editorial

Membership Matters

This editorial seeks to answer a few simple questions and scotch some misconceptions about the Ulster Medical Society. It offers an invitation to those who have never joined the Society to consider membership. At the same time occasional attenders are reminded that the vitality of any society is best maintained by the regular interest of all its members. In turn it seeks your comments.

What is it? – The history of the Ulster Medical Society makes for illuminating reading and I commend it to you.[1, 2] It was formed in 1862 through the amalgamation of the Belfast Medical Society (founded in 1806 and revived in 1822) and the Belfast Clinical and Pathological Society. A spirit for mutual improvement in their common profession by the contribution of all to a common purpose was to be the guiding principle. Key words to describe its original ethos would seem to include education, information, unity and friendship.

Who's in it? – While membership of any society may not necessarily reflect its current state of health it does however provide some indication of wellbeing and direction. Details of membership by speciality for 1997 are given in the Table. Pooling data from other sources it can be calculated that only around a quarter of all qualified doctors in the province (Hospital Doctors, GPs, practising and retired) are members of the Society and only a tenth of these (2.5% of all doctors) attend meetings regularly. Some specialities are poorly represented.

What's on offer? – In a world of increasing sub-specialisation, the Ulster Medical Society aims to offer general education to all. Recent programmes have both educated and entertained us on such diverse subjects as Coping with Babies and Toddlers, Myalgic Encephalitis, Brain Death, Limb Lengthening Procedures, Hormone Replacement Therapy and an Ulsterman's View of the House of Lords each delivered by experts in their fields. Another exciting and broadly based programme is planned for the forthcoming session (1998/99) and is to include topics such as Pharmacoenonomics, Obesity and Ageing. Those gasping for CME points are also catered for. Special events during the year include the Annual Clinical Pathological Conference and the Annual Presidential Dinner. The golfing fraternity can be relied on to enter for the Victory Perpetual Challenge Cup (but are not necessarily the best attenders at meetings!). The Journal comes with Membership and has provided an outlet for many aspiring to be authors.

TABLE

Ulster Medical Society Membership 1997

	Working Northern Ireland	*Retired*	*GB/ Overseas*
General Practice	180	51	22
Hospital Practice			
Physicians	168	32	15
Surgeons	77	32	6
A/E	5	2	
Psychiatry	25	8	2
ENT	13	1	1
Ophthalmology	10	3	2
Paediatrics	20	5	3
Laboratory (etc)	50	12	4
Anaesthetics	32	8	1
Radiology	14	2	1
Obstetrics/Gynaecology	35	8	5
Community	35	4	4
Other	52	7	16
	716	175	82

Total 973

Where and when does it meet? – The programme begins with the Annual Presidential Address in early October. Subsequently some ten or eleven meetings are held between October and March on alternate Thursday evenings usually at 8.30 pm in the Ulster Medical Society Rooms, Whitla Medical Building. This year a very successful autumn meeting was held in Londonderry at the

invitation of the Altnagelvin Hospital medical staff and such a venture may well be repeated in the future. If you would like the Society to hold a meeting at a venue near you why not write to the Secretary?

How do I join? – Membership is by proposal and seconder submitted to the Secretary.

The best way to promote the society is by word of mouth. When was the last time you invited a colleague to consider membership and happened to have an application form to hand? You will find one at the back of the Journal – if completed by each of us this would double membership overnight. But perhaps of equal importance, why not come and join us on alternate Thursday evenings, broaden your knowledge and make new acquaintances? As we move towards the next millennium the future health of the Ulster Medical Society rests in your hands. Your membership matters.

DAVID R. McCANCE
Honorary Secretary
Ulster Medical Society.

REFERENCES

1. Strain R W M. The history of the Ulster Medical Society. *Ulster Medical Journal* 1967; **36**: 1.
2. Montgomery D A D. The Ulster Medical Society, Quo Vadis? *Ulster Medical Journal* 1976; **45**: 1.

The Ulster Medical Journal, Volume 67, No. 1, pp. 3-12, May 1998.

Your life in your hands

A Surgeon's view of Hand Function – Art expression and form

J W Calderwood

Presidential Address, FRCS Ulster Medical Society, 16 October 1997

Just as the eye is the window of the Heart, the hand is the Mirror of the Man. ("Behold the Window of my Heart, Mine Eye" – Shakespeare – Love's Labour's Lost)

My interest in Hand Surgery was initially encouraged by Professor R. I. Wilson, a man who had an immense influence on orthopaedics and a father figure to many of the orthopaedic surgeons. It was further stimulated when I rotated through the plastic surgery unit in the Ulster Hospital, Dundonald, and I worked with Mr. John Colville and learned very much from his wisdom and skills.

In 1974 I took my family to Paris and commenced work with Professor Tubiana at l'Institut de la Main. I met many of the great personalities in hand surgery who tended to visit Paris as an attractive stop on their visits to Europe. I later visited Harold Kleinert in Louisville, Kentucky, for a short period.

This paper will emphasise the importance and significance of the hand in all aspects of our lives and the great influence of its function in our lives. Considering the relative importance of hand function in everyday life, I think that Michelangelo put it in perspective in its central role in "Creation" (Fig. 1). More recently a less attractive art work portrays Penfield's Homunculus, (Fig. 2), and brings back for many of us memories of pre-clinical days. It shows the large area of representation of the hand in the brain, both motor and sensory, in relation to other body parts and in so doing indicates the relative importance of the hand in our lives.

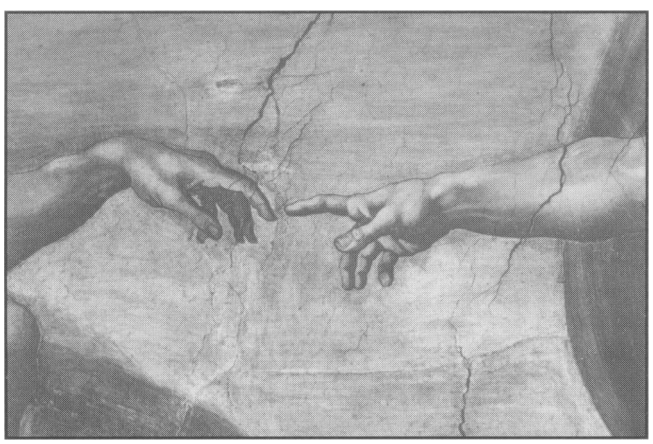

Fig 1. Creation of Man – (Detail) Michelangelo.

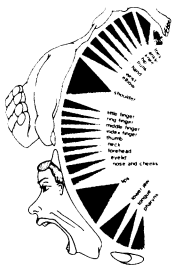

Fig 2. Penfield's Homunculus

Returning to beginnings, the hand has been represented among the earliest known forms of art. The prehistoric picture of the hand as illustrated is found in a grotto in Peche Merle in the Dordogne, (Fig. 3). The representation of the

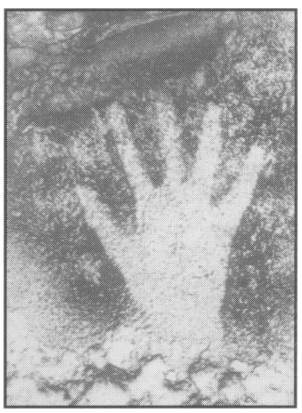

Fig 3. Prehistoric Picture of a Hand found in Peche Merle, Dordogne.

Belfast City Hospital, Lisburn Road, Belfast.
Royal Victoria Hospital, Belfast.
Musgrave Park Hospital, Belfast.

Mr. J W Calderwood, FRCS, Consultant Orthopaedic Surgeon.

Fig 4. Image and Personality shown by the Hand.

hand in art has played a very important role in communicating the image and personality of the subjects, just as our own image and personality are enhanced and emphasised by the expressive use of our hands each day, (Fig. 4).

Occasionally disease or deformity may be portrayed, (Fig. 5), which gives an insight into the condition of the subject but more often the hand is presented as an image of beauty as in a sculpture by Canova, (Fig. 6), or an image of strength as in Michelangelo's David, (Fig. 7).

Fig 5. Deformity of the Hand portrayed in Sculpture.

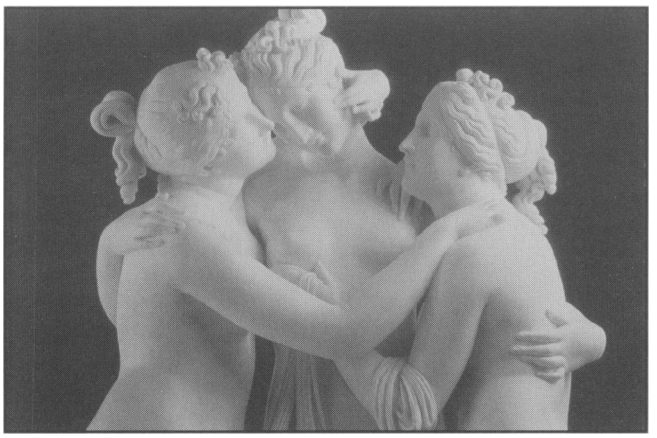

Fig 6. The Three Graces –Canova

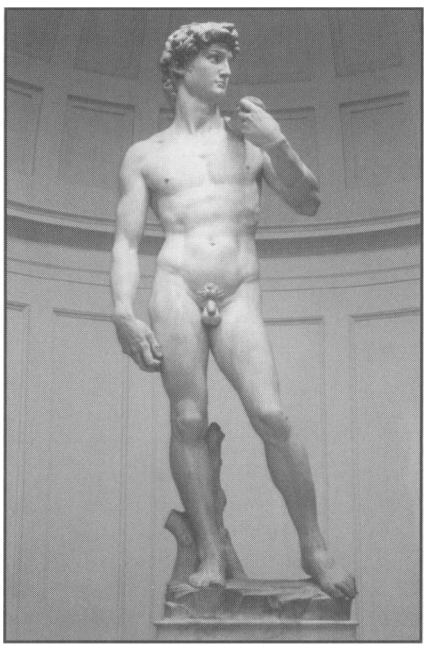

Fig 7 David – Michelangelo

Through the centuries the hand has been portrayed in art to a greater or lesser extent. In Greek and Roman art it was found particularly in sculpture, but it was largely absent from Islamic art. It again gained prominence in the Middle Ages and in the Renaissance. Since then and up to modern times, frequent attention has been given to the form, beauty and particularly the expression of the hand as found for example in The Bronzes of Rodin, (Fig. 8).

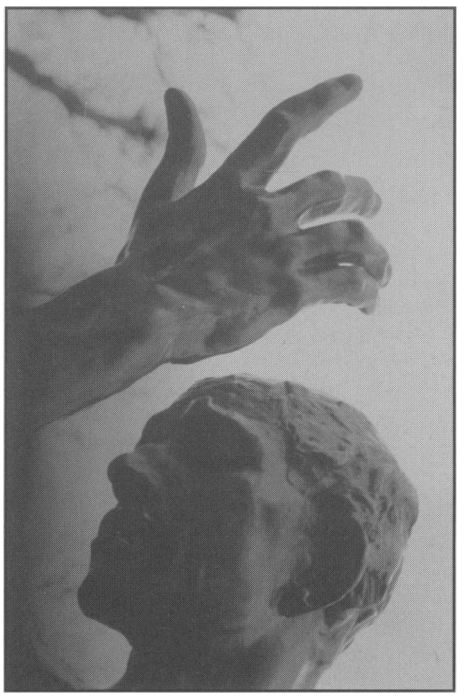

Fig 8. Bronzes of Rodin – An Example

The hand is involved in a major part of our lives – hence its prominence in Penfield's Homunculus. When we consider the miracle of skill which lies within the hand we can only be amazed at our own indifference to its everyday function. Voltaire said of Sir Isaac Newton that "in *spite* of all his knowledge, he did not know how his own hand functioned".

Broadly speaking, function of the hand may be classified as motor and sensory but this provides no depth of knowledge or understanding of the capacity and extent of function of the hand. As surgeons we are most often involved in improving and restoring motor function of the hand by reconstruction as in rheumatoid arthritis, (Fig. 9), although following injury we may indeed have to repair various nerves.

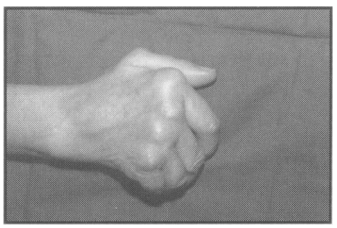

Fig 9.
The Deformity of
Rheumatoid Arthritis.

It is surprising to consider that most of man's physical activity takes place with use of the arms and hands carrying out the actions conceived and desired by the brain. The legs mainly provide locomotion and even then although locomotion may be indeed the conceived and desired action, the arms are often used to supplement the leg action.

The primary function of the upper limb is that of exploration. The hand can sweep round on a radius formed by the arm and this circle overlaps that of the other hand which can also move in a similar fashion. The areas accessible to the hand therefore overlap in the same way as the visual fields overlap. The hand and the brain function together in such a unified fashion that I would question the validity of the term 'manual worker' and suggest that there is no such person. One of the particular assets of the mobility of the arm is that it allows the hand to move outside the fields of vision and explore the environment.

On proceeding to consider the primary motor function of the hand it is recognised that all motor functions require some sensory input.

Basic functions of the hand may be considered as follows:

1. **Eating** – (Fig. 10). The ability to reach out, take food and place it in the mouth is necessary for survival. Such is the recognition of this as a basic function that we describe the minimum possible lifestyle as a "hand to mouth existence". The ability of the upper limb to move and enable the hand to explore space is therefore a very great asset in allowing man to contact food as in reaching and grasping and indeed helping himself.

Fig 10. Eating an Orange – Harold Harvey

2. **Hygiene** – The hand can reach virtually any part of the body, including the perineal region. Personal hygiene is a very significant function and may be a problem when mobility of the upper limbs is limited and the hand cannot be delivered in space by the restricted mobility of the arm due to disease or injury. Care of the hair, face and

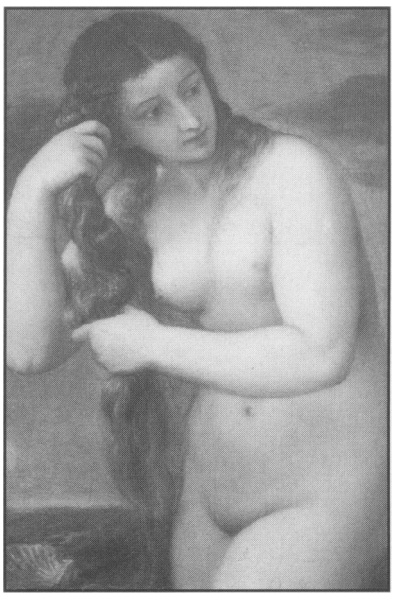

Fig 11. Venus Anadyomene (Detail) – Titian

body may cause difficulty, (Fig. 11). The upper limb surgeon must be aware of this when there is the prospect of arthrodesis of both wrists. Arthrodesis of one wrist is done with slight extension of the wrist in order to allow maximum power grip but if bilateral arthrodesis is required, one wrist must be placed in slight flexion so that it is possible to place the hand in confined spaces, a function not possible if an immobile wrist is in a position of extension.

3. **Work Activity and Recreation** – A study of the anatomy of the hand shows the numerous interdependent structures. This very complex anatomy is capable of the most delicate and intricate activities while it is also capable of carrying out heavy work where power and force are essential, (Fig. 12). It may act as a tool – gripping, turning, twisting, lifting and carrying or even digging, (Fig. 13). The hand of man, however, is unique in that, in addition to its role as a tool, it can itself manufacture a suitable tool to assist or take over a particular task.

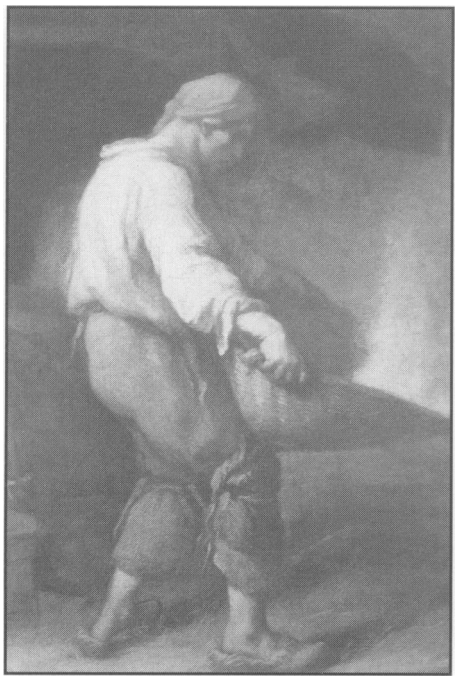

Fig 12. The Winnower – Jean Francois Milet.

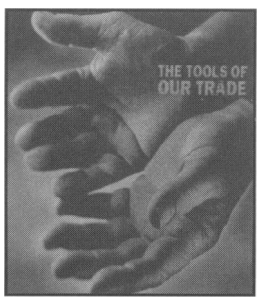

Fig 13.
The Tools of
Our Trade.

The hand may also act in a primitive manner as a weapon of attack or defence. The fist may be clenched and the wrist locked to effectively produce a weapon, (Fig. 14). This most delicate and sophisticated structure may thus be used simply to inflict force as an assailant attacks or it can be used as a mobile and protective instrument in absorbing the effects of an assault and protecting more vital areas, such as the head including the face and brain, or other more vulnerable parts.

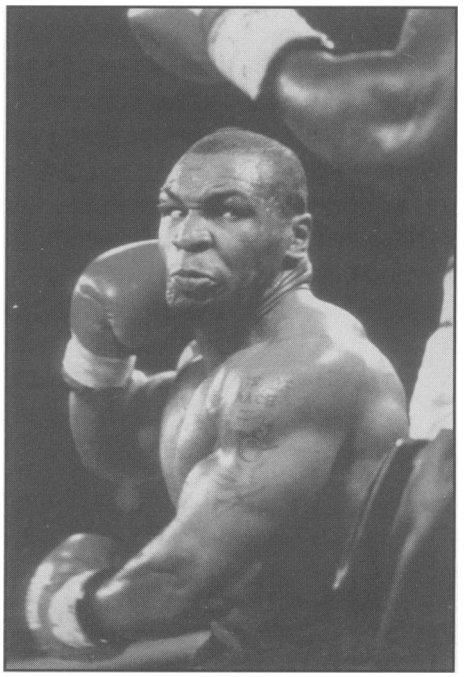

Fig 14. Mike Tyson

The diversity of action of which the hand is capable is truly remarkable, (Fig. 15). Not only do some individuals, to the admiration of all, excel in particular manual skills but even the average person is capable of an immense range of skills. For example, it is conceivable that a concert pianist may have an interest in gardening or the dress designer may enjoy rowing.

Fig 15
Diversity of
Hand Function –
Van Gogh

On a personal level, fracture care frequently involves intramedullary nailing of femur or tibia which demands considerable manual force while on the same operating list I may release a carpal tunnel or repair a digital nerve – not a claim of any exceptional skills on my own behalf but rather an example of the extraordinary range of action of which the hand is capable. Do we know of any machine or mechanism which has such a diversity of function?

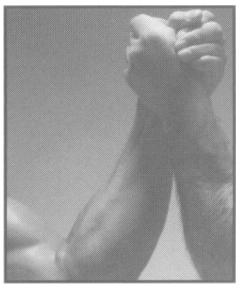

Fig 16.
Grip Strength

It is interesting to realise that the limit of precision in surgery is not manual skill which seems almost unlimited, but rather visualisation. The microscope or loops enable the surgeon to see the structure, and 'if you can see it you can suture it!'

The limits of function may be extended by training, for example, grip strength may be increased by a programme of training, (Fig. 16), but skills may also be increased whether in sport or other activities. Much of training establishes a pattern – do any of the golfers know exactly where the club head is at a particular point during their swing? (Fig. 17). Much skilled action occurs involuntarily. The concept is followed by the action and the involuntary movements occur precisely and sequentially as the well practiced action takes place. The concert pianist practises for long hours so that much of his playing occurs subconsciously. (Fig. 18), even driving a car involves much isubconscious manual activity.

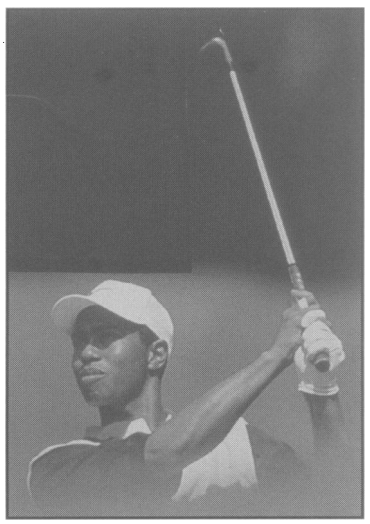

Fig 17. Tiger Woods.

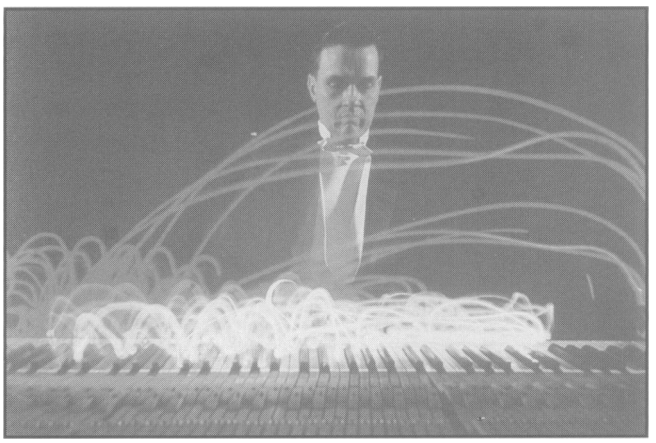

Fig 18. The Concert Pianist.

I have already referred to the role of the upper limb in all activity. All games involve the upper limb – even soccer for example, requires the balancing effect of the arms in executing the football skills. Again various games demand different degrees of power and precision. Racquet and bat games require strength and skill with hand/eye co-ordination while in snooker or darts, precision is the main requirement. Some sports however require a wide range of power and touch. Perhaps this explains the fascination of golf for so many of the medical profession.

4. **Communication** – Sensory function plays a significant part in communication. Movement, touch and expression are forms of communication and it is perhaps this function which is least obvious to us. While expression of the hands is recognisable to all, it is often carried out subconsciously.

In daily communication with one another, only the face and hands are exposed – with the obvious exception of the female legs. Second only to the face, the hands indicate mood and personality. The background of a person, the work activity and much other information may be indicated by

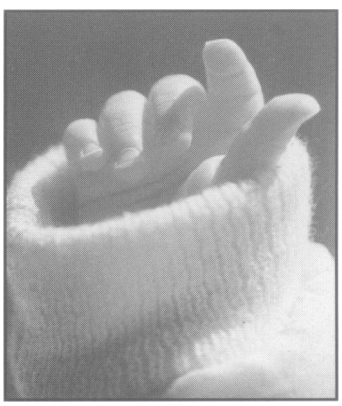

Fig 19. A Child's Hand.

the hands. The hands may be well cared for or may demonstrate a lack of care. They may be deformed or a thing of beauty. Age is reflected in the hands, (Fig. 19), as it is in the face although I am not certain whether or not plastic surgery on the hand can be considered as an adjunctive procedure to a face lift. We communicate with our hands to express nuances and enhance the meaning or our words or emphasise our thoughts. We may express emotions such as despair, (Fig. 20), tension, happiness, grief or tenderness. We may indicate apprehension, (Fig. 21), or triumph.

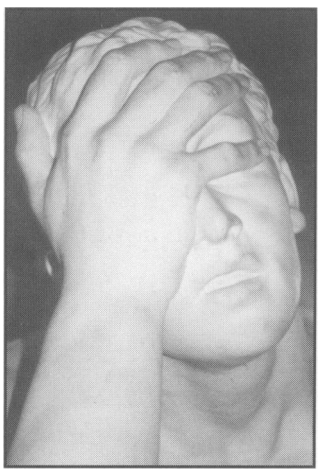

Fig 20. Despair.

Fig 21. Apprehension.

The absence of hands produces a curiously deficient image, (Fig. 22).

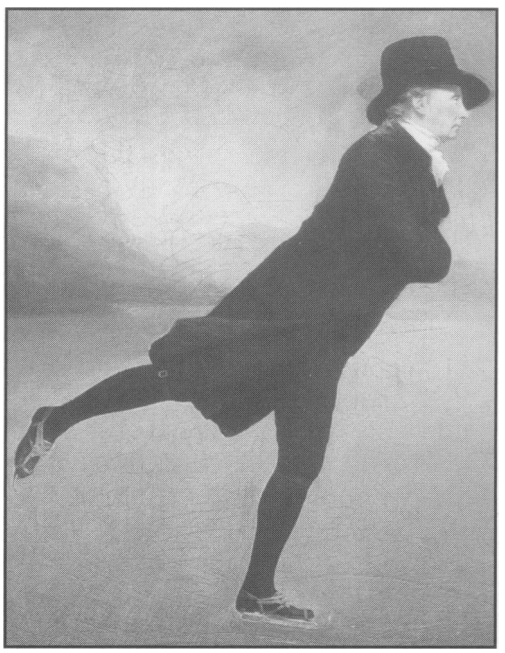

Fig 22. The Reverend Walker Skating – Raeburn.

People in the public eye are conscious of the effect of the hands. In recent history, orators and leaders, (Fig. 23), good and bad, made great use of expression of the hands to underline and emphasise their viewpoints.

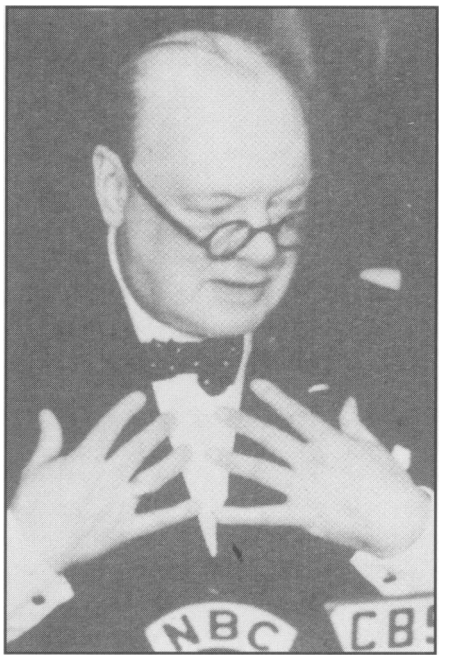

Fig 23. Churchill Emphasises his Words.

In addition to expression of mood or emotion, a touch of the hand may indicate friendship or give reassurance. Love is expressed by the hands and may result in an embrace, (Fig. 24). But the hand is also a potent vehicle of sexual advance and may produce erotic arousal. This function is a good example of the law of physics which states that to every action there is an equal and opposite

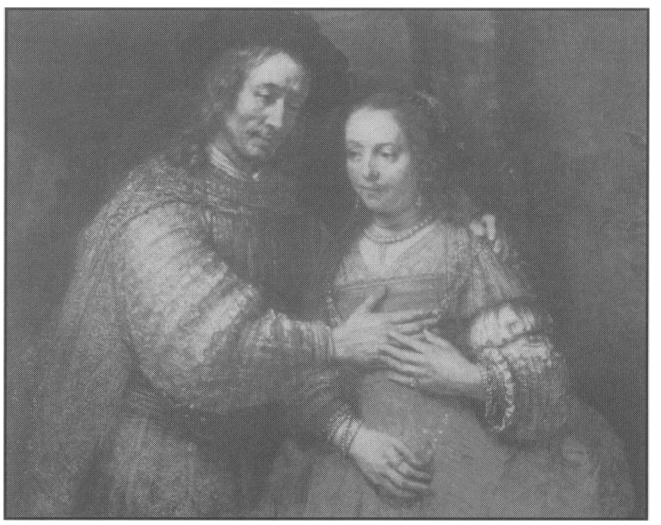

Fig 24. The Jewish Bride –Rembrandt.

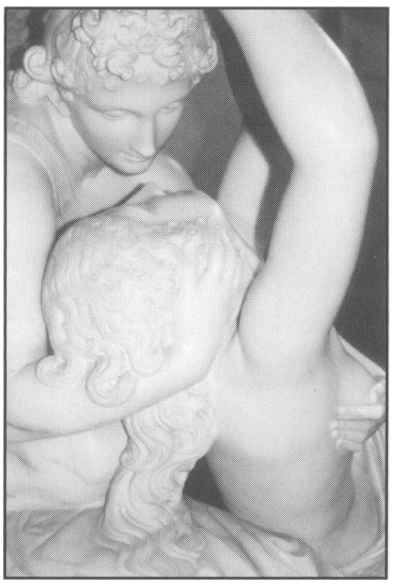

Fig 25. Sexual Arousal – Canova.

reaction. Not only may the hand act as a vehicle of sexual arousal but it, by its own sensory response, acts to arouse the initiator of the advance, (Fig. 25).

Moberg, who was a Swedish Hand Surgeon, brought to our awareness the sexual problems associated with loss of hand function. As an instrument of touch and caress, flexibility of the hand is all important. Where disease or injury has affected the hand, he found that in some instances the patient preferred the retention of flexibility even though an arthrodesis might have increased the power pinch and grip.

By the use of sign language and Braille, the hand does of course achieve its greatest place in communication. Those who cannot hear can communicate, through Braille those who cannot see can communicate. The use of the hand in communication therefore opens a whole further dimension for those with these disabilities. This skill can surpass the recognition of the written word as the deaf and blind can even enjoy music.

Primary Sensory Function of the Hand

In some respects the hand acts as a third eye – indeed it has the added ability to see around corners which is not possible with the eye, so that by feel alone an object may be clearly recognised and an exact image may be visualised even though it is out of sight. As a sensory organ, the hand has the great advantage of mobility. It can reach towards the object of interest, explore and feel it to acquire more information with regard to surface texture, temperature, compliance, density and weight. The proprioceptive aspect of sensation is very important as actions are initiated or carried out without having to localise the position of the hands. In any case, as I have commented earlier the hands are not always within the fields of vision. It seems remarkable that without consciously thinking, we always know the position of our hands. It would be difficult for us to achieve the Biblical exhortation 'let not thy left hand know what thy right hand doeth' – Matthew ch.6 v. 3.

The sensory function of the hand therefore is essential in the recognition of our environment. Initially we recognise by sight but the appearance only becomes reality as a result of touch and feel.

Within medicine the sensory function of the hand perhaps reaches its greatest when diagnosis by examination is carried out. The visual information of inspection is followed by palpation, percussion and auscultation, all of which are manual but mainly sensory skills. Much of diagnosis is therefore made by manual skills, (Fig. 26). The hand is not only inexorably involved in diagnosis, but the patient's hand may itself be a fertile source of information. Various abnormalities may be readily seen in the hand and may give an indication or local or generalised disease, for example, Dupuytren's contracture may be obvious, while finger clubbing may possibly indicate respiratory disease.

I recently became aware that in addition to clawing of the ring and fifth fingers and associated sensory deficiencies in ulnar nerve lesions, the more subtle deficiencies of ulnar nerve function in partial lesions may be detected by demonstrating what I describe as the "Flick Test". The normal

Fig 26. Science and Charity – Picasso.

ability to flick the index or middle fingers against the opposite palm and produce an audible click is lost due to weakness of the interosseous muscles so that a sound cannot be produced. One hand may be compared with the other, (Fig. 27).

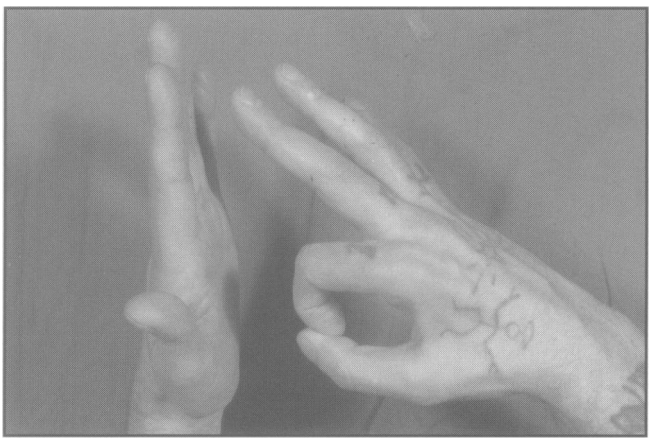

Fig 27. The Flick Test – Ulnar Nerve Lesion.

For the same reason, the middle finger may not be able to cross over the index finger – the Sign of Hope, (Fig. 28).

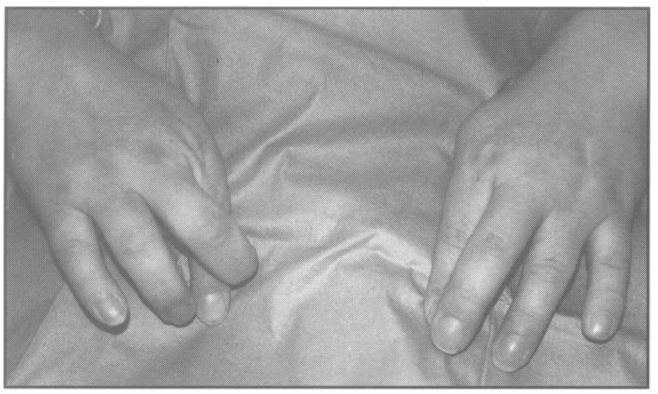

Fig 28. Sign of Hope – Ulnar Nerve Lesion.

Having considered the motor and sensory function of the hand, it is obvious that disease, deformity and injury may cause very serious disability with loss of sensation and loss of mobility. Injury in particular may affect all aspects of hand function, both motor and sensory. Even a relatively small loss of movement may cause a significant reduction in normal dexterity of the hand. In Dupuytren's contracture, (Fig. 29), the affected finger may prevent effective grasp and often the patient may find that he or she may catch the finger when washing the face or have difficulty placing the hand in the pocket. A person with a hand deformity is of course at risk when working with machinery because of the loss of normal

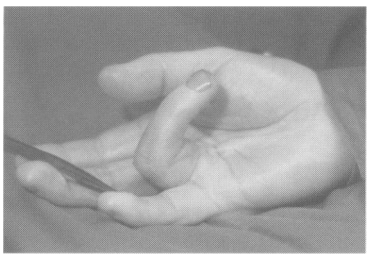

Fig 29. Dupuytren's Contracture.

dexterity. The lack of the involuntary response of the hand which is normally the outcome of an intended action can be dangerous as it exposes a digit or digits to danger. Loss of joint mobility may prevent the hand from carrying out skilled activities and in some cases a stiff finger may be an impediment to hand function so that occasionally removal of a digit may improve function.

On considering amputation, I personally am very conservative and often my first reaction when faced with the possibility of this procedure is to stop and think again. Perhaps the influence of Mrs. X has a part to play in this reaction.

I met her at the Royal Victoria Hospital after she had presented with a crush injury to the ring fingertip. She had an extremely sensitive fingertip having lost a centimetre of length. She prevailed on me to explore and excise the neuroma. This lady had such sensitivity of the finger that she shuddered and tensed even when I sat down opposite to her. The vibrations of my entry were enough to make her withdraw the hand indicating a remarkable degree of sensitivity. With more good intention than hope, the neuroma was excised but the sensitivity persisted. After many reviews she persuaded me to amputate the terminal phalanx. This again was a complete failure. Months passed and I became very familiar with her at review. I even went as far as dividing both digital nerves at the base of the finger which in fact resulted in a numb finger but without relief of the sensitivity. Her family contacted me and I spoke to her daughter with some foreboding but in fact the interview was very pleasant and she simply requested me to refer her mother privately to a London Surgeon for a second opinion. I referred her to Mr Rolfe Birch in Harley Street who is now the acknowledged authority on peripheral nerve injuries. She duly attended him and he referred her on to Mr Wynn Parry whose reputation in rehabilitation was established in the RAF. He admitted the patient to the Royal National Orthopaedic Hospital under the National Health

Service and she was kept there for 3 weeks. During this time she had a series of treatments, including intravenous blocks, a desensitisation programme, various analgesics. All this, however, was to no avail and she returned unchanged. At that point she told me that she was intent of pursuing her case for injury at the factory where she had worked. She told me she had two daughters and she had decided to make her claim and when it was settled she would pass on the proceeds to her daughters and then commit suicide. My relationship with her was very good and I did believe her. I of course protested and asked her to see a psychiatrist. She readily agreed and told me that it would make no difference.

This lady, a most gentle person, saw the consultant psychiatrist, who told me that she had no mental illness and that in all probability she would eventually commit suicide but that he had no means of preventing this action. Sadly I have to say that the last I heard of her was that she was admitted as a suicide attempt but was resuscitated on that occasion. I have since lost contact with her.

This case has naturally left me with a lasting reluctance to carry out amputation of the finger.

I will now proceed to describe a second interesting hand case which was referred to me by a rheumatologist colleague some time ago. Mrs. Y had a grossly swollen finger which had not resolved by various medications. I carried out exploration and found a grossly inflamed synovium. I did a synovectomy.

The specimen was duly sent off and the histology came back – tuberculous synovitis. I referred her to Dr J. MacMahon, respiratory physician, who undertook further investigation and treatment. No primary lesion was found. At the follow-up I talked to her and it transpired that she had a large garden and was in the habit of walking around it towards a stream at the back. She had a problem with badgers which tended to damage the lawn, burrowing into it and she told me that she often repaired the defects patting down the soil with her hand. I immediately concluded that her infection of the finger had been due to the presence of tuberculosis in the badger colony. However, Dr. MacMahon continued to review Mrs Y and he found that she had a fish tank which she tended diligently. One of her more beautiful fish died and was shown to be harbouring *mycobacterium marinum*.

Medicine, as ever, never ceases to fascinate and challenge us and for that we are grateful. We are a very privileged people to work in a profession which provides such an absorbing interest.

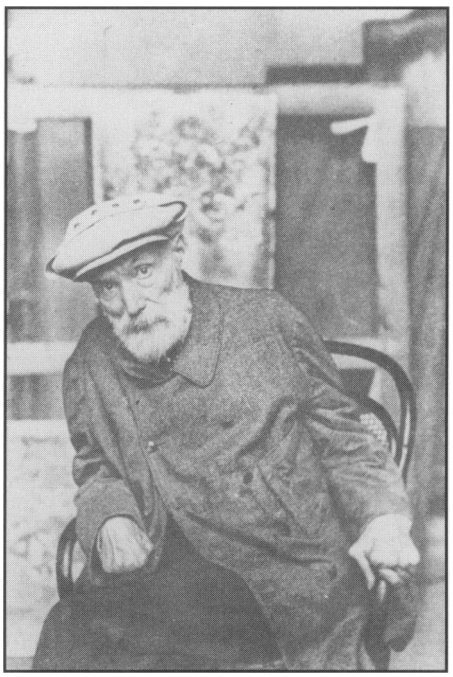

Fig 30. Renoir.

Finally, let me return to the hand and its intimate connection with the brain. The photograph shows Renoir, (Fig. 30), but indicates clearly the hand deformity typical of rheumatoid arthritis, (Fig. 31). Renoir developed the disease at approxi-mately fifty years of age and as it progressed it gradually led to poor hand function, particularly in his later years. During this time, he

Fig 31. Renoir – Deformity of the Hand – (Detail).

continued to produce very many paintings. He became confined to a wheelchair and wore splints on the hands. Disease in the right shoulder limited the area over which he could paint without moving his whole body. He developed a technique of moving the canvas on spindles so that he could cover its fullest extent and paint large works of art.

The limitation of hand function did not affect his skills in painting as he continued, despite his disabilities, to paint until the end of his life.

We must recognise that the hand, deformed and damaged by disease, was still capable of delivering his great talent to produce further masterpieces and we should realise that his immense skill did not really lie within the hand but in the vision of his mind and soul.

We must therefore conclude that the hand is the organ of skill, expression, communication, power and achievement – the most responsive to the brain of all the organs of the body.

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Drug overdoses requiring temporary cardiac pacing;

A study of six cases treated at Altnagelvin Hospital, Londonderry

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SUMMARY

Drug overdoses in general are increasing and overdoses of cardiac medications are also increasing; some are associated with a high mortality. Temporary cardiac pacing has a valuable role in cases of hypotension related to dysrhythmia, or when it is necessary to provide overdrive pacing. However, despite technically successful and uncomplicated pacemaker insertion and restoration of cardiac electrical activity, patients developing bradyarrhythmia and hypotension after an overdose are in a high risk group.

INTRODUCTION

Deliberate drug overdose is a common presentation at Accident and Emergency departments throughout the country. In the year 1st April 1996 to 3rd March 1997, 807 cases of deliberate overdose were admitted to Altnagelvin Hospital; this represents almost 12% of all medical admissions at this hospital (6745 admissions, including 2190 cardiology cases), compared with 321 cases in 1980, 290 in 1981 and 444 in 1982, when they represented 14% of acute medical admissions.[1] In the vast majority of cases, after initial assessment and treatment, most are medically well. However, a number can be in danger of either acute respiratory or cardiac arrest, which may be fatal; artificial ventilation and temporary cardiac pacing respectively may be required.

METHODS

This is a retrospective review of six known cases of drug overdose requiring temporary cardiac pacing between November 1994 and February 1997. Data were collected on age, sex, past cardiac and psychiatric history, haemodynamic signs, electrocardiographic changes, therapeutic intervention and final outcome in the six patients.

CASE HISTORIES

Case 1. A 46 year old woman was admitted having taken an overdose of 1200 mg of flecainide and an unknown quantity of sotalol at an unknown time. She had a past history of paroxysmal atrial fibrillation with an exercise stress test mildly positive for ischaemia and she had been taking these drugs prophylactically for three and a half years and nine months respectively. She had no past history of psychiatric disease or of prior overdose.

On admission to the Accident and Emergency department she was drowsy but still rousable; her heart rate was 30 beats per minute and her blood pressure (BP) 60/25 mmHg. Her ECG showed a broad complex bradycardia. She was treated initially with 3mg of intravenous atropine given as five boluses of 600 mcg. Although her heart rate rose to 53 beats per minute, her BP became unrecordable and she was transferred to the cardiology unit for emergency temporary cardiac pacing. During transit, she developed electro-mechanical dissociation. She was intubated and treated with 3 mg of intravenous adrenaline given as 1 mg boluses, along with cardiopulmonary resuscitation and inotropic support with intravenous dobutamine. A temporary pacing wire was inserted via the right supraclavicular approach; the wire was placed in a satisfactory position but there was failure to respond and the patient died.

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Case 2. A 33 year old woman presented at Accident and Emergency having taken her father's diltiazem, atenolol, isosorbide mononitrate and nifedipine. The of tablets and the time when taken were not known. She had a past psychiatric history of anorexia and severe depression requiring formal psychiatric admissions. She had no history of deliberate self-harm but has since been re-admitted with a second overdose.

She was very agitated on admission, heart rate was 35 beats per minute, systolic blood pressure 60 mmHg and ECG showed a nodal bradycardia. She was treated with an intravenous infusion of *Gelofusine*, intravenous atropine 600 mcg given three times, 10 mls of 10% calcium gluconate intravenously, intravenous glucagon 3 mg in 20 mls of 5% dextrose over five minutes and an intravenous infusion of dobutamine. As there was little improvement in her haemodynamic status a cardiac pacing wire was inserted. Blood pressure after pacemaker insertion increased to 85/33 mmHg.

The dobutamine infusion rate was increased and the blood pressure further improved to 90/50 mmHg that evening, and was 105/60 mmHg the next morning. She was later seen by the psychiatry team who felt the diagnosis was of a borderline personality disorder. The remainder of her stay was uneventful and she was discharged.

Case 3. A 17 year old boy was transferred from another hospital for temporary pacemaker insertion. He had presented there at 12.30 am having taken an overdose of digoxin 15 mg, mefenamic acid 2000 mg and *Tylex* (two tablets) at 10 pm the previous night. He had a history of depression and had three previous admissions following overdose in the past. Cardiac monitoring showed him to be having episodes of asystole lasting up to six seconds, and episodes of ventricular bigeminal rhythm. A digoxin level taken at 5 am had shown a level of 10.77 mcg per litre (therapeutic range 0.8 to 2.0). His initial heart rate was 89 beats per minute and blood pressure 155/88 mmHg, both of which had been maintained despite his arrythmias. He was transferred to Altnagelvin hospital for temporary cardiac pacing.

On arrival he was awake and alert, heart rate was 74 beats per minute and blood pressure 137/82 mmHg. ECG showed sinus rhythm with ST changes attributable to digoxin ("reverse tick"). He was treated initially with 12 vials of *Digibind* and later with a further 4 vials. In view of his documented periods of asystole, a temporary pacing wire was inserted via the right subclavian vein without complication, and capture was achieved. Temporary pacing was only required for a short time after return to the Coronary Care Unit as the patient remained in sinus rhythm without pauses after further administration of *Digibind.*

The serum digoxin level gradually decreased to acceptable levels. No further periods of asystole were noted and he remained haemodynamically stable and the wire was removed. He was seen by the psychiatry team who felt there was no evidence of mental illness and that the overdose had been an impetuous gesture.

Case 4. A 20 year old lady presented to the Accident and Emergency department having taken an overdose of 20 tablets of quinine sulphate 300 mg (total dose 6000 mg) two hours prior to her arrival. She had no relevant past medical history. The medication belonged to a relative.

On arrival she was drowsy but rousable; she complained of having hearing loss but initially had no visual disturbance. Although her heart rate was 108 beats per minute and blood pressure 115/60 mmHg, the ECG showed right axis deviation and first degree heart block. Gastric lavage was performed and she was also given activated charcoal. Blood sent for analysis later showed a quinine level of 20.6 mg/l (toxic levels >10.0 mg/l) and a potassium concentration of 2.6 mmol/l. Intravenous fluids containing potassium supplementation were commenced. She complained later of having total loss of vision and her pupils were found to be dilated and non-reactive.

Soon after admission she developed ventricular tachycardia, but her blood pressure was well maintained and she was treated with a bolus of amiodarone 150 mg intravenously, followed by an infusion of amiodarone (900 mg over 24 hours). Forty minutes after admission she developed a tonic-clonic seizure lasting 5 minutes which resolved with the administration of 5 mg of intravenous diazepam. Post-seizure monitoring showed her to be in persistent ventricular tachycardia with a good blood pressure (120/50 mmHg). She was treated with DC cardioversion, requiring shocks of 100J and then 200J before sinus rhythm returned. It was felt that temporary pacing was required as a prophylactic measure, given the combination of first degree atrio-

ventricular dissociation and ventricular tachycardia. Temporary pacemaker insertion was successfully performed via the right femoral vein as the subclavian and internal jugular veins could not be entered due to the patient's agitation. Overdrive pacing was not required.

The remainder of her stay was unremarkable from a cardiac viewpoint. Her heart rate and blood pressure were well maintained and there were no further episodes of ventricular tachycardia. Her hearing returned to normal quite quickly and her vision made similar, but slower, progress. She was seen by the psychiatry team who did not find her to be mentally unwell, and she was discharged.

Case 5. A 32 year old man was admitted having taken an overdose of 1200 mg of diltiazem, 900 mg of isosorbide mononitrate and possibly bisoprolol while under the influence of alcohol about three hours previously. He had a past history of ischaemic heart disease with an inferolateral myocardial infarction and subsequent episodes of angina requiring hospital admission. He had a prior diagnosis of depression, which was felt to be reactive, and also of phobic anxiety, but he had not taken an overdose before. The medications taken were for his own use.

He was drowsy on admission but awake; his pulse rate was 48 beats per minute, blood pressure 40/10 mmHg and ECG showed a nodal bradycardia. He was treated with gastric lavage, charcoal, intravenous atropine 1200 mcg in two equal doses and 10 mls of 10% calcium gluconate initially. Blood alcohol level was 198 mg%. The heart rate improved to 61 beats per minute and the blood pressure rose to 60/40mmHg. He was commenced on intravenous infusions of dobutamine and dopamine to provide inotropic support.

The following morning his blood pressure had risen to 78/44 mmHg but he remained bradycardic at 50 beats per minute. ECG continued to demonstrate a nodal bradycardia. For this reason (nodal bradycardia with hypotension) a temporary cardiac pacing wire was successfully inserted via the left supraclavicular approach.

Blood pressure rose to 105/70 mmHg following this procedure and he was gradually weaned off both dopamine and dobutamine. Haemodynamic stability was restored and the pacing wire was removed. He was seen by the psychiatrists who offered him admission to the local psychiatric hospital but this was declined and he was discharged.

Case 6. A 14 year old boy presented at the Accident and Emergency department via ambulance having taken an overdose estimated at 2800 mg of atenolol and 5400 mg of diltiazem around ten hours prior to admission. The drugs taken were not for his personal use and his past medical history was totally unremarkable.

On admission he was drowsy but still able to communicate coherently. His heart rate was 51 beats per minute, blood pressure 60/31 mmHg and ECG showed a nodal bradycardia. Twenty minutes later his heart rate dropped to 36 beats per minute with an unrecordable blood pressure. He was treated with intravenous atropine 3 mg given in five boluses of 600 mcg, 10 mls of 10% calcium gluconate, 1 mg of intravenous glucagon, intravenous *Gelofusine* and was commenced on an intravenous infusion of dobutamine; it was decided that temporary cardiac pacing was necessary. Before transfer to the cardiology unit he developed asystole which was treated with cardiopulmonary resuscitation and intravenous boluses of adrenaline; he was successfully resuscitated and moved immediately to the Cardiology unit where a temporary cardiac pacing wire was inserted via the left supraclavicular approach without difficulty. He subsequently developed electromechanical dissociation with a satisfactory pacing rhythm on the monitor but he had no palpable cardiac output and no ventricular systole was seen on X-ray screening of the mediastinum. He had been intubated and was commenced on cardiopulmonary resuscitation combined with intensive inotropic support with high dose adrenaline, noradrenaline, dobutamine and *Gelofusine*. He also received further doses of calcium gluconate and glucagon. Although he had a strong output with CPR, no spontaneous cardiac output was detected despite clear electrical activity on monitor. After a period of resuscitation which lasted three hours in total with no detectable recovery, this was discontinued and he was pronounced dead.

DISCUSSION

Although these data are retrospective and uncontrolled, we feel that they are useful indicators of the type of rhythm disturbance that

TABLE

Summary of information in the six cases presented

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Age	46 years	33 years	20 years	17 years	32 years	14 years
Sex	Female	Female	Female	Male	Male	Male
Cardiac History	Paroxysmal atrial fibrillation; mildly positive EST	None	None	None	Myocardial infarcation; angina	None
Psychiatric History	None	Anorexia, Depression, Personality disorder	None	Depression	Depression; Phobic anxiety	None
Prior Overdose	None	No, but one since	No	Three known	None	None
Drugs taken and dosage	Flecainide 1200 mg; Sotalol (unknown dose)	Diltiazem, Atenolol, Isosorbide mononitrate Nifedipine (doses unknown)	Quinine sulphate 600 mg	Digoxin 15 mg; Mefenamic acid 2 g; Tylex 2tabs	Diltiazem 1200 mg; Isosorbide momonitrate 900 mg; ?Bisoprolol	Atenolol 2800 mg; Diltiazem 5400 mg
Time until presentation after taking overdose	Unknown	Unknown	2 hours	2 hours, 30 mins	3 hours	10 hours
Heart rate on admission	30 bpm	35 bpm	108 bpm	89 bpm	48 bpm	51 bpm
Blood pressure on admission	60/25 mmHg	60 mmHg systolic	115/60 mmHg	155/88 mmHg	40/10 mmHg	60/31 mmHg
ECG findings	Broad complex bradycardia	Nodal bradycardia	Right axis deviation; first degree heart block, ventricular tachycardia (later)	Episodes of asystole; ventricular bigeminy	Nodal bradycardia	Nodal bradycardia
Indication for pacing	Bradycardia with hypotension	Bradycardia with hypotension	Persistent ventricular tachycardia	Episodes of asystole up to 6 secs	Bradycardia with hypotension	Bradycardia with hypotension
Blood pressure after pacing		85/33 mmHg	132/71 mmHg	97/55 mmHg	105/70 mmHg	
Outcome	Dead	Alive	Alive	Alive	Alive	Dead

may occur after overdose of cardiotoxic medications. It is impossible to prove whether pacing influences the outcome in these patients, but it would be unethical and clinically unacceptable to withhold temporary pacemaker insertion in a patient with documented bradydysrhythmia and hypotension; given the negative chonotropic effects of all the drugs ingested, temporary pacemaker insertion is a rational therapy to adopt.

Although deliberate drug overdose is a common reason for acute medical admission to hospital, few such cases require temporary cardiac pacing. However, the mortality in this group of patients is an indicator of the worrying prognosis in patients who require temporary cardiac pacing as a consequence of drug-induced arrhythmias. Beta-blockers and calcium channel blockers were the drugs most commonly implicated. Calcium channel blockers are increasingly prescribed for hypertension, angina and arrhythmias and with their rise in popularity there has been an increase in the incidence of overdose of these drugs.[2, 3]

Three of the six cases described involved overdose of calcium channel blockers; all presented with nodal bradycardia and hypotension which are predictable from their pharmacological action on the myocardium, vascular smooth muscle and cardiac conducting system.[4] Other clearly documented side effects include sinus bradycardia, accelerated atrioventricular node conduction, second and third degree heart block.[5]

The ideal emergency treatment of calcium channel overdose has yet to be defined, but includes calcium, glucagon, atropine, isoproterenol, dopamine, dobutamine, adrenaline and noradrenaline.[2] The administration of calcium is not always effective in treating overdoses of a calcium channel blocker, but clinical improvement has been demonstrated in most instances.[3]

The use of cardiac pacing in calcium channel blocker overdose has been mentioned in a number of sources with an overall positive response but also acknowledgment that there may be a failure to capture successfully or with no haemodynamic improvement, as in case 6.[2, 3, 5]

Beta adrenergic blocking agents are used in angina, hypertension and as anti-arrhythmic agents. The usual clinical manifestations of beta-blocker overdose include bradycardia, hypotension, low cardiac output, cardiac failure, cardiogenic shock, bronchospasm, respiratory failure, seizures and prolonged intraventricular conduction.[6]

The haemodynamic compromise induced by beta-blocker overdose usually responds to sympathomimetics, parasympatholytics, glucagon, phosphodiesterase inhibitors and cardiac pacing. Glucagon in particular has been proposed as the drug of choice due to its effect in increasing levels of intracellular cyclic adenine monophosphate (cAMP) independently of adrenergic receptors. Cardiac pacing has been documented in addition to glucagon as the treatment of first choice for the management of beta-blocker overdose.[6]

Flecainide is a class 1C anti-arrhythmic drug indicated for the treatment of ventricular and supraventricular dysrhythmias including Wolff-Parkinson-White syndrome. In general, overdose with this class of drug is associated with a high mortality.[7] It is felt that there is a correlation between toxic levels in the blood and ECG changes, in particular broadening of the QRS complex. Other ECG changes reported include prolongation of the PR interval, right bundle branch block and giant inverted T waves. Flecainide has a negative inotropic effect which has been suggested as the cause of the profound hypotension seen in many cases of overdose. Survival after overdose is associated with persistent ECG changes lasting more than 15 days. The hypotension has been found to respond to intravenous dobutamine, dopamine and isoprenaline.[7]

Quinine is used for the treatment of nocturnal leg cramps, malaria and myotonia. As it is an optical isomer of quinidine, a class 1A anti-arrhythmic drug, quinine and quinidine have a number of cardiotoxic effects including sinus tachycardia, sinus bradycardia, widening of the QRS complex, prolongation of the PR and QT intervals, atrioventricular blocks, broad complex tachycardias, torsades de pointes and idioventricular rhythms. Other effects include tinnitus, deafness and visual disturbance.[8]

Standard poisoning treatment and supportive care are the mainstay of therapy in quinine poisoning. Therapeutic inotropic support and cardiac pacing may be required.[8]

Digoxin is used in the treatment of atrial fibrillation, paroxysmal supraventricular

tachycardia, atrial flutter, and in cardiac failure. Because of its effects on the myocardium, sinoatrial and atrioventricular nodes via the vagus nerve, digoxin in overdose will induce arrhythmias including ventricular ectopic beats, ventricular tachydysrhythmias, paroxysmal supraventiicular tachycardia and heart block. Death from digoxin intoxication usually results from ventricular fibrillation, asystole, pump failure or from mesenteric infarction.[9]

Digoxin overdose is treated by use of digoxin-specific Fab antibody fragments as antidote.[10] Indications for pacing include severe bradycardia (mostly secondary to atrioventricular block or to sinoatrial block) and hyperkalaemia > 5 mmol/L.[9] There had been a suggestion that during digoxin poisoning, the fibrillary threshold is lowered and thus cardiac pacing may lead to overdrive inhibition of spontaneous rhythms. Although the failure rate with use of the pacemaker was higher than with use of the Fab antibody fragments, the difference was not significant.[9]

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A review of direct current cardioversions for atrial arrhythmia

S D Johnston, T G Trouton, C Wilson

SUMMARY

The risk of arterial embolism is well recognised following Direct Current Cardioversion (DCC) for atrial fibrillation although the use of prophylactic anticoagulation remains controversial.

Aim: **To determine the risk of arterial embolism post-cardioversion and which factors predict successful cardioversion and maintenance of sinus rhythm.**

Materials and Methods: **A retrospective study was carried out of all cardioversions performed for atrial fibrillation and atrial flutter at the Waveney Hospital Ballymena, during 1989-1993. A review of medical records and electrocardiograms was carried out to assess demographic characteristics, co-existent diseases, anticoagulant status, echocardiographic features and characteristics of the arrhythmia. Embolic events in the six weeks post-cardioversion were noted.**

Results: **The study included 157 cardioversions in 109 patients. The predominant arrhythmia was atrial fibrillation (n=108, 69%). Three of 109 patients (2.7%) experienced embolic complications, none of whom had anticoagulation prior to the procedure. No risk factors for cerebro-vascular disease or significant valvular heart disease were present. Return to sinus rhythm was achieved in 143 (91%) procedures. Increasing coarseness of atrial fibrillation was associated with a non-significant trend towards successful restoration of sinus rhythm (p=0.1 8). Recurrence of the original arrhythmia was predicted by an increase in coarseness of atrial fibrillation ($p<0.05$).**

Conclusions: **These findings indicate that embolic complications can occur in patients undergoing DCC with normal echocardiographic dimensions, and that prophylactic anticoagulation should be considered in all patients. Coarseness of atrial fibrillation may be used as a guide to predict the chance of successful cardioversion and of the likelihood of maintaining sinus rhythm once this has been achieved.**

INTRODUCTION

Atrial fibrillation is the commonest sustained arrhythmia. Chronic atrial fibrillation is associated with a doubling of mortality,[1] largely due to an increase in the incidence of stroke.[2] The potential benefits of a return to sinus rhythm are a reduction in the risk of systemic embolisation and an improvement in haemodynamics secondary to the return of atrial mechanical function.[3]

Return to sinus rhythm may occur spontaneously or may be achieved by chemical or electrical cardioversion. Direct Current Cardioversion (DCC) in atrial fibrillation is a successful, safe procedure and should be considered for all patients regardless of aetiology. DCC is known to be highly effective for the immediate conversion of atrial fibrillation to sinus rhythm.[4] The risk of embolism following DCC for atrial fibrillation is well recognised but the use of routine prophylactic anticoagulation remains somewhat controversial. Certain factors, such as duration of atrial fibrillation, left atrial size as assessed by M-mode echocardiography and aetiological associations may be predictive of a successful

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restoration of sinus rhythm, although these have not been confirmed by Dittrich.[5]

We reviewed the results of 202 cardioversions for atrial fibrillation and atrial flutter over a five year period to assess further the need for prophylactic anticoagulation. In addition, we have attempted to determine factors predicting the risk of thrombo-embolism post-cardioversion and also those parameters predicting successful cardioversion and maintenance of sinus rhythm once this had been achieved.

MATERIALS AND METHODS

Between 1st January 1989 and 31st December 1993, 202 elective DCC procedures were undertaken in 116 adult patients (82 male) with atrial fibrillation or atrial flutter as the primary arrhythmia. Cardioversions in which atrial fibrillation or atrial flutter were present for less than 48 hours and cardioversions in which anticoagulant status was indeterminate were excluded from subsequent analysis.

All cardioversions were carried out in the Coronary Care Unit under intravenous sedation with midazolam (usual dose 5-10 mgs). A series of synchronised direct current shocks of increasing energy was delivered starting with 50 Joules, and increasing incrementally until sinus rhythm was achieved or 360 Joules was reached. Pre- and post-cardioversion 12-lead electrocardiograms were available to verify heart rhythm.

The medical records were reviewed for demographic characteristics and coexistent diseases (ischaemic heart disease, valvular heart disease, hypertension, diabetes mellitus or cerebro-vascular disease). Electrocardiographs were examined to confirm heart rhythm prior to DCC and to determine the duration of the arrhythmia. In patients with atrial fibrillation the "coarseness" of atrial electrical activity was assessed subjectively and determined to be "coarse", "intermediate" or "fine", depending on the baseline atrial electrical activity and mean "f" wave cycle length. In addition, left atrial size, left ventricular end-diastolic diameter and the presence or absence of valvular heart disease were noted in those patients in whom echocardiography had been performed. Anticoagulant status was recorded and an International Normalised Ratio greater than 1.5 was considered therapeutic. Suspected embolic events in the subsequent six weeks were recorded.

STATISTICS

Values, where applicable, are given as mean (SD). Group comparisons of categoric variables were made using the Chi-square test. Continuous variables were compared by using the Mann Whitney U-test. A value of $p < 0.05$ was considered significant.

RESULTS

Two hundred and two procedures in 116 patients were identified during the study period. Forty-five cardioversions were excluded from the study on account of cardioversion performed within 48 hours of the onset of the arrhythmia (n=38) or indeterminate anticoagulant status (n=7). The study group therefore consisted of 157 procedures in 109 patients. In 22 (14%) cardioversions prophylactic anticoagulation with oral warfarin had been prescribed prior to the procedure.

Of the 109 patients, 77 (71%) were male and their ages ranged from 17 to 86 years (mean 65.0 (11.1) years). Seventy-five patients required cardioversion on a single occasion and the remaining 34 patients accounted for the other 82 cardioversions included in the study. The predominant rhythm disturbance requiring cardioversion was atrial fibrillation (atrial fibrillation in 108, atrial flutter in 49 cardioversions). Concomitant ischaemic heart disease was present in 31 patients (28%), mitral valve disease in 33 patients (30%), hypertension in 24 patients (22%), diabetes mellitus in 4 patients (4%) and a history of prior cerebro-vascular accident in 3 patients (3%). Mitral valve disease was significantly more prevalent in the anticoagulated group ($p<0.05$) whereas ischaemic heart disease was significantly more prevalent in the group not receiving anticoagulation ($p<0.05$). There was no significant difference in anticoagulant status for the sub-groups with hypertension ($p=0.75$), diabetes mellitus ($p=0.33$) or previous cerebro-vascular disease ($p=0.33$) (Table 1).

The duration of the arrhythmia ranged from 48 hours to two years. The duration was less than eight days in 80 cardioversions (51%), eight to 28 days in 40 (25%), 29 days to one year in 36 (23%) and greater than one year in one cardioversion. Two patients had prosthetic aortic valves and one patient had both mitral and aortic prostheses. Echocardiographic data was available in 52 patients (48%) in the study group. The mean left atrial size was 49.9 (6.2) mm (range 36-61 mm).

When the arrhythmia had been present for less than 30 days the mean left atrial size was 49.8 (5.9) mm compared to 50.3 (7.3) mm when present for more than 30 days (p=0.89). The mean left ventricular end diastolic diameter was 56.2 (10.1) mm when the arrhythmia was present for less than 30 days, compared to 56.6 (7.5) mm when present for more than 30 days (p=0.98).

TABLE I

A comparison of risk factors present for cerebro-vascular disease between the groups of patients undergoing cardioversion with and without prophylactic anticoagulation

	Patients undergoing DCC with prophylactic anticiagulation (n=18)	*Patients undergoing DCC without prophylactic anticoagulation (n=91)*	
Ischaemic heart disease	1	30	p<0.05
Mitral valve disease	10	23	p<0.05
Hypertension	3	21	p=0.75
Diabetes mellitus	1	3	p=0.33
Previous cerebro-vascular disease	0	3	p=0.33
No risk factors	3	11	

EMBOLIC PHENOMENA

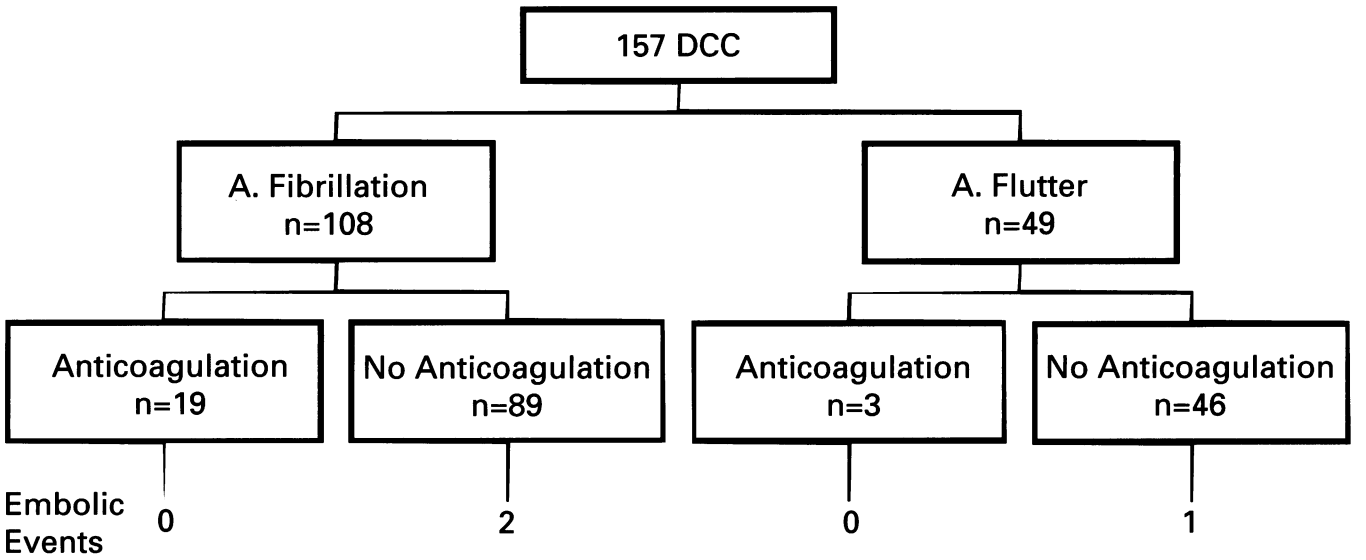
Cerebral embolic events occurred within six weeks of DCC following two of 89 procedures performed for atrial fibrillation and one of 46 procedures performed for atrial flutter, undertaken without anticoagulant prophylaxis (Figure 1). The patients were aged 55, 56, and 63 years (mean 58 years). The arrhythmias were present for 7, 10 and 50 days (mean 22 days). These patients had no co-existent risk factors for cerebro-vascular events. Echocardiographic data were available in two of these patients in whom the left atrial size, left ventricular end-diastolic diameter and left ventricular function were normal. Neither had significant valvular heart disease; one had mild mitral regurgitation and the other had mitral valve prolapse. One patient had a transient right hemiparesis and right homonymous hemianopia and was prescribed aspirin. The other two patients experienced a left hemiparesis and both were prescribed warfarin following CT brain scan confirming an ischaemic infarction. No fatalities resulted from these embolic events.

SUCCESSFUL CARDIOVERSION

Immediate restoration of sinus rhythm was achieved in 143 (91%) of the 157 cardioversions performed. In patients who were successfully cardioverted to sinus rhythm the mean left atrial size was 49.9 (5.9) mm whereas in patients whose cardioversions were not successful the mean left atrial size was 49.2 (9.7) mm (p=0.73). In patients who were successfully cardioverted, the mean left ventricular end-diastolic diameter was 56.1 (9.8) mm whereas in patients whose cardioversions were not successful it was 58.2 (4.0) mm (p=0.73). Increasing coarseness of atrial fibrillation was associated with a successful restoration of sinus rhythm. All 19 patients with coarse atrial fibrillation were successfully cardioverted compared to 36 of 41 (88%) with intermediate atrial fibrillation and 30 of 36 (83%) of those with fine atrial fibrillation (p=0.18). Age, duration of the arrhythmia, left ventricular end-diastolic diameter and aetiology were not predictive of successful restoration of sinus rhythm.

Energy requirements in successful cardioversions for atrial flutter were significantly lower than those required for atrial fibrillation. Sixty-six (69%) of 96 successful cardioversions for atrial fibrillation required ≤ 200 Joules whereas 42 (89%) of 47 successful cardioversions for atrial flutter required ≤ 200 Joules (p<0.001). Energy

Figure 1
Summary of 157 cardioversions reviewed with relation to the number of embolic events which occurred.



requirements for those patients with coarse atrial fibrillation were significantly lower compared with those required for intermediate or fine atrial fibrillation (p=0.014). Table II shows the energy requirements for 96 cardioversions for coarse, intermediate and fine atrial fibrillation. One cardioversion for atrial fibrillation had no record of the energy required for successful cardioversion; eleven pre-DCC electrocardiograms were unavailable for analysis.

TABLE II

Energy Requirements for the subgroups of Atrial Fibrillation undergoing Successful Cardioversion (n=96)

Energy (J)	*Coarse n(%)*	*Intermediate n(%)*	*Fine n(%)*
50,100J	8(42)	1(2)	7(19)
150, 200J	9(47)	26(63)	15(42)
300, 360J	2(11)	14(35)	14(39)
	19(100%)	41(100%)	36(100%)
		χ^2=8.47	p=0.014

RELAPSE RATES

Following successful restoration of sinus rhythm in 143 cases, recurrence of the original arrhythmia occurred in 75 cases (52%). The mean time to relapse was 217 days (range 1 day to 45 months). At one month 25 (17%) patients were found to have relapsed, compared to 53 (37%) at six months. In patients who subsequently reverted to their original rhythm the mean left atrial size was 50.7 (5.9) mm compared to those who remained in sinus rhythm in whom the mean left atrial size was 48.8 (5.9) mm (p=0.43). In patients who subsequently reverted to their original rhythm the mean left ventricular end-diastolic diameter was 55.8 (8.2) mm compared to those who remained in sinus rhythm in whom the mean left ventricular size was 56.4 (11.7) mm (p=0.9).

Seven of 19 cardioversions for coarse atrial fibrillation resulted in a recurrence of the original rhythm compared to 20 of 36 for intermediate and 17 of 30 for fine atrial fibrillation (p<0.05).

Ischaemic heart disease was the aetiological factor in 13 of 75 cardioversions which were followed by a recurrence of the original rhythm, compared to 23 of 68 cardioversions in which sinus rhythm was maintained (p<0.01). Although a higher number of patients with mitral valve disease, hypertension and unknown aetiology experienced

a recurrence of the original rhythm, these differences did not reach a significant level. The age of the patient and the duration of the arrhythmia were not predictive of relapse. The effect of anti-arrhythmic agents on relapse rates was not examined.

DISCUSSION

Synchronised electrical cardioversion for atrial fibrillation and atrial flutter may be complicated by systemic embolisation. This has been estimated to occur in up to 7% of patients.[6] The atrial arrhythmia most likely to be associated with embolic events is atrial fibrillation,[7, 8] although one of three patients developing cerebral thromboembolism in our study had been cardioverted for atrial flutter.

Although it has been suggested that patients with atrial fibrillation should receive anticoagulation prior to undergoing direct current cardioversion,[4, 8] there has been a lack of consensus on such treatment prior to the period included in this study. Recently it has been recommended that anticoagulation should be given for three weeks before and four weeks after DCC for atrial fibrillation of more than 48 hours duration.[9] Data is sparse on the incidence of embolism after cardioversion for specific arrhythmias. Lown, in 1967, reported a 1.2% incidence rate of embolisation after reviewing 456 cardioversion attempts in which none of the patients were anticoagulated.[4] In 1969 Bjerkelund and Orning reported that cardioversion without anticoagulation resulted in a 6.8% incidence rate of embolism, compared with a significantly lower rate of 1.1% with anticoagulation, following successful cardioversion.[10] Mancini and Weinberg reviewed cardioversions over a 10 year period and found no embolic events in the group prescribed anticoagulation, whereas 7% of the group without anticoagulation had embolic complications.[11] Our 2.1% incidence rate of embolic events after successful cardioversion for atrial fibrillation and atrial flutter is higher than other reports.[12-15]

Atrial fibrillation of long duration is a well documented risk factor for stroke.[16] It has been recommended[7, 8] that patients with atrial fibrillation of short duration (<1 week) do not need anticoagulation before undergoing cardioversion. In our three patients suffering cerebral thromboembolism following DCC the arrhythmia had been present for 7, 10 and 150 days respectively.

It remains unclear how long anticoagulation therapy should be continued after cardioversion. Mechanical atrial activity may take several weeks to resume as shown by Manning who assessed patients by means of serial Doppler echocardiography. They found that peak "A" wave velocity did not return to normal until three weeks after cardioversion in patients who maintained sinus rhythm.[17]

In our series of patients we were able to cardiovert 91% (143 of 157) of patients to sinus rhythm. This is similar to results published by Dalzell.[18] Our findings show that smaller left atrial size is not a useful factor in determining which patients are more likely to be successfully cardioverted. Zipes reports that the only important predictor is the duration of atrial fibrillation.[19] The duration of the arrhythmia was not shown to be a significant predictor in our study. We have identified increasing coarseness of atrial fibrillation as a useful predictor of successful restoration of sinus rhythm.

Without further treatment there is a high rate of relapse following successful cardioversion. The influence of anti-arrhythmic agents was not considered in this review. Sinus rhythm was maintained in 118 of 143 cases (82%) at one month and 90 of 143 cases (63%) at 6 months which compares favourably with those of Dittrich[5] who has reported rates of 69% and 58% at 1 and 6 months respectively. Useful and safe prophylaxis of atrial fibrillation has been demonstrated in selected groups of patients using anti-arrhythmic drugs from different classes, although a metanalysis of randomised control trials in the use of quinidine has indicated an increased total mortality.[20, 21]

Atrial fibrillation is a common arrhythmia that is associated with significant morbidity and mortality. Direct current cardioversion should be considered in all patients regardless of aetiology since initial success rates are high. Our findings indicate that embolic complications can occur in patients undergoing direct current cardioversion without prophylactic anticoagulation who have normal echocardiographic findings and no risk factors for cerebrovascular events. We therefore recommend that prophylactic anticoagulation should be considered in all patients with atrial fibrillation or atrial flutter of >1 week's duration

prior to attempted cardioversion. Anticoagulation should be given for 4 weeks prior to and, at least, 4 weeks following elective cardioversion. Coarseness of atrial fibrillation may be used as a subjective guide to predicting the probability of achieving and maintaining sinus rhythm. The high relapse rates following successful cardioversion remain problematic and the role of prophylactic anti-arrhythmic drugs and non-pharmacological measures remains to be established in these patients with recurrent atrial arrhythmias.

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The haematuria clinic – referral patterns in Northern Ireland

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SUMMARY

One hundred consecutive patients with haematuria were seen over a three month period at the haematuria clinic, Belfast City Hospital. 14% of patients were found to have transitional cell carcinoma of the urinary bladder; all of these presented with frank haematuria and were over 50 years of age. No malignancy was detected in the microscopic haematuria group. 14% of patients with macroscopic haematuria held back for longer than one month before seeking advice from their general practitioner. 23% with macroscopic and 30% with microscopic haematuria had their symptoms noted by the general practitioner for more than a month before they were referred for investigation. The waiting time for initial investigation at the haematuria clinic took longer than six weeks in 52% with macroscopic and 39% with microscopic haematuria. Our study has identified a high-risk group who need immediate referral and investigation. The importance of patient education, rapid referral by general practitioners and also the need to increase the capacity of the haematuria clinic are emphasized.

INTRODUCTION

Haematuria is often the first presenting sign of an underlying urological malignancy. Urological malignancy has been reported in 14.7 to 21.8% of patients with macroscopic haematuria[1, 2] and in up to 2% of patients with microscopic haematuria.[3] The commonest malignancy presenting with this symptom is transitional cell carcinoma most often involving the bladder (TCCB).

Most bladder tumours are curable if detected at an early stage and if appropriate treatment is instituted.[4,5] As the disease progresses, prognosis decreases markedly and the 5 year survival for muscle-invasive bladder cancer after conventional treatment with radiotherapy or cystectomy or in combination is at best 35% - 45%.[6-8] Both the depth of invasion and development of metastases are time-dependent and Hendry et al[9] has shown that with early diagnosis an increased proportion of potentially curable T2 tumours are detected. The data support the assertion that delay in detecting and instituting treatment has an adverse effect on patient survival.

The investigation of haematuria commonly involves a delay before treatment is undertaken. The causes of this may be attributed to delay by the patient in seeking medical advice, general practitioner delay in referring for investigation and hospital delay in providing timely investigation.

Patients with haematuria as a rule tend to seek advice quickly.[4, 9, 10] Therefore the delays in initiating treatment may be the responsibility of medical personnel. Studies have indicated that the average time delay from general practitioner referral to diagnostic cystoscopy is four weeks.[11]

Haematuria clinics have been set up across the United Kingdom to provide an investigative service for patients with haematuria in an effort to decrease the morbidity and mortality by earlier diagnosis of urological malignancy. This paper audits the diagnoses, referral patterns and time delays in the investigation of patients with haematuria referred to the Department of Urology, Belfast City Hospital, Northern Ireland.

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PATIENTS AND METHODS

Our study included 100 consecutive patients attending the haematuria clinic between May 1995 and August 1995. Following referral by the general practitioner for investigation of frank or microscopic haematuria, patients were seen at the haematuria clinic (scheduled once a week) at the Day Procedure Unit, Belfast City Hospital. A history was taken of their complaint including details of the onset of symptoms and when they were seen by their general practitioner. The date of referral was taken from the general practitioner's referral letter. Following this, patients underwent a flexible cystoscopy under local anaesthesia (2% lignocaine gel).

Any new case of bladder cancer detected had an urgent intravenous urogram (IVU) and they were placed on the next available operating list of the appropriate consultant for transurethral resection under general or regional anaesthesia to stage and grade the tumour. All other patients had a routine IVU requested on an outpatient basis and were given early appointments to the clinic for further assessment.

RESULTS

Over the period May 1995 to August 1995, 100 patients (64 males, 36 females) with mean age of 57 years (range 18 - 87) were referred to the haematuria clinic. 56 patients (56%) were referred with frank haematuria, and 44 patients (44%) with microscopic haematuria discovered on routine testing.

TABLE 1

Diagnosis made in 100 patients at the haematuria clinic.

Diagnosis	Number of patients (%)
No Diagnosis	73
Transitional cell carcinoma bladder (TCCB)	14
Benign prostatic hyperplasia	5
Previous transurethral resection of prostate	3
Bladder neck stenosis	2
Urethral stricture	2
Meatal Stenosis	1

Diagnoses made at the haematuria clinic are shown in Table I. Fourteen patients (14%) had TCCB and all these presented with frank haematuria. The mean age of these patients was 71 years (range 53 -82). No malignancies were detected in the microscopic haematuria group in this study.

Eight patients (8%) had macroscopic haematuria related to their prostate. Three patients had previously undergone transurethral resection of their prostate and had prominent vessels at the bladder neck which bled on contact. The remaining five patients had mild to moderate outflow obstructive symptoms, and were found to have an enlarged prostate with congested mucosa overlying the gland and bladder neck.

Thirty-three (60%) patients with macroscopic haematuria presented to their general practitioners within one week, and by four weeks, forty-eight (86%) patients had sought advice from their general practitioner Table II.

TABLE II

Time between patient noticing/detecting symptoms and attending general practitioner in the macroscopic haematuria group (patient delay).

Patient delay	*Macroscopic haematuria*
< 1 week	33 (60%)
1 - 4 weeks	15 (26%)
> 4 weeks	8 (14%)
Total	56

77% with macroscopic, and 70% with microscopic haematuria were referred by the general practitioner for investigation within one month of presentation Table III. Within the first week of attending, 32% of patients with macroscopic, and 25% with microscopic haematuria had been referred. However, 14% of patients with macroscopic haematuria had symptoms for more than two months before being referred for hospital investigation.

The mean time from referral by the general practitioner to attendance at the clinic was six weeks (range 2 - 12 weeks). Only 18% with macroscopic and 25% with microscopic

Table III

Time between attending general practitioner to referral (general practitioner delay).

GP delay	*Macroscopic haematuria*	*Microscopic haematuria*	*All patients (%)*
< 1 week	18 (32%)	11 (25%)	29
1 - 4 weeks	25 (45%)	20 (45%)	45
5 - 8 weeks	5 (9%)	6 (14%)	11
> 8 weeks	8 (14%)	7 (16%)	15
Total	56	44	100

haematuria were investigated within four weeks of referral. However, 48% with macroscopic and 61% with microscopic haematuria were investigated within six weeks of referral by their general practitioner. The remaining patients, 52% with macroscopic, and 39% with microscopic haematuria, took longer than six weeks to have their initial investigation Table IV.

TABLE IV

Time between general practitioner referral to attendance at the haematuria clinic (hospital delay).

Hospital delay	*Macroscopic haematuria*	*Microscopic haematuria*
< 2 weeks	0	2 (5%)
2 - 4 weeks	10 (18%)	9 (20%)
4 - 6 weeks	17 (30%)	16 (36%)
6 - 8 weeks	23 (41%)	13 (30%)
> 8 weeks	6 (11%)	4 (9%)
Total	56	44

DISCUSSION

The importance of investigating haematuria with minimal delay is well established. Our study has shown that patients attending a haematuria clinic have a 27% rate of a definitive pathological diagnosis being made. In particular, 14% of patients had urothelial malignancy, all having had macroscopic haematuria as the presenting symptom; and all were over 50 years of age. The malignancy rate in this study is higher than the rate of 2 - 11% reported by others.[3,12] Although in this study, no patients with microscopic haematuria had malignancy in the lower urinary tract, other larger studies' have reported up to 2% pick-up rate of malignancy in patients with microscopic haematuria. While all patients with haematuria require investigation, our results suggest that those with macroscopic haematuria require urgent referral and should have completed investigations within four weeks of presentation.

This study supports the findings of others that patients with macroscopic haematuria tend to seek advice early. [4,9,10] Wallace and Harris[4] showed that 75% of their patients attended their general practitioner within one month of developing haematuria and our study showed that 86% attended their general practitioner within the month. However, 14% patients with macroscopic haematuria waited more than one month before seeking the general practitioner's help. We were able to identify two major reasons for the delay in patients with macroscopic haematuria referring themselves for diagnosis. One was the fear of the diagnosis of malignancy; and the second was the lack of urgency because of previous history of haematuria either due to urinary tract infections or calculus disease. Among the eight patients who delayed more than four weeks in attending the general practitioner, one was found to have malignancy. This finding would suggest that better patient education as to the significance of haematuria is required.

In our study, 77% with macroscopic haematuria, and 70% with microscopic haematuria were referred within four weeks of attending the general practitioner. However, 23% of those with macroscopic haematuria, and up to 30% with

microscopic haematuria had their symptoms for more than a month before they were referred for hospital investigation. It is clear from our results that patients with macroscopic haematuria need to be referred immediately by their general practitioner. Repeated urinalysis and urinary bacteriology should not delay referral in this macroscopic category. Those with microscopic haematuria can be commenced on antibiotic therapy if symptoms of UTI are present and if asymptomatic microscopic haematuria persists then they should be referred for hospital investigations without delay. It is clear that the waiting time for patients to attend the haematuria clinic is too long, with 82% with macroscopic haematuria and 75% with microscopic haematuria waiting longer than four weeks. Some reasons for delay were:-

1. patients who could /did not attend and had to be re-appointed
2. inappropriate referrals to the general surgeon, gynaecologist or renal physician etc., who then refers these patients to us
3. insufficient number of sessions allocated to the haematuria clinic

The ideal would be for patients to be seen at the haematuria clinic within four weeks of the onset of symptoms.[11] Therefore, to reduce the waiting time for patients to be seen at the haematuria clinic, all patients with haematuria should be referred to the urology service and this service should increase the number of haematuria clinics to two per week.

Our study has highlighted a high risk group of patients with painless frank haematuria above the age of 50 years of age who require immediate referral and investigation. It has also emphasised the importance of patient education in reducing delay in seeking medical attention and the need for the general practitioner to refer patients earlier especially if the patient presents with painless frank haematuria. It has become apparent to us that in order to offer an efficient service for the investigation of haematuria, we would need two clinics per week to reduce the waiting time of those referred.

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Trans-cervical resection of the endometrium: the first four years' experience at the Belfast City Hospital

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SUMMARY

We have evaluated the quality of service provided in performing trans cervical resection of the endometrium (TCRE) in the treatment of women presenting with menstrual dysfunction.

Of the 78 patients who underwent TCRE, ten had, at the time of writing, subsequently undergone hysterectomy. A chart review was carried out on those patients. Two others were on the waiting list for hysterectomy. Sixty-one of 68 (90%) patients responded to the postal questionnaires. Of these, 50 (82%) were satisfied with the result of the procedure. Twenty-three (38%) were rendered amenorrhoeic. Thirty-eight still had bleeding, but of these, 19 (31%) had very light regular periods and 13 (21%) had only an occasional stain. Overall, 90% of women felt that there had been an improvement in their bleeding. Six (10%) women claimed that there had been no improvement.

TCRE is a safe and effective alternative to hysterectomy or medical treatment for the treatment of menstrual dysfunction.

INTRODUCTION

Since its introduction in 1983[1] TCRE has become an increasingly popular treatment for menstrual disorders, in particular, for menorrhagia. Although patient satisfaction rates are lower than for hysterectomy,[2] operative hysteroscopy provides a suitable alternative to medical therapy in women wishing to avoid hysterectomy. The technique, once learned, is relatively simple, with average operating times of approximately 20 minutes.

Complications occur most frequently when learning the procedure, but with experience, complication rates of less than 2% are reported.[3,4] Those which occur most frequently are fluid overload and uterine perforation.

TCRE is both cost effective and clinically efficacious in the treatment of menstrual disorders.[5] The equipment used costs considerably less to install than for laser endometrial ablation and is more readily available than radiofrequency thermal inducers. Another advantage is that it provides specimens for histopathological analysis, where laser ablation, radiofrequency ablation, and thermal balloon ablation do not.

It has been shown in the Scottish Hysteroscopy Audit Group study that TCRE has a significant role to play in the management of menorrhagia.[6] This retrospective study set out to establish the quality of service provided when TCRE is performed for the treatment of menorrhagia at the Belfast City Hospital.

METHODS

All women who attended the Gynaecology clinic of H R McClelland complaining of menorrhagia are offered hysteroscopic surgical treatment as an additional choice between medical treatment and hysterectomy. Seventy-eight women who underwent TCRE in the Belfast City Hospital between January 1992 and June 1995 were identified through computerised procedure coding.

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All patients had endometrial preparation with either two or three doses of goserelin 3.6mg administered 28 days apart. The procedures were performed according to a standard resection technique, using a 5mm resection loop. The uterine fundus and cornua were treated with a 5mm rollerball in all but two cases, in whom a 3mm resection loop and rollerball were used. The smaller resection loop, although theoretically safer, can be problematic as the endometrial gland depth can be as deep as 4mm and at least this depth of resection is required to ensure complete resection of the endometrium.[7] Thermal injury to glands below the 3mm depth of resection cannot guarantee their destruction.

A postal questionnaire was sent to each of the sixty-eight patients who had not subsequently undergone hysterectomy. Patients were asked to describe their menstrual cycle, menstrual blood loss, and dysmenorrhoea both prior to and following TCRE. The women were also invited to comment on the procedure and on any benefit or disadvantage they could perceive.

Six of the seven patients who failed to return a questionnaire were contacted by telephone. The seventh patient could not be contacted.

The ten patients who undergone hysterectomy were not contacted but a careful chart review was made to determine the indication for hysterectomy.

RESULTS

Sixty-one postal questionnaires were returned completed and six of the seven non-responders were contacted by telephone. The average age of the patients was 42.2 years, range 27-53.

Fifty four (80%) complained that their bleeding was excessive, thirteen (20%) complained of heavy bleeding and dysmenorrhoea. Twenty-eight had either very irregular bleeding or a cycle length of less than 24 days. The duration of bleeding was variable, but in 50 women it was more than seven days.

Six women (10%) had no improvement in their bleeding pattern. If women with slight staining are included in the group reporting amenorrhoea, 39 (58%) were rendered amenorrhoeic. Twenty-two (32%) had regular light periods.

Of the thirteen women who had pain as a significant symptom, eight (62%) experienced either minimal or no dysmenorrhoea post-operatively. Ten (15%) women reported that they experienced significant dysmenorrhoea following the procedure, five of whom had not reported pain pre-operatively. One of these women claimed to be in constant pain; this was not due to haematometra.

In one patient (who had undergone two previous Caesarean sections), uterine perforation occurred as the scope was re-introduced following the resection; she underwent hysterectomy. No extra-uterine visceral damage was noted. In two women intra-operative haemorrhage was not easily controlled with diathermy; however, uterine tamponade with the inflated balloon of a Foley catheter for 24 hours was sufficient to achieve haemostasis.

Twenty-seven women went home the day following surgery, 19 on day two, ten on day three; four patients remained in hospital for four or more days. Of these, one patient was transferred to a medical ward following a cerebrovascular accident. She had been on warfarin pre-operatively and was considered unfit for hysterectomy. Review of the post-operative time spent in hospital shows that latterly almost all patients were discharged on the first postoperative day. Earlier in the learning curve for the procedure caution dictated that patients remain in hospital under supervision for rather longer.

Ten women had undergone hysterectomy at the time of writing, one intra-operatively and nine others subsequently. Two of the nine complained of persistent heavy vaginal bleeding, three of dysmenorrhoea and four of dysmenorrhagia. Histopathological examination of the hysterectomy specimens showed adenomyosis in three, endometriosis in two, fibroids in two, tubal adhesions in one and hydrosalpinx in one. The length of time for TCRE to hysterectomy ranged from eight to 30 months. All of these patients were considered to be dissatisfied, although only two were dissatisfied because of continuing heavy bleeding. Two others were on the waiting list for hysterectomy, one who had severe constant pain, and one who complained of dysmenorrhoea.

Outcome was not dependent on duration of menses, duration or severity of bleeding or regularity of the cycle.

DISCUSSION

Minimally invasive surgical techniques have become increasingly popular with the reduction

in the number of hospital beds available and the demand for increased turnover of patients. Patients are more aware of minimally invasive surgical techniques and many inquire regarding this. Since its development through the 1980s the technique of transcervical resection of the endometrium has remained basically unchanged. The introduction of fluid management systems (costing around £10,000) has been claimed to reduce fluid deficits,[8] but careful control of infusion pressure and shorter operating times achieved by experienced surgeons limit the need for such technology. No cases of fluid overload (deficit >1.5L) were noted in this series when a gravitational infusion system was used.

Strict selection criteria have been shown in a number of studies to be of paramount importance, not only in achieving good objective results, but also in achieving high patient satisfaction rates. Selection criteria include: completed family, a wish to avoid or contra indication to hysterectomy, no coexistent gynaecological pathology, and a normal smear within three years.[3] The presence of dysmenorrhoea has not been shown in other studies to affect the degree of patient satisfaction.[2,6] Counselling regarding dysmenorrhoea post-operatively is also of importance. Eight of 13(61%) women in the series above were considerably relieved of pain; however, five of 48(11%) who had not experienced dysmenorrhoea pre-operatively reported this symptom post-operatively.

Thorough counselling of women is needed pre-operatively to minimise post-operative disappointment in the bleeding pattern achieved. Hysterectomy remains the only surgical technique which guarantees amenorrhoea; however, hysterectomy does not guarantee patient satisfaction. Minimally invasive surgical techniques should not, therefore, be offered to women who are adamant that amenorrhoea is the desired result. Results from follow-up for more than four years can now be reported to women requesting TCRE at this unit, and if truthful estimates of likely outcomes are given, dissatisfaction is less likely. Results from the most-recently performed procedures indicate that with experience in operating, outcomes can be expected to improve.

Complications occur intra-operatively and post-operatively. In this series there were two cases of mild intra-operative haemorrhage, one case of uterine perforation and no cases of fluid overload. This compares favourably with other authors.[9,10] Both cases of mild haemorrhage were controlled by tamponade with a Foley catheter and neither patient required blood transfusion. Post operative endometritis did not occur in this series but is a recognised complication.[11]

Concerns regarding long-term healing have been voiced. Most benign is the recurrence of abnormal bleeding, which has been quoted at 22.5% of patients at five years; however, more sinister is the possibility of the masking of malignant change in residual islands of endometrium.[12]

Transcervical resection of the endometrium is a safe and effective treatment for menorrhagia. It can be offered to women as an alternative to medical or more radical surgical therapies and is not associated with the side effects of hormone preparations and long-term morbidity of invasive surgery. Unlike medical treatments, it can achieve a long-term cure. With shorter hospital stay than for hysterectomy, and more rapid convalescence, the financial cost to both hospital and the patient is potentially considerably less than for invasive procedures. The quality of the service provided at the Belfast City Hospital is comparable to that of teaching units elsewhere, with 15% hysterectomy rates following conservative surgery.

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The effects of depression awareness seminars on general practitioners knowledge of depressive illness

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SUMMARY

The Royal Colleges of Psychiatry and General Practice wish to increase knowledge of depressive illness among patients and professionals. This study reports the results of a series of depression education seminars in a Health Board in Northern Ireland. Seminars lasted 2½ hours and included didactic teaching and interactive case management vignettes. 88 general practitioners took part (39% of those eligible). Seminars increased knowledge as measured by questionnaire immediately, but this did not appear to be sustained at one year, when compared to a group of general practitioners with no access to such seminars. Educational programmes as described do not appear to have a sustained effect on general practitioners' knowledge of depressive illness.

INTRODUCTION

The Defeat Depression Campaign is being organized in the UK by the Royal College of Psychiatrists in association with the Royal College of General Practitioners. Its aim is to improve knowledge of depressive illness and its treatment among patients and professionals. There is evidence (on a small scale) that such campaigns can lead to increased detection, treatment and reduced disability from depression[1]. There has been limited published evaluation in the UK, with some work suggesting little impact on general practitioners' clinical practice in particular.[2] In the USA intensive programmes of education have been evaluated by questionnaire with positive and substantial results.[3] The aim of the present study was to assess whether a series of depression awareness seminars made available across a specific area to general practitioners would increase knowledge of depressive illness, and whether this was sustained at one year.

SUBJECTS AND METHODS

The Northern Health and Social Services Board (NHSSB) has a population of 406,000 served by 245 GPs. A series of eight seminars was held throughout the Health Board. As many general practitioners as possible were contacted and invited to seminars in their area. Twenty general practitioners were not canvassed as they had recently received teaching on depression.

Each seminar lasted 2½ hours, comprising 1 hour of didactic teaching based on the consensus statement from the Royal Colleges[4] and 1½ hours of interactive case vignettes discussing common diagnostic and management problems.

Knowledge of depressive illness and its management was assessed by a twelve item true or false questionnaire given before the seminar (Table) and repeated afterwards. General practitioners who took part in the original seminars were contacted at one year and a second twelve item questionnaire was used (to avoid practice effects, but covering the same areas). The results, at one year, were compared to a group of general practitioners from outside the area with no exposure to such seminars. This second group were also seeking to attend an educational meeting on depression and therefore showed a similar level of motivation to the study group. To ensure compliance all questionnaires were completed anonymously. Scoring was +1 for correct answers and 0 for incorrect; missing replies were treated as incorrect. Scores were normally distributed and the unpaired t-test was applied (all results two tailed).

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TABLE

First GP Questionnaire

1. Depressive illness can be diagnosed if mood and associated changes have been present for 1 week. YES/NO
2. When depressive illness is triggered by life difficulties antidepressants have no place in management. YES/NO
3. Depressive illness always presents with marked lowering of mood as the main symptom. YES/NO
4. Asking about suicidal ideas may be dangerous and precipitate suicidal actions. YES/NO
5. People who commit suicide do not tell others. YES/NO
6. Depressive illness when treated usually does not recur. YES/NO
7. Antidepressants should be used for 2 months after the patient recovers. YES/NO
8. If two antidepressants fail there is little point in trying other treatments. YES/NO
9. Compliance with antidepressant treatment is usually very good. YES/NO
10. Most people who commit suicide have personality difficulties and are not depressed. YES/NO
11. Antidepressants, when used, should always be combined with supportive counselling. YES/NO
12. Suicide is most common in middle aged females. YES/NO

Correct Answers

YES = 11

NO = 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12.

RESULTS

Of the 225 general practitioners eligible for inclusion, 88 took part in one seminar (39.1%). The mean (SD) score was 9.9(1.4) before and 10.9 (1.2) after. The range was 5-12 on both occasions. The increase was statistically significant ($t = -3.1$, $df = 174$, $p<0.002$).

44 general practitioners were contacted at one year (50% of original sample, 20% of eligible general practitioners in Health Board) and completed the second questionnaire. They were compared with 30 general practitioners not exposed to a local defeat depression seminar. There was no difference in scoring between those exposed 9.5 (1.3) and not exposed 9.5 (1.5) ($t = 0.1$, $df = 72$, $p = 0.9$) at one year follow-up.

DISCUSSION

This study attempts to recruit general practitioners into educational seminars and assess the outcome. Informal discussion suggested they would be unlikely to attend longer sessions devoted to one disorder. The findings suggest little evidence of sustained benefit from seminars as described above. The Iowa Study[3] demonstrated protracted benefits from their educational programme. This may be related to the duration of educational input (two days), or that their audience was primarily mental health professionals. The Iowa study also demonstrated a substantial attrition rate (41% were unable to be contacted or did not reply) at long term follow-up (6 months). Non-responders in this study were not significantly different on several key demographic variables from those who did reply, suggesting the high attrition rate did not bias the study.

A Swedish study,[1] carried out on the island of Gotland, did not look at knowledge gains but found intensive education on depression and suicide for general practitioners (over several days), did reduce suicide rate and days lost from work related to depression. The study was relatively small and it is unlikely the methodology could be repeated on a larger scale. Despite this it has been a major theoretical and practical underpinning of the Defeat Depression Campaign.

There are several limitations to the present study which assesses accumulation of knowledge. This may be a marker towards outcome of care but does not necessarily reflect what happens in the surgery. However it is unlikely care can be improved without knowledge gains.

There was no active randomization process as the aim was to capture as many general practitioners as possible. With regard to the immediate effects of the seminars, the general practitioners acted as their own controls. At one year a group of equally motivated general practitioners were available for comparison.

The questionnaires were answered anonymously to encourage forthright replies; this prevents any

sub-analysis with regard to age, psychiatric experience etc.

The questionnaire has been used with other mental health practitioners and can discriminate between different groups. The questions are based on Royal College recommendations[4] and related documents. The author was involved in training at all seminars. On certain occasions this was shared with colleagues. Training material, slides etc, were standard at all meetings, and based on national recommendations.[4]

It is unclear how questionnaire assessment relates to clinical practice. However, the scores in this study do suggest scope for improvement in knowledge of depressive illness. The questions posed should not be considered esoteric. Most general practitioners' education follows a format similar to the seminars provided; reassessment of this method may be necessary.

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A prospective study of the process of assessment and care management in the discharge of elderly patients from hospital

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SUMMARY

Assessment and care management (ACM) of elderly patients prior to discharge from hospital has been in place since 1993. It involves a complex multi-disciplinary assessment of needs which may delay discharge from hospital. We prospectively studied the process of ACM in a group of patients discharged from hospital over a three month period. The times taken for completion of the necessary reports, and any delays in the process were recorded. The times of each individual step in the process were correlated to overall length of stay and to the length of the care management process. The effect of intercurrent illnesses or other delays was studied. Of the available sample (n=83), 16 patients died and two required long term hospital care. The median length of stay of the remainder (n=65) was 36 days (range 5-149 days). The median time from the start of the ACM process to discharge was 22 days (0-89 days). The strongest correlation with total length of stay was the time from admission until ACM commenced (ρ=0.661, p<0.0001). The time spent in the ACM process was related strongly to the time taken for the Care Manager to process the applications (ρ=0.682, p<0.0001). Delay was recorded in 17 (24%) cases, resulting in an increased length of stay (p<0.001). While care management may help in appropriate placement after hospital discharge, these results suggest that it is prone to delays outside the hospital setting. Such delays result in patients waiting in hospital for care packages to be set up in the community. This has implications for acute hospital services.

INTRODUCTION

The rise in the number of elderly people and demand on institutional care were factors leading to the introduction of the Community Care Act (1990),[1] and care management in 1993.[2] The aim of these was to fit placement according to need, rather than the availability of services, and to prevent inappropriate placements in institutional care from community and hospital. Speedy and appropriate discharge from hospital was to be achieved by comprehensive assessment, and appointing a Care Manager to oversee the process of care management (ACM). The Care Manager was empowered to purchase appropriate services, and had the final say in placement after discussion with the patient, relatives and hospital staff.

The hospital based social worker collates medical, nursing, physiotherapy and occupational therapy reports and forwards them to the Care Manager appointed for the patient. This can take up to five hours of social worker time.[3]

There is little evidence that the introduction of ACM has resulted in shorter length of stay,[4, 5] with one study showing a 52% increase in length of stay.[6] However all of these studies were

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retrospective. We have carried out a prospective study of elderly patients in a district general hospital. Our aim was to quantify the time spent in different aspects of the ACM process and to identify possible delays.

METHODS

The study was carried out on elderly patients, referred for assessment and care management by their consultant, in the geriatric, medical, and surgical wards of a district general hospital (Ulster Hospital, Belfast). Patients were assessed between October 1994 and January 1995, by one of the authors (FT), who followed them up weekly until discharge. Any inter-current illness or other obvious cause for delay was noted. Written reports from the doctor, named nurse and therapists concerning each patient were sent to the Care Manager by the hospital social worker.

Times taken to complete each stage of ACM from admission to discharge were recorded. General demographic data, home circumstances, medical diagnoses, Barthel activities of daily living score[7] (maximum independence = 20), and abbreviated mental test score[8] (best score = 10) were stored on a computerised database.

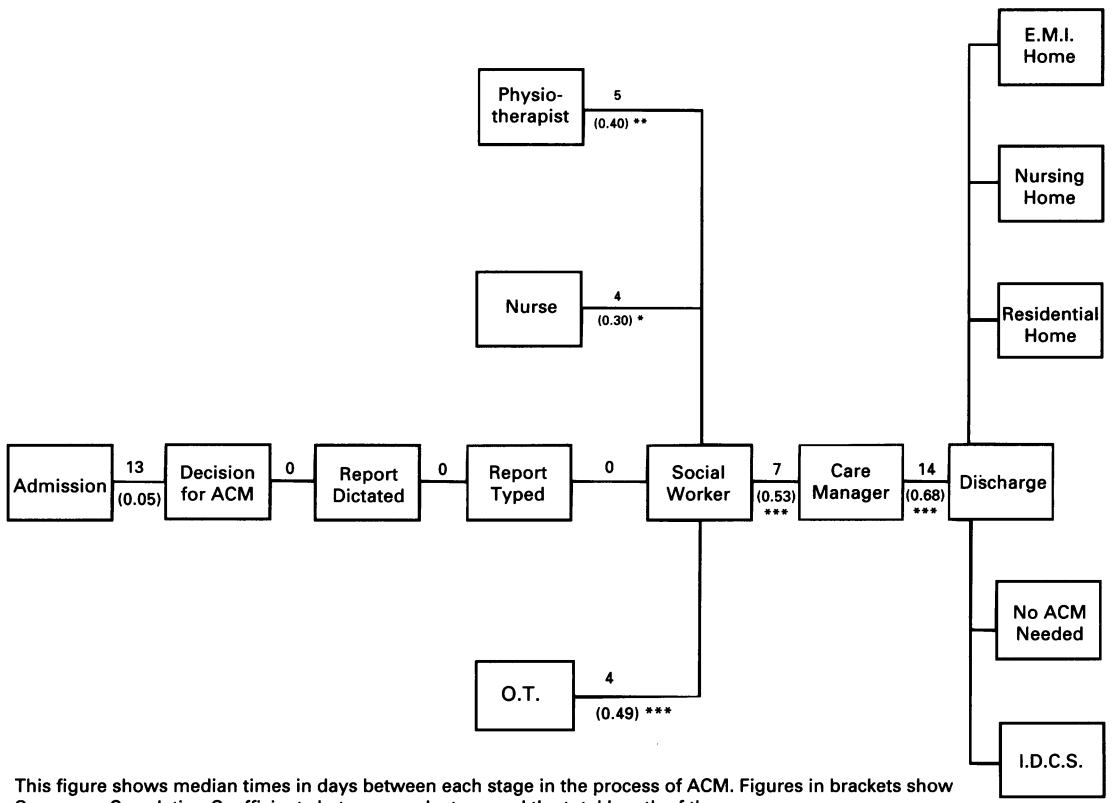
Patients were divided into groups based on the final place of care: (a) home on the intensive domiciliary care scheme (continuing care at home – IDCS), (b) ordinary private nursing home, (c) elderly mentally-infirm private nursing home (EMI), (d) residential home (private and statutory), (e) discharged without care management (including sheltered accommodation), (f) died in hospital or required continuing care in hospital.

Most data were analysed non-parametrically. Spearman rank correlation was used to compare relationships between two continuous variables. If significant, these were submitted to stepwise multiple regression analysis.

RESULTS

Eighty-three patients were entered in the study. Two patients had not been discharged four months after the conclusion of recruitment, and were designated as requiring long-term hospital care. They were not considered further. Sixteen patients died before discharge. Sixty-five cases (46 female) were subjected to analysis. The mean age of the patients was 81.7 years (range 65.7-101.8 years, median 82 years).

Figure. **The process of Assessment and Care Management (ACM)**



This figure shows median times in days between each stage in the process of ACM. Figures in brackets show Spearman Correlation Coefficients between each stage and the total length of the process.
Significance values: * p<0.05, ** p<0.01, *** p<0.001.

The structure of ACM, and the median time taken in each step of it are shown in figure. The destinations of patients in the available sample are summarised in Table 1. There were no significant differences in overall length of stay, time taken for ACM, sex or mental test score, between patients discharged to different types of accommodation. The only significant difference across the destinations was that Barthel ADL scores were higher in the IDCS group (median 13) compared with nursing home group (median 7, p = 0.003), although those who went to nursing homes tended to be older (median age 84 versus 77 years).

TABLE I

Outcome. median length of stay and the median time in days taken for Care Management of patients entered in the study.

Destination/Outcome	*Number (%)*	*Length of Stay (Range)*	*Time Spent in ACM (Range)*
E.M.I.	3 (4.6)	28 (27-82)	25 (23-68)
I.D.C.S.	13 (20)	43 (5-97)	21 (0.45)
Discharged without ACM	12 (18.5	23* (7-48)	13.5* (7-23)
Private Nursing Home	33 (50.8)	39 (16-149)	27 (3-89)
Residential Home	4 (6.1)	34.5 (21-54)	27 (15-38)
Total	65 (100)	36 (5-149)	22 (0-89)

• $0.01<p<0.05$ with respect to private nursing home group.

The total length of stay correlated strongly with the time between admission and the decision to submit the patient to ACM ($\rho=0.661$, $p<0.0001$ Spearman rank correlation). It also correlated with the length of time spent in the process of ACM ($\rho=0.705$, $p<0.0001$). Within this period it was most strongly correlated with the time between the Care Manager receiving the relevant documents to the discharge from hospital, ($\rho=0.464$, $p=0.003$). The time taken to prepare all the reports for the social worker was weakly correlated with the length of stay ($\rho=0.327$, $p=0.025$).

The time spent in ACM was related to the time between the Care Manager receiving the documents and discharge. It was also correlated with the times taken to produce the various reports and the time taken to get these reports to the Care Manager (figure). Multivariate analysis showed that the only stages of the process related significantly ($p< 0.05$) to the time spent in ACM were: (a) between the Care Manager receiving the documents and discharge, (b) the time between the social worker receiving all reports to the papers being passed on to the Care Manager, and (c) the time for the nursing staff to get their reports to the social worker.

Delay was recorded in 17 (26%) of patients, in all cases for administrative reasons (Table II). The presence of delay resulted in an increase in the median length of stay from 35 to 46 days, ($p<0.001$) and an increase in median length of care management time from 17 to 37 days, ($p=0.0001$) compared to those without delay. In patients subject to delay, the median time for all completed reports to arrive with social worker was increased from one day to seven days ($p=0.001$). The time from the social worker receiving all the reports to the papers being passed to the Care Manager increased from 6.5 days to nine days ($p=0.04$). The time from the Care Manager receiving all the documents until discharge increased from 12 days to 18.5 days ($p=0.008$).

Fourteen (21.5%) of the patients were recorded as having suffered an inter-current illness. The group suffering an illness had a median length of stay of 46 days, ten days longer than those who did not. This was not statistically significant.

TABLE II

Reasons given for delay in Care Management Process.

Age	*Sex*	*Days in ACM*	*Reason for Delay*
82	F	27	Waiting release of funds from care manager
76	F	38	No social work cover available
66	F	37	Patient did not want to go to nursing home despite disability
89	F	89	Doubt in ACM team as to appropriate placement
82	F	34	Relatives disagree with recommendations of ACM team
74	F	27	Relatives disagree with recommendation of ACM team
86	F	41	Patient changed her mind
82	M	50	Patient's wife changed her mind about patient coming home
75	F	19	Awaiting home oxygen
84	F	21	Awaiting Stairlift installation
91	F	29	Relatives unavailable for contact for 6 days
83	F	37	Relatives live overseas
85	F	31	Relatives unavailable for contact for 4 days
87	F	7	No places in home of choice
65	F	45	No places in home of choice
84	M	21	No places in home of choice
78	F	68	No places in home of choice

In 20 (30.7%) cases the original suggestion of discharge destination by the consultant was different from the eventual destination of the patient after discharge. Of ten patients where the suggested destination after discharge was nursing home, four were discharged to both residential home and home without need for ACM, and two were accepted into the IDCS scheme. Of the seven patients recommended for discharge to IDCS, four went to nursing home, three went home without ACM and one went to residential accommodation. In the remaining two cases (one initially EMI home and one residential home) both went to nursing home. A difference between suggested destination and eventual destination was not significantly related either to length of stay or to time in ACM, but was related to time from admission to commencement of ACM (no difference 21 days, difference 11 days $p<0.001$).

The 16 patients who died were all submitted to ACM and had a suggested destination after discharge (13 to nursing home, 2 to IDCS, and 1 to residential home). On reviewing their casenotes it was felt that in seven (43%) of these cases death was likely within a short period of time, in the others the deaths were sudden and unexpected.

DISCUSSION

The development of structured assessment on patients being discharged from hospital is a welcome development. Studies of the situation before April 1993 have shown a considerable degree of inappropriate placement.[9, 10] This paper describes a prospective study of the process of ACM before discharge from hospital. We aimed to identify delays in the process, and to point the way to a more efficient discharge system.

Compared to a previous retrospective study of hospital discharges, carried out in the same unit,[6] there has been improvement in the mean length of stay (41.1 versus 59.8 days), but no improvement in mean time under care management (22.6 days in previous study versus 24.2 days in this). It would appear from the present study that there are many administrative delays. Some were due to patient choice. Others were due to difficulty in meeting relatives, or having community services organised.

Hospital consultants may attempt to compensate for delays by starting the ACM process as soon as possible. The mean time between admission and referral for care management in this study was 16.8 days, compared with a previous study, (37.2 days).[6] However, this study suggests that early referral carries the risk of inappropriate recommendation for placement. This undermines the benefit of quick referral to the ACM process. Thus relatives may have been interviewed and may have gone to visit several nursing homes, only to see the patient improve so that such a level of care is no longer necessary. The finding that seven deaths, which may have been expected

according to the hospital notes, occurred in patients who were in ACM gives rise to some concern. This suggests a degree of inappropriate referral. Further research on predicting which patients will ultimately require care management would he useful.

For practical purposes the time intervals in the ACM process were broken into blocks for analysis. These blocks of time may not reflect the unique circumstances that each patient faces during an illness. This study was also limited by the failure of the care managers to cooperate with it. Once the papers had gone to the "black box" of the care manager's office, we have no indication of their progress thereafter. Further studies of this aspect of ACM with the involvement of the care managers would be of value.

This study would suggest that hospital wards are becoming more efficient at early discharge, but the length of the ACM process has not shown any significant improvement. There are still many reasons for an unnecessarily long wait in hospital. Many of these are outside the control of the hospital staff. Further research on the community provision of services will be needed to identify ways of dealing with this ongoing problem.

ACKNOWLEDGEMENTS

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Audit of the management of spontaneous pneumothorax

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SUMMARY

This audit suggests that clinical practice in the management of spontaneous pneumothorax differs from guidelines issued by the British Thoracic Society. In particular simple aspiration was attempted in only seven out of 65 patients and clamping of an intercostal chest drain occurred in 12 out of 50 cases. Junior medical staff require more training in intercostal drainage.

INTRODUCTION

In July 1993 the British Thoracic Society (BTS) issued guidelines for the management of spontaneous pneumothorax[1] which stimulated much dis-cussion, and it was suggested that they could be used as a basis for audit. We performed such an audit in a district general hospital with no resident respiratory physician or thoracic surgeon. Patients with a diagnosis of spontaneous pneumothorax were admitted to a general medical ward under the care of the consultant physician on call. A specialist respiratory centre including a thoracic surgery department is located within eight miles of the hospital where the audit was performed.

METHOD

The cases were identified from the coded diagnosis of spontaneous pneumothorax, and information was recorded on a detailed proforma. We found 65 cases in the designated time period between April 1994 and April 1996. It was predicted that a hospital with a catchment population of 200,000 would treat 25 such patients each year so our figure of 65 for a two year period with a catchment of 270,000 is typical. The auditors examined the casenotes and radiographs of each patient.

OBSERVATIONS

Of the 65 patients, 49 had their first pneumothorax, 10 their second, four their third and two were admitted with a fourth pneumothorax. There was no history of underlying disease in 40 cases. The pneumothoraces were small in 26 cases and required observation only. The remaining 39 patients were treated by a drainage procedure.

Simple aspiration had been attempted in only seven patients. It was successful in only two cases and the other five eventually were treated by intercostal drainage. There were no cases of tension pneumothorax. Of 18 patients with no underlying disease who required a drainage procedure only two had aspiration attempted. The majority of these patients had pneumothoraces which would have seemed suitable for aspiration. Of the 19 patients with underlying lung disease who required a drainage procedure aspiration was attempted in five cases.

There were 37 patients who required intercostal drainage, and with re-siting a total of 50 chest drainage procedures were performed. There was no example of delaying a procedure until a more experienced person was available. There was no documentation of trocar-use, and all drains were inserted in the triangle of safety. This is bounded anteriorly by the posterior pectoral fold, posteriorly in the mid axillary line and inferiorly a horizontal line from the nipple approximates to the level of the diaphragm. In the group of 37 patients treated by intercostal drainage 27 were successful; 23 that were associated with a clinical problem. In 13 of these cases there was either persistent air leak or failure of the lung to expand despite an adequate chest drain, and thoracic surgery referral was necessary. The remaining 10 drains were either inadequately positioned or

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subsequently lost their position. One of these cases was associated with severe subcutaneous emphysema.

Only a minority of chest drains were inserted by physicians despite all cases eventually being admitted medically. Of the 10 drains that were inadequate all were inserted by SHOs (surgical 5, A and medical 1 and 2 not specified.) In 32 cases local anaesthetic cover only was given during intercostal drainage (18 of these had underlying lung disease and 14 did not.) Opiate analgesia was given to 13 patients in addition to local anaesthetic. One patient had midazolam and local anaesthetic, 2 patients had all three (opiate, midazolam and local anaesthetic.) Respiratory distress with severe breathlessness and hypoxia occurred in 14 cases, all of whom had underlying disease. Midazolam was not given to any patients with respiratory distress. There was no recorded incidence of premedication with atropine.

Suction was applied in seven cases to attempt reinflation but was not successful. Clamping was performed in 12 of the 50 drains.

There were 15 patients referred to thoracic surgery for further management. Of these, 12 cases were accounted for by either persistent air leak or failure of the lung to re-expand despite an adequately-sited chest drain. The remaining three consisted of two patients with their third pneumothorax and one patient with a fourth. Twelve patients had a surgical procedure performed, with equal numbers having pleurodesis and pleurectomy (plus or minus bullectomy or oversewing of bullae). One patient was managed successfully with an additional chest drain and the remaining two consisted of one patient who died of bronchopneumonia and another who was in the third trimester of pregnancy. She was managed with a flutter valve, and surgery was deferred until after delivery.

Only seven patients were not followed up and all those who left hospital with a residual pneumothorax were reviewed at least once.

COMMENTS

The publication of the BTS guidelines was associated with some controversy and it is useful to observe how closely clinical practice is adhering to the recommendations. We feel that our results are likely to be typical of other district general hospitals but this will require verification. An audit of the initial management of spontaneous pneumothorax performed in a large Scottish teaching hospital showed similar findings with regard to a low rate of attempted aspiration and the practice of clamping.[2] The BTS guidelines commented that "it is common for intercostal tubes to be inserted by inexperienced junior doctors, to fit poorly, to leak, and to become dislodged or infected; there is continuing confusion about suction, clamping, management of surgical emphysema and when to seek specialist advice."[1] Simple aspiration was advocated strongly as an effective and well tolerated treatment for spontaneous pneumothorax. It is desirable because there is less associated morbidity than with intercostal drainage and if successful is associated with a shorter hospital stay and a more rapid return to work. Despite this continuing evidence for the effectiveness and convenience of simple aspiration [3,4] it was rarely performed. This may be due to a less successful experience with simple aspiration in practice than has been suggested.

There was a strong negative reaction to the use of trocars following the BTS guidelines[5] and blunt dissection was performed in all cases in this series. It is widely accepted that the clamping of chest drains is a dangerous practice,[6] potentially converting simple pneumothoraces to life threatening tension pneumothoraces.[7] It is alarming that 12 out of 50 drains were clamped. This audit would appear to support the impression that the application of suction is of little value outside specialist centres. If suction is to be used it must be at high volume and low pressure. A low volume pump should not be used as it will not be able to handle a large air leak and will allow air to accumulate, worsening the pneumothorax.[8]

Attention was also drawn to the fact that an agitated patient may be hypoxic[9] and care should be taken with sedation, especially if the patient has underlying lung disease. This must be balanced against the fact that intercostal drainage is a traumatic and unpleasant experience. It was appropriate that midazolam was not administered to patients with respiratory distress.

We observed that over 75% of intercostal drains were inserted by doctors of SHO grade. It was never recorded if this was under supervision. It is not surprising that all the inadequate procedures were performed by the most junior staff. The referrals to thoracic surgery seemed appropriate in all cases.

RECOMMENDATIONS

It would be useful to see data from other comparable hospitals and indeed from those with a respiratory specialist on site. On the basis of this audit we would recommend that aspiration should be more commonly performed, especially in patients without underlying lung disease. It should be emphasised that chest drains should not be clamped and suction avoided outside specialist centres. It may be possible in non urgent situations to delay intercostal drainage until a more experienced doctor is available. Simple aspiration is much less likely to be successful in patients with underlying lung disease[10] and it could be argued that this group of patients should be managed by intercostal drainage in the first instance. We have noted that spontaneous pneumothorax is initially more often managed by surgeons than physicians in our hospital which raises the additional controversy of whether or not it should become a surgical admission. The practice of blunt dissection is certainly more familiar to surgeons than physicians. The subsequent management of patients with underlying lung disease may be more appropriate in a medical unit or one with a medical input, but patients with primary spontaneous pneumothorax could be admitted surgically. If spontaneous pneumothorax is to continue as a medical admission junior medical staff should receive more formal training in the procedure of intercostal drainage.

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An audit of acute psychiatric admission bed occupancy in Northern Ireland

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SUMMARY

The Northern Ireland Section (Irish Division) of the Royal College of Psychiatrists were requested to investigate apparent increasing pressures on acute psychiatric beds. Information on bed occupancy and associated service activity was collected by clinicians on site in every psychiatric unit in Northern Ireland over the past eight years. Three separate years (1987, 1991 and 1995) were studied. Bed occupancy rose across these three years. There was an associated reduction in the number of acute psychiatric beds, reduction in adult continuing care beds, increased recorded referrals to psychiatric units and evidence of considerable numbers of new long-stay patients and difficulties with community placements. Acute bed occupancy in Northern Ireland is high, frequently over 100% and rising. Occupancy rose between each of the years studied. The problem is not confined to urban areas and several associated service factors may be contributing. Without change, acute bed provision will inevitably fail to match mental health needs.

INTRODUCTION

Acute admission beds for patients with psychiatric illness remain essential for the assessment and treatment of the most severely mentally ill. Demonstration projects have shown that with considerable extra resources, reduction in the number of acute admissions and their duration is possible.[1,2] These projects have not been evaluated over prolonged periods in standard clinical settings, without additional resources, in the United Kingdom. They are limited to large urban centres.

Concern has been expressed about the reduction in numbers in acute psychiatric beds throughout the United Kingdom.[3,4] Anecdote originally suggested this was most severe in London. Subsequent research, from London, has shown excessive levels of bed occupancy during the early 1990s[5] which have continued to rise.[6] Reductions in the provision for new long-stay patients have been noted and their impact on service provision discussed.[7,8]

Northern Ireland is served by six large mental hospitals and several district general hospital facilities. All have acute or short-stay psychiatric beds. The large mental hospitals also have a varying number of continuing care (non-dementia) psychiatric beds for patients requiring longer-term hospital care. The latter group comprises a declining number of patients who have spent long periods in hospital and a group of more recent admissions who are unable to survive outside hospital because of complex disabilities associated with their psychiatric disorder (new long-stay).

There have been substantial reductions in both acute admission beds and adult (non-dementia) continuing care beds over the last 15 years. As in Great Britain, concern had been expressed that units are frequently full and it was not possible to arrange urgent admissions. Anecdotally pressures were thought to be greatest in Belfast. The above situation pre-dated suggestions from the Department of Health and Social Services

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Northern Ireland (DHSS NI) to further reduce bed day occupancy by 20%.

The Northern Ireland Section (Irish Division) of the Royal College of Psychiatrists set up an Acute Bed Project Board, with representatives from all hospitals with acute psychiatric admission beds (excluding addictions) to assess the extent of bed occupancy past and present. In addition, factors which were considered possible influences on bed utilisation were investigated, such as referral rates, numbers of new long-stay patients in acute psychiatric units and difficulty obtaining community placements.

METHODS

A structured questionnaire was sent to all members of the Project Board. It was decided to examine bed occupancy in 1995, 1991 and 1987. Four-year intervals were felt likely to allow demonstration of any trends.

Information requested included numbers of acute functional psychiatric beds in each year, whether the service was sectorised, and data relating to bed occupancy for three months (February, June and October) in each of the above three years. These months were chosen to observe any seasonal trends. Addiction, child and adolescent beds were not included.

The following definitions were used:

Inpatient Days – Number of inpatients daily summated over one calendar month.

Leave Days – Number of patients on leave daily summated over one calendar month.

Total Bed Days – Number of acute admission beds multiplied by number of days in one calendar month.

Bed Occupancy/Month – $\frac{\text{Inpatient + leave days}}{\text{total bed days.}}$

Percentage Occupancy – Bed occupancy/month x 100.

Each member of the Acute Project Board was asked to review all inpatients in acute beds on 28 March 1996. Data on new long stay, those unable to be found community facilities, and those where specialist inpatient facilities might possibly have been more appropriate were recorded.

In addition, the numbers of adult (non-dementia) continuing care beds at hospital sites were requested for the years 1987, 1991 and 1995. The total number of psychiatric referrals to hospitals for the above years was also sought. Some additional information was obtained from DHSS NI sources.

Descriptive statistics are used to report data.

RESULTS

Acute Bed Occupancy

Returns from all 13 hospital units showed that in 1996 there were 616 acute admission beds. This represents 0.37/1000 for Greater Belfast, 0.38/1000 for outside Belfast and Northern Ireland as a whole.

Data for each month studied is presented in Table 1. Information is given for Belfast and hospitals outside Belfast separately. However, there was no difference between urban and rural hospitals.

The bed occupancy for all units in Northern Ireland combined is shown in Figure. Occupancy rose in each study-month progressively across years. The acute bed occupancy ranged from 98%-105% in the three months studied in 1995. Data was available for seven hospitals in 1987, nine in 1991 and 13 in 1995.

TABLE I

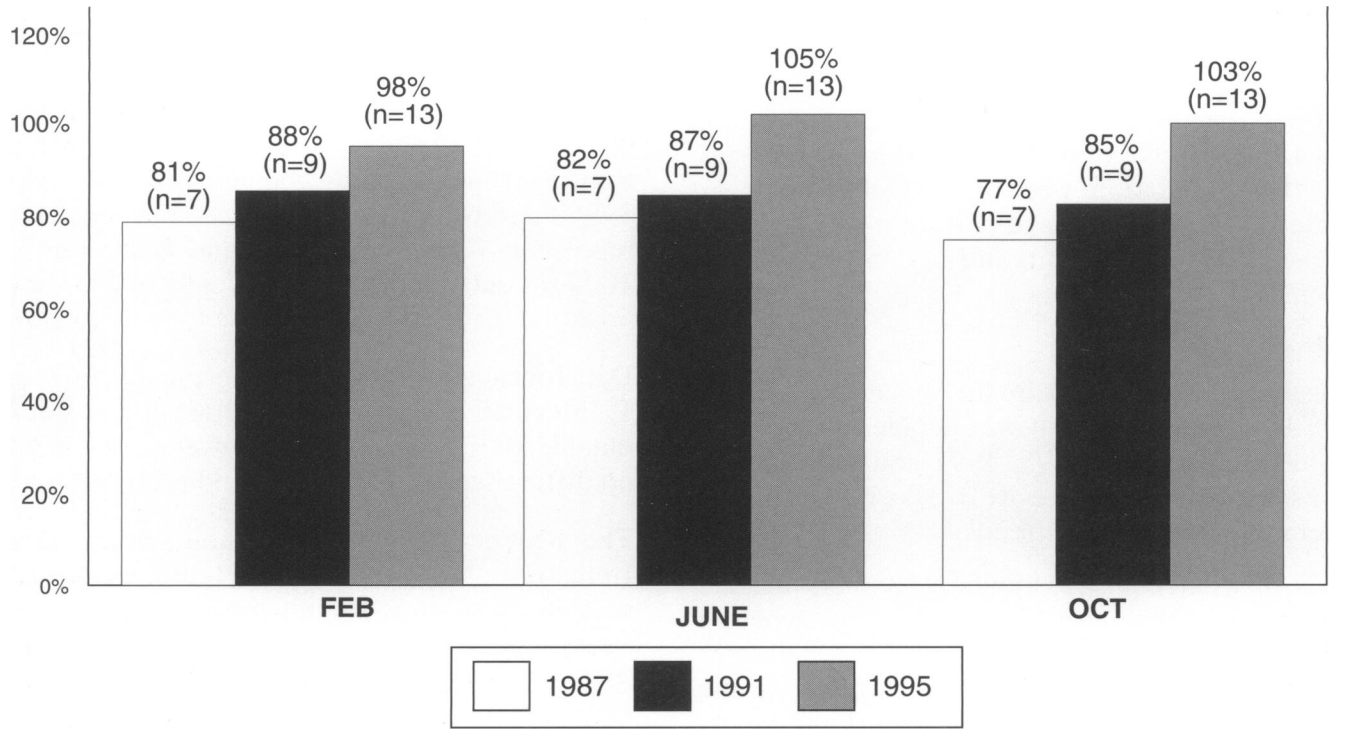
Percentage occupancy for Acute Psychiatric Beds in Northern Ireland

		Belfast	*Outside Belfast*
February	1987	90% (n=1)	79% (n=6)
June	1987	89% (n=1)	81% (n=6)
October	1987	90% (n=1)	75% (n=6)
February	1991	94% n=2)	87% (n=7)
June	1991	88% (n=2)	87% (n=7)
October	1991	99% (n=2)	77% (n=7)
February	1995	98% (n=3)	98% (n=10)
June	1995	112% (n=3)	104% (n=10)
October	1995	98% (n=3)	105% (n=10)

n = number of psychiatric units for which data is available

Figure

Percentage Occupancy for Acute Psychiatric Beds in Northern Ireland
(All Units)



n=number of psychiatric units for which data is available

TABLE II

Inpatient Census on 28 March 1996

	Belfast	*Outside Belfast*	*Northern Ireland*
	(133 Beds)	(483 Beds)	(616 Beds)
Patient admitted > 6 months	7	33	40
Patients < 18 years old	5	9	14
Patients with alcohol or drug use only	10	21	31
Patients with head injury only	0	7	7
Patients awaiting community placement	5	25	30
Patients admitted as no bes available in another hospital	4	2	6
Total	31	97	128
	(23%)	(20%)	(21%)

Inpatient Census of Acute Admission Beds

Table II gives the categories in the inpatient census on 28 March 1996. On the day chosen, these groups accounted for 21% of adult acute psychiatric admissions in Northern Ireland. Patients present for over six months and those where community placement was unavailable, occupied 11 % of available acute admission beds.

Referrals to Psychiatric Services

Only four hospitals were able to obtain data for the three study years. Five hospitals had information for the years 1991 and 1995. Referrals increased by over 100% between 1987 and 1995 for the four hospitals studied and by 49% between 1991 and 1995 for the larger sample. The referral numbers are shown in Table III.

TABLE III

Referrals to Psychiatric Services

	Data for all years (n=4)	*Data for two years (n=5)*
1987	1105	
1991	1577	2163
1995	2320	3224

Adult Continuing Care Beds

Only six of the 13 hospitals ever had adult continuing care beds on site. Of those six hospitals, four were able to detail number of beds. They declined from 858 beds in 1987, 649 in 1991 to 369 in 1995, a total reduction over the period of 57%.

DISCUSSION

The main finding of the acute bed project is a steady increase in acute psychiatric bed occupancy throughout Northern Ireland (see Figure) over the last eight years. Occupancy figures are now frequently over 100%. The rise in occupancy is not restricted to the larger urban setting.

One advantage of this study is the collection of data locally by clinicians using agreed definitions. Also, as there are no private psychiatric facilities in Northern Ireland, no patients will be lost via extra-contractual referrals to the private sector. A limitation of the study is that the occupancy data was obtained retrospectively and not all hospitals were able to furnish complete data. However all individual hospitals showed increased bed usage over the period. The study design make it unlikely that the results are occurring by chance.

It is possible that those hospitals without data for the earlier years could bias the results. For this reason the percentage bed occupancy was calculated for those hospitals with information at all time points in the study (n=7). The results were essentially the same with a rise in percentage bed occupancy to over 100% by 1995 in all of the months studied (February 1995=119%, June 1995=108%, October 1995=110%).

The increased occupancy supports the results of Powell et al[5] and extends them to another area of the United Kingdom using a different methodology. Of major concern is recent data by that group[6] suggesting bed occupancy in Greater London is continuing to rise. Based on their findings and the progressive rise in bed occupancy in Northern Ireland from 1987 to 1995, it is likely that increased difficulties will also occur in Northern Ireland without corrective action.

As well as defining the extent of the problem, it is also important to attempt to identify the possible reasons for its occurrence. One obvious possibility is a reduction in acute beds. This has been so in Northern Ireland. The number of short-stay (acute) beds has declined in the Province by 17% between 1991 and 1995 (DHSS NI). This is a figure not dissimilar to the increase in occupancy. As with associated service changes, it is not possible to imply direct causation. Estimates of psychiatric bed requirements have been made previously but these have not always been taken into account when planning services.[9]

Referrals to psychiatric units would appear to have risen substantially. Although these figures appear somewhat low, and poor recording may be the cause, it is likely that the systems in place have detected an increased demand. This is perhaps not surprising given educational programmes such as the 'Defeat Depression Campaign' trying to raise awareness and detection of mental illness in the community.

Occupancy presents important information on acute bed pressures but does not describe details of bed usage. Instruments for the audit of bed usage[10] allow intensive study of admissions to

acute psychiatric units. However with no funding such a device could not be used in this project. Instead, clinicians assessed inpatients on a given census date. The most striking feature was the large number of individuals who were resident for over six months or where difficulties in community placement were evident. The effects such patients have on acute units have been reported previously both in terms of their numbers[8] and the effects of failed placements on the patients themselves.[3] Smaller reductions in acute psychiatric bed occupancy may also be achieved by comprehensive specialist services for other groups. As with other areas of the United Kingdom, adult continuing care bed numbers have fallen in Northern Ireland.

Lack of beds for new long stay patients, difficulties in obtaining community placements and reduced facilities for those requiring continuing care have all contributed to this worrying situation.

Clinicians' attitudes regarding difficulties with bed shortages will be the subject of a separate report.

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Anaesthesia and the broken hearted

170th Annual Oration: Royal Victoria Hospital, Belfast 2nd October 1997

S M Lyons

I would like to thank the Medical Staff of the Royal Victoria Hospital for the honour accorded to me with their invitation to deliver the Annual Oration in this Bicentenary year. Studying the list of Orators in "The Royal Victoria Hospital – A History 1797-1997" by Professor Richard Clarke has, if anything, served to increase a natural nervousness and maybe reinforces the idea that, at my age, there is a fatalistic tendency to agree to almost any request a year ahead, aware that actuarial statistics give increasing odds against having actually to perform.

The broken hearted of my title are not for this occasion either my surgical colleagues or the administration but rather patients with significant heart disease which can be relieved by surgery. I will be describing the development of anaesthesia, and cardiopulmonary bypass which allows open and prolonged surgery of the damaged heart to be undertaken.

Selective memory fortunately ignores the tremendous surgical and anaesthetic problems which beset the early days of cardiac surgery. The 'norm' today, after the temporary cessation of the heart's action necessary for heart surgery, is survival without further damage either to the heart or other organs. The vast majority of the patients can expect to go home after operation with a renewed spring in their step and the prospect of a good long term prognosis. These may be further improved by the present positive attitudes to prevention and rehabilitation. The changes responsible for the improving picture have more often been subtle rather than dramatic.

Cardiopulmonary bypass is the system allowing the heart to be isolated from the rest of the circulation with the work of the heart and lungs temporarily taken over by a machine. Venous blood is collected from the right atrium or the vena cavae, taken to an oxygenator where carbon dioxide is removed, oxygen restored and the blood is then pumped back into the aorta. This allows the heart to be isolated from the rest of the circulatory system and produces conditions for a reparative heart operation.

The birth and developmental steps of open heart surgery were encompassed in the professional life spans of those of us early in our seventh decades. The beginnings of an artificial circulation to replace the heart in an animal and at the same time modern anaesthesia took place in the 1930s. The end of school in 1953 coincided with the first successful open heart operation in a human using a heart lung machine. Student days saw the first open heart case in the United Kingdom in 1958. Graduation in 1960 saw the beginning of open heart cases in the Royal Victoria Hospital. For me the establishment of the present Cardiac Surgical Unit in the Royal in 1968 was the year of my appointment to a career post. The evolution of the Unit finally moves me inexorably towards retirement. Whatever overview is taken, these dates simply emphasise the youth of the specially.

Many factors influence career choice. The early 1960s in this hospital were a significant time in anaesthesia. The strong base of anaesthetists, comprising Maurice Brown, Jim Elliott and Jim Reid, was catalysed by the whirlwind effect of the arrival in 1958 of Professor John Dundee and by the pioneering activities of Bob Gray in Intensive Care. My own option for specialisation in cardiac anaesthesia began at a date in 1966 when I had an interview with Professor Dundee about the future of cardiac surgery and cardiac anaesthesia.

However, it is necessary to go back a little further.

The observation that "The heart alone of all viscera, cannot withstand serious injury" is attributed to Aristotle, born 384 BC. Though it does beg the question about the central nervous system, there is undoubtedly still much truth in this statement. It is certainly widely accepted by

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the general public and was closer to the truth than a statement by Sir Stephen Paget who in 1896 stated that "Surgery of the heart has reached the limits set by nature toward all surgery, no new method and no new technique will overcome the natural obstacles surrounding a wound of the heart".[1] This type of medical misjudgement is not unique to any particular era. The Krebs Cycle, Magill's pioneer work on tracheal intubation[2] and Brock's mitral valvotomy operation each struggled in their turn for recognition. It was therefore almost inevitable that very soon after Paget's pronouncement, the first successful suturing of a stab wound of the right ventricle of the heart was achieved by Rehn.[3]

The major developments in heart surgery were dependent on the achievement of two factors:-

Firstly the perfection of a means of supporting an effective circulation to the rest of the body, allowing the heart to be isolated from its normal function and repaired.

Secondly, the understanding of the anaesthetic problems inherent in the management of the open chest and the inevitable pneumothorax of thoracic surgery.

The stimulus for many of the developments of the artificial circulation and the monitoring of the cardiovascular and respiratory parameters came from the 19th Century physiologists such as de Gallois [4] and Loebell [5] but the problems of managing the open chest were a long way from resolution and would not be solved until the much later improvements in anaesthetics and the introduction of muscle relaxants.

While the heart's action proved relatively easy to replace on a temporary basis with roller type pumps it was early appreciated that it was more feasible on a temporary basis to replace the function of both the heart and lungs. Over time three different methods have evolved to allow the temporary replacement of lung function and to reproduce the large surface area for gas exchange, imitating that which is available so efficiently in the lungs.

i) The first created a film of blood spread over a large surface area with oxygen circulating round the film, the so called screen and disc oxygenators.

ii) In the second oxygen was bubbled through the blood creating the necessary large surface area, the bubble oxygenator. This was the oxygenator pioneered by Lillehei[6] and popularised by Denton Cooley which allowed volume cardiac surgery to take place with the use of minimal blood.

iii) Thirdly the system in which gas transfer occurs into the blood across a membrane, the membrane oxygenator. This is now by far the most widely used oxygenator, being the machine of choice in over 90% of cases. It is still debatable if the membranes are in fact intact or are a refined form of bubble oxygenator.

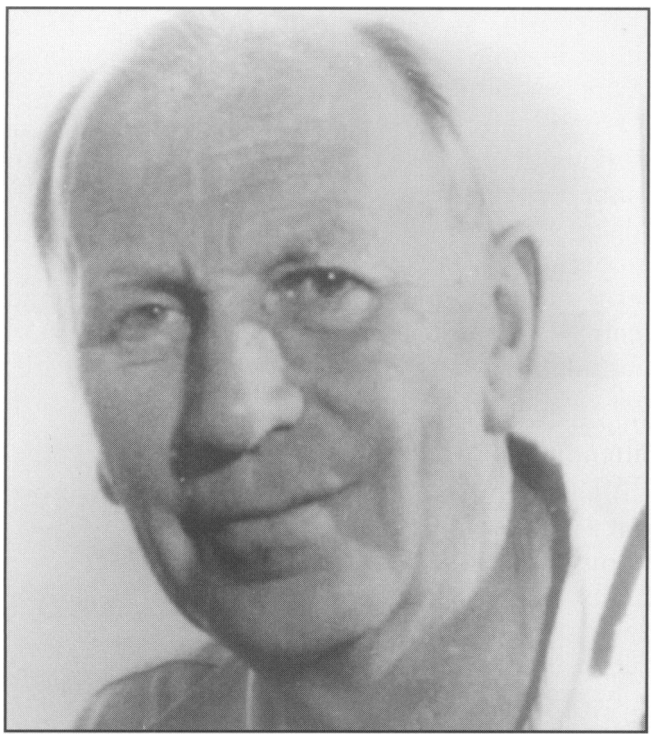
The physiologists did their work in the 19th and early 20th centuries using defibrinated blood, as they were without the means to prevent blood clotting. The discovery of heparin in 1916 by McLean,[7] when still a second year medical student, was therefore a highly significant event. Protamine became available 21 years later in 1937.

There are some other noteworthy landmarks. After the First World War Rowbotham and Magill[9] popularised the technique of tracheal intubation to secure the airway and allow extensive facial reparative surgery. This paved the way for the management of respiration in both open chest and abdominal surgery. The operation for relief of constrictive pericarditis was described at this same time and the first successful mitral valvotomy was attributed to Souttar in 1925.[10] The encouragement for the parallel technical developments to provide circulatory support gathered momentum. In 1935, Carrel and Lindbergh (the same Charles Lindbergh who was first to fly the Atlantic from New York to Paris non stop in 1925), described a perfusion apparatus which was successful in keeping an organ alive outside the body.[11] Soon afterwards Gibbon in 1937,[12] using a rotating cylinder to create a film of blood through which gas exchange could take place, described the first application of cardio-pulmonary bypass in experimental animals. The pumps used were of the roller type already mentioned, still in use today. They were designed by the then young Michael De Bakey,[13] the same De Bakey who, more than 50 years later, was in 1997 prominent in the cardiac advisory medical team for Boris Yeltsin, the Russian President.

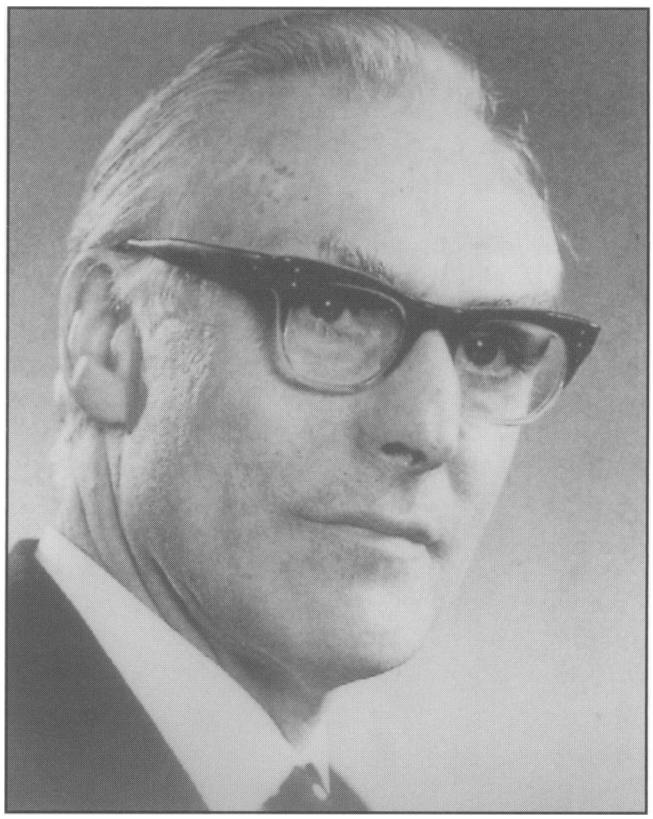
Concurrently there were rapid advances in anaesthesia, and with that the knowledge of how to manage the open chest. The early trials with

thiopentone were reported by Lundy in 1934[14] and the muscle relaxant curare was introduced by Griffith in 1942.[15] With these advances came surgical progress, and Gross in 1939[16] reported the surgical approach for ligation of the patent or persistent ductus arteriosus: modem cardiac surgery was under way.

The 1940s saw great advances in most fields of medicine, many stimulated by events of the Second World War. The treatment of casualties saw rapid improvement in surgical and anaesthetic techniques, allied to improvements in the expertise of the personnel and in overall patient management. The use of blood transfusion became commonplace and antibiotics were more widely available. These changes also had marked effects away from the battle zones. The first repair of coarctation of the aorta was described in 1945 by Crafoord[17] in Sweden and in that same year Blalock and Taussig[18] in the United States described the subclavian artery to pulmonary artery shunt for the palliative relief of cyanotic congenital heart disease. As anaesthetists became more familiar with relaxants and with the management of the open chest, Brock in London in 1950[19] was able to report the successful management of six cases of valvulotomy for mitral stenosis. This operation required only minimal interruption of the circulation, and



Tom Smiley



Maurice Brown

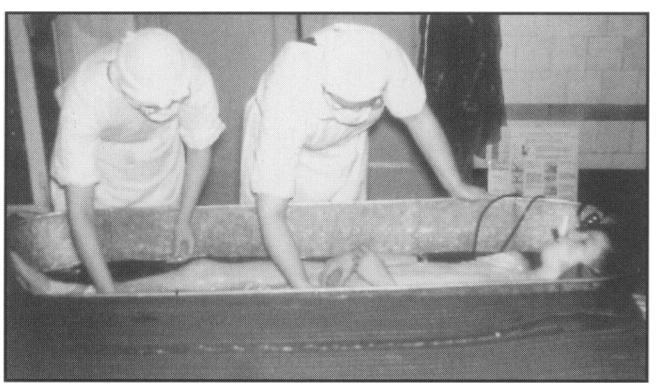
anaesthesia was with thiopentone, curare, nitrous oxide or cyclopropane, and ether. These early cases were done by the same surgeon but by three different anaesthetists so teamwork was still a far-off concept.

The Royal Victoria Hospital did not lag behind, and in 1948 the first ligation of a patent ductus arteriosus and indeed a shunt to relieve cyanotic congenital heart disease were both performed by Barney Purce. The first mitral valvotomy in the hospital is attributed to Tom Smiley in 1950 when he was still a Registrar.[20] This was the first operation of any kind on the heart itself done in Ireland and the anaesthetist was Maurice Brown.

These were great and exciting advances but they represented only the tip of the very large iceberg of potential cardiac surgery and the pressure was increasing for a total circulatory support system in order that more complex lesions could be treated. In 1951 Dennis[21] used a combined film and disc oxygenator to perform the first total human heart bypass. The patient was a six year old girl scheduled for repair of a secundum atrial septal defect. At operation the girl turned out to have a primum type of atrial septal defect and died at the end of the operation. This unfortunate episode set back the momentum for development

of the total circulatory system, and alternative methods were again sought. One such method was inflow occlusion to produce a dry heart, the system of cutting off the venous return to the heart by clamping the superior vena cava and the inferior vena cava allowing for short periods of total circulatory arrest. The time limit before cerebral damage was two minutes at normal temperature. Such a limitation was too restrictive to allow the surgeons to do anything meaningful. It was then argued that if the brain was cooled the circulatory arrest time could be prolonged. Lewis in 1952[22] used the technique of total body cooling with surface cooling, followed by inflow occlusion to successfully repair a large atrial septal defect in a five year old child. The occlusion time was 5.5 minutes and the patient survived. Thus was born the technique which was to allow a series of successful operations for the simpler defects.

In the technique of moderate hypothermia the anaesthetised patient was cooled most usually by total immersion in a bath of ice cold water. When the temperature fell to 31 or 32 degrees centigrade the patient was put on to the operating table on a cold water blanket. The timing of the rate of cooling was difficult, and ventricular fibrillation an ever-present hazard. The technique required the surgeons to be resolute, precise, speedy and able to work against great pressure. The management was tricky, and there was no room either for a mistake on their part or any error in the diagnosis. The anaesthetists had very primitive monitoring and could resort only to philosophy and prayer. The surgeons and anaesthetists of the day in the Royal were particularly successful at the technique using hypothermia. I carried out the procedure once or twice when a Senior Registrar in this hospital. Maybe one loses courage with time but the memory of those days still gives rise to nightmares.



Hypothermia
Patient being cooled in bath

As in some other centres the very success with this technique may have in the long run slowed down the developments of the more complicated open heart surgery in this hospital. When open heart surgery began, the commonest case presented was not the well understood secundum atrial septal defect but the more difficult ventricular septal defect repair. It was anatomically ill-understood, the position of the bundle was still being determined with the help of the emerging electron microscope, it was not fully accepted that a patch rather than direct suturing was essential, and the sutures then available were as binder twine compared to the sutures of today. Sadly and ironically the procedure was later often shown to be unnecessary. The use of surface cooling re-emerged in the late 1960s in small infants to facilitate a technique involving total circulatory arrest.[23] Surface cooling resulted in an evenness of cooling at all layers, and was extended on bypass by further cooling to 15 degrees centigrade, allowing a period up to sixty minutes of circulatory arrest. Hypothermia, and indeed profound hypothermia, is still induced today but periods of circulatory arrest are minimal.

However cardiac surgery was not to be held back. Moderate hypothermia, though very important, allowing simpler open heart procedures to be undertaken, was only a diversion. The great breakthrough came finally in 1953. Gibbon, Professor of Surgery and Director of Surgical Research at the Jefferson Medical College, Philadelphia, a long time pioneer, used complete human heart bypass to carry out a successful repair of a large secundum atrial septal defect in an eighteen year old girl, who has been a long term survivor.[24] Three other cases by the same team were unsuccessful to such a degree that, although it was recognised as the way forward, once again other techniques were tried in preference.

Lillehei, another dominating figure, used a cross circulation technique from an adult donor to allow repair of a ventricular septal defect to be undertaken in an eleven month child. The child's father was the donor. The blood flow from the father was 55ml/kg/min and the cross circulation time was slightly over 19 minutes. Unfortunately the patient died from pneumonia 11 days post operatively. One has to wonder if the defect was

totally closed. Lillehei did a series of patients by variation of this method, 45 in all, with 28 survivors but the method gradually fell into disrepute. Another curiosity was in 1956 when Campbell used the lung of a dog as the oxygenator, operating on fifteen adults.[25]

Progress was being made despite these different, exotic and sometimes bizarre ideas, and Kirklin in 1955 at the Mayo Clinic reported an improved survival rate of 50%.[26] Two systems were coming to the fore, the screen or disc oxygenators, and the bubble. The Rygg bubble oxygenator was the oxygenator which was selected in Belfast when the present unit started in 1968.

It is alarming to think that as these events were going forward the pacemaker and defibrillator were still in their infancy, and consequently rather crude devices. Progress was swift and in 1958 the first successful open heart operation in the United Kingdom was done by Bill Cleland in the Hammersmith Hospital, the anaesthetist being John Beard.[27] The oxygenator used was a disc oxygenator designed by Dennis Melrose in the same hospital.

What was happening in Belfast in the late 1950s? Closed mitral valvotomy was a well-established operation, and repairs of patent ductus arteriosus and coarctation of the aorta were performed as needed. The use of moderate hypothermia with brief circulatory arrest for atrial septal defect repairs and pulmonary valvotomies was also established. Moves were afoot to start an open heart unit and Harold Love has noted that in 1958 John Bingham presented a feasibility study to the Medical Staff of the Royal Belfast Hospital for Sick Children to set up an open heart unit there. He favoured the Lillehei bubble oxygenator but the quotation of £1500 for the machine may have been too costly for the Children's Hospital, and in the long run nothing came of these moves.

John Bingham

In 1960 came the next major step forward. The first open heart operation using a heart lung machine was done by Tom Smiley, another of the three thoracic and cardiac surgeons in Belfast at that time. Mr Smiley was a unique personality, in some ways a larger than life character, who was a spare time farmer and an enthusiastic huntsman. He had the heart of a lion, and at times of crisis in the operating theatre was well known to raise his voice more than a little, and then, when all the excitement was over, to revert to singing hymns, a favourite being the 23rd Psalm.The unique operation was in 15/16 Theatre on the Royal corridor, and the anaesthetist again was Maurice Brown. Dr Brown was an impressive looking man, a talented anaesthetist and a formidable adversary. He had done much to protect the status of anaesthetists at the start of the National Health Service in Northern Ireland and he was a most forceful ally to me at the start of the present Unit.

The perfusion apparatus used was the Melrose NEP Disc oxygenator, pioneered in the Hammersmith Hospital and the funds for this were given by the Royal Victoria Hospital's Working Men's Committee. This was the beginning of a five year phase of development. Professor Pantridge was an important influence and Manus O'Donnell, George Patterson and later Professor Richard Clarke were involved with the perfusion. Kathleen Galbraith was the theatre sister.

From today's vantage point it is easy to be critical of the disc as a large, cumbersome and difficult machine. It took about three hours to set up, and four to five hours to take apart, clean, resiliconise the discs and leave ready to be sterilised for another day. A large priming volume of 5 litres was needed, and fresh heparinised blood was the

prime of choice. However it helped to establish open heart surgery as a practical procedure.

On a day of an open heart operation, the theatre staff came on duty about 6.00 am to be ready for the commencement of surgery at 10.00 am. At that same time blood donors were assembled to produce the requisite twelve to sixteen units of fresh blood, the patient was anaesthetised about 9.30 and all was ready to go about 10.00 am. On this schedule they must have been exhausted before they started. In today's world it is hard to recall the stresses which were part of this pioneering work.

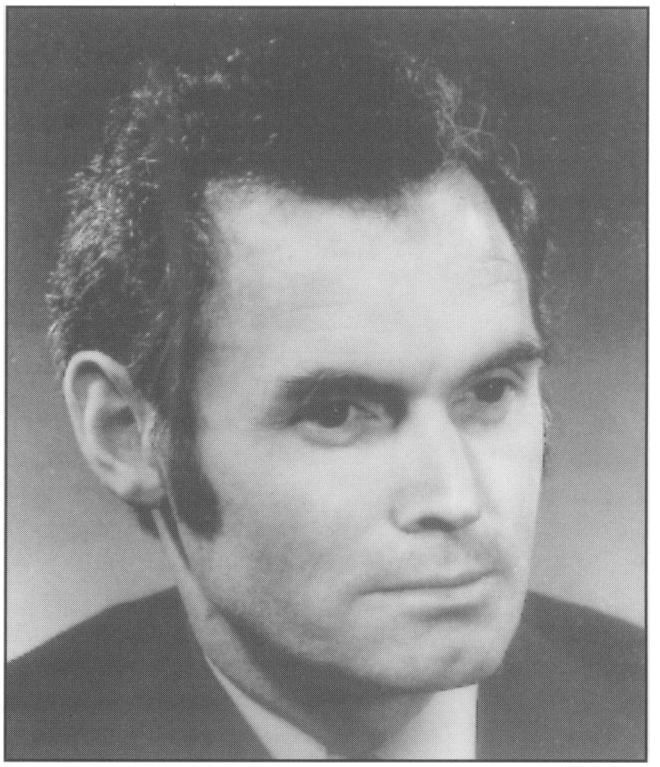
There was a long learning curve. The early cases were done in the Royal. Later there was a move to the Royal Belfast Hospital for Sick Children for logistical reasons with a change of surgeon to Maurice Stevenson, a much quieter and more thoughtful character than Smiley. He was a man of consummate skill, great understanding, humble in success, quiet in adversity. His anaesthetic colleagues were Harold Love and Gerry Black; Richard Clarke and Conor Mulholland became part of the team. The work later moved back to the Royal but problems persisted.

In those days, monitoring was very primitive, pulse oximeters were still twenty years away, the microchip was just a dream and intensive care was in its infancy. Success was hard-earned here, as in many other units. In 1965 it was decided that the time had come to consider a fresh approach and after a report by Professor D'Abreu from Birmingham, a new stand-alone unit and team were recommended, with ward beds, an intensive care unit, a dedicated theatre and all staffed appropriately.

Thus new opportunities arose and arrangements were made allowing me to go to Houston, Texas to work in the very busy Cardiovascular Surgical Units of the Methodist, St Luke's and Texas Children's Hospitals, with Dr Arthur Keats the recognised anaesthetic authority of the day. Houston was dominated at that time by two very powerful figures in cardiovascular surgery, Michael de Bakey, a visiting lecturer to this hospital in 1967, and the doyen of aortic surgeons, and Denton Cooley, the fastest and slickest cardiac surgeon in the world at that time, maybe ever, who made surgery look fatally easy. My period there came to an end coinciding with his doing heart transplants numbers 7, 8 and 10 in the world during May 1968. His work load was eight cases per day in two operating theatres to the accompaniment of Country and Western music. Houston was a vibrant growing city with the NASA space centre nearby, an expanding microchip industry and the medical centre growing in all directions.

The first attempt to find a dedicated cardiac surgeon for the Royal Victoria Hospital failed but at a second attempt, and some head-hunting by Professor Pantridge there was a successful outcome. Early in 1968 Pat Molloy was appointed as cardiac surgeon to the Royal. He was an interesting person widely known for being the father of nine daughters when he came to Belfast. His optimism was further demonstrated in that while in Belfast his wife had her tenth child, a boy.

After his appointment early in 1968, Mr Molloy began to assemble a team. The senior theatre and intensive care nurses and the perfusionist went across to Broad Green Hospital, Liverpool where Mr Molloy was working and spent time there learning techniques. Communications were different in 1968 and there was an element of isolation in Houston. By the time my interview was eventually arranged for late May 1968, the rest of the team was well established and had done a series of animal operations in the dog laboratory.



Pat Molloy

This was an essential part of the team-building process. The dogs, usually greyhounds, were often from the dog track at Celtic Park, now the Park Centre. Some of the nurses who were resident in the Towers, with their grandstand view of the dog track, were able to spot likely candidates for bypass. Dogs who finished last seemed to have a high probability of helping in the establishment of cardiac surgery. I never enjoyed this phase and I must say I was glad when it passed. The Royal Staff as a body gave wholehearted support to the new venture. Mr J W S Irwin and Mr Reggie Livingston moved out of Wards 13/14 to provide beds for cardiac and thoracic surgery and a dedicated theatre was soon provided in Main Theatre Block to go with a Cardiac Surgical Intensive Care Unit with 8 beds situated beside the theatre.

This goodwill on the part of the Royal staff as a whole and the support by the surgical and anaesthetic personnel was an essential part of our smooth beginning. The help from Richard Clarke and the sage advice from Bob Gray were both needed and appreciated as was the full hearted help from all departments, especially haematology, biochemistry and radiology. But it was quite tense as 'zero hour' approached.

June 19th 1968 was D Day. Seventeen year old Anne Stevens was scheduled as the first patient, (a repair of a secundum atrial septal defect) and despite the considerable apprehension felt by everybody involved, the case went uneventfully. After extensive debriefing and some more dog experiments there was a repeat performance the next week. After this quiet beginning the next case was a double valve replacement in a patient with a permanent pacemaker in situ, the first such operation in Belfast. Fortunately that patient also did well. By the middle of August a total of fifteen cases, mostly open, had been done, with no mortality and it was time for a holiday. The novelty and the initial pressures wore off and cardiac surgery became just another service able to set about establishing itself and its pattern of work.

Those early cases are etched indelibly in the mind of everyone involved, such as a teenage female with Fallot's Tetralogy, a haemoglobin of 21 grams and a haematocrit of 80%. She had passed from blue to a dreadful blue grey colour never seen today. After a successful operation she looked totally different, being blonde and attractive, and in fact her only complaint when she came to her follow up appointment was that no one in Lurgan recognised her. Many patients were end-stage and sadly there were frequent failures. Strangely, I anaesthetised a man this summer for coronary artery bypass grafts, whose father had died after a mitral valve operation with us in 1968, at the age of 45 years.

Compared to today, problems and complications were frequent. The standby order for blood was twelve units of fresh whole blood per patient compared to four packed cells today, and it was all needed. One patient out of every five returned to theatre because of excessive bleeding. Air embolism was a heartbreak, literally and metaphorically, and there was a dramatic improvement with some adjustments to the machine and later by the introduction of arterial filters. Surgery was slow, monitors were erratic and basic. Fluid balance was difficult to achieve. Post operative care was taxing, complications common and a tracheotomy was virtually a death sentence. While the excitement of the successes kept us going it is hard to appreciate today how difficult and frustrating the work often was.

The work load in 1969 was 220 open and closed cases with an overall mortality rate of about 17%. The mortality in double valves was more than 20% and in triple valves about 30%. Surgery for congenital heart disease was always difficult and remains so. In those early days definitive repairs were only attempted in children of more than four years of age. Later the cut off point was less than 10kg but at any rate by natural selection we were operating on the best of the survivors. Even so the mortality was still in the region of 20%.

After a few years of stasis, many arguments with the Eastern Board and the Department of Health, and more than one commissioned report the work has grown in an erratic fashion to the present figure of 1150 operations per annum. The patients now average over 60 years of age, with a range of 0 to 84, and 3% are greater than 80 years of age. This compares to an average of 43 years at the beginning, ranging from 4 to 64 years. In contrast to 1969 today's mortality is 2.8% overall, the rate in valve surgery is 1.8%, in coronary artery bypass grafting 2.5%, and in paediatric work (now encompassing definitive repairs in neonates) is about 10%. The mortality statistics of the unit have always been among the best in the United Kingdom and it is comforting that they remain so.

The 225 cases in 1969 were done from a base of 20 ward beds and 8 intensive care beds. Today's 1150 patients have as a base 30 beds and 12 intensive care beds.

With time, anaesthesia and surgical personnel have changed. Jack Cleland was appointed a consultant surgeon in 1970. Pat Molloy's time was short and he returned to New Zealand in January 1973, to be replaced by Hugh O'Kane. Mr Cleland, who had returned from the Mayo Clinic shortly after our D Day, had worked in the unit as a Senior Registrar. He gave dedicated service until his retirement in 1995. He had particular excellence in valve surgery and he pioneered many new ideas and techniques.

Jim Morrison was a member of the anaesthetic team from 1970 but after making his mark left for Canada in the major emigration of 1974, creating a huge gap. Professor Richard Clarke and I struggled along for most of a year until the arrival of Ian Carson who gave encouragement to our developments both in monitoring and in narcotic anaesthesia.

Mr O'Kane's appointment introduced coronary artery surgery to Belfast, a region with a very high incidence of coronary artery disease. There was initial reluctance on the part of some cardiologists to submit patients for this surgical treatment and it is interesting that the first patient referred from this hospital was referred by one of the general physicians. Mr O'Kane's work proved itself, and his pioneering efforts in this field have been of great benefit for very many.

The paediatric work has always been very taxing, especially once the definitive repairs of congenital defects began to be the norm in the smallest infants. It remains so. Freddie Wood had a brief sojourn but Dublin claimed its son soon afterwards. The appointment of Dennis Gladstone has fortunately proved to be a quantum leap forward for Northern Ireland in this area. The whole surgical team has been strengthened and its base broadened by more recent appointments.

There is now a very strong anaesthetic team with each individual having their own personal area of expertise. I feel privileged to work with such capable, effective and yet pleasant colleagues. Thirty years ago the standard anaesthetics of the day had several deficiencies and this was more apparent with the beginnings of coronary artery surgery. The perioperative infarction rate was unacceptable, and anaesthesia had a responsibility in this regard. Houston was interested in high dose narcotic anaesthesia but concentrated on pethidine which turned out to be unsatisfactory. The studies in Belfast by Professor Clarke and myself [28] on the available induction agents and relaxants in 1969 to 1973 helped to highlight their deficiencies further.

Lowenstein then published his work on high dose morphine anaesthesia[29] and this was a real advance. The dose used was 1-3 mg per kg body weight so that the standard 70 kg man received 150 mg intravenously over a 2-3 hour period. This gave a more stable situation, with a decreased myocardial oxygen consumption. Unfortunately 10% of patients had some awareness for events. Diazepam was added which resolved that problem but the patients then had a prolonged recovery – days not hours. Finally Stanley recommended fentanyl in doses of 25-50 micrograms per kg[30] and this regime has dominated the past 20 years. Further changes are now taking place but that is a lecture on its own. The techniques now allow safe, low oxygen demand anaesthesia with early awakening and the next years promise exciting developments.

The invasive monitoring which was part of cardiac surgery from the beginning gave an opportunity to do haemodynamic studies and this interest has been maintained. Dr Carson studied high dose narcotics and the effects of midazolam both in theatre and in intensive care. Other projects followed as different personnel pursued their interests; these ranged from post operative ventilation studies, total intravenous anaesthesia, propofol in intensive care, psychometric tests of cerebral function and now immunology responses on cardiopulmonary bypass. There are many opportunities and it is important for this hospital that they should be encouraged.

The steady improvement in morbidity and mortality since the Unit began is in keeping with world trends. Recent high profile cases have demonstrated that not keeping pace with the required standards leaves units open to severe criticism. The cardiac surgeons have always taken part in a strict audit and while sometimes seeming to have a stick to beat their own backs, their example should be the norm in other disciplines.

What has contributed to these improvements? Today's patients are older and in many ways less fit. However they are better prepared, the

diagnosis is almost invariably comprehensive and correct, surgical techniques and materials have improved enormously and there has been a revolution in myocardial preservation.

The anaesthesia changes have already been mentioned. The management of patients in intensive care has improved with better agents, better monitoring and above all there are now highly trained nursing and other personnel. The message is clear that the best results are achieved with good teamwork and the best use of the available expertise.

There are many unsung heroes and heroines in this work. We owe huge debts to the dedication of nurses, perfusionists, technicians, physiotherapists, radiographers and all who work in the Unit. For example the senior perfusionist Mr E Stewart came to us in a time of crisis in September 1968 via the shipyard and then the electric maintenance department of the hospital. Ernie was naturally gifted, has absorbed change, initiated change, anticipated disaster and averted it with equal facility. He is a unique man of outstanding quality and the thousands of patients whom he has perfused so expertly owe him a lot.

Today you may hear many negative comments about the problems of being a student, then a young doctor. However, medicine offers unique opportunities. You have involvement with people at times of their greatest need and with their life under threat, and you have the opportunity to set them on the path to recovery. What could be better? Listen to the positive messages (and there are many), and take some of the negative comments with a pinch of salt. When I was a student there was great depression about job prospects. Fortunately they were wrong. At the end of my career I have no regrets and I remain an optimist for the future.

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Case Report

The successful use of spinal cord stimulation to alleviate intractable angina pectoris

G J McCleane

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The first reported use of electricity for its numbing effect was in AD 46 by a Roman.[1,2] Since then electro analgesia has been used in the USA in the 1850s and peripheral nerve stimulation has been used sporadically since. In 1965 Shealy and colleagues demonstrated that spinal cord stimulation (SCS) in cats blocked behavioural responses to acute pain,[3,4] and the same group subsequently implanted a spinal cord stimulator in a patient in 1967, reducing his chest wall cancer pain.[5] With these results SCS has been used with mixed results in a variety of pain conditions. In 1984, Sandric and colleagues reported that patients undergoing SCS for various conditions who also had ischaemic heart disease showed improvements in their ECG, and two had no further angina.[6] Murphy and Giles (1987) were the first to report the technique being used specifically for angina.

This is the first reported use of a Medtronic spinal cord stimulator for angina in Northern Ireland.

CASE REPORT A 52-year-old man with intractable angina was referred. He had two acute myocardial infarctions and coronary artery bypass grafting (CABG) in March 1989. There was a gradual return of angina since 1993. There was further angiographically demonstrated coronary artery disease progression and so CABG was performed again in 1995 but it was not possible to bypass all the occlusions. Subsequently he had frequent episodes of angina, requiring hospital admission for analgesia and nitrate infusions for a total of 78 days over the previous calendar year.

METHOD

The patient was given pre-medication of clonazepam 1mg. orally 2 hours prior to the procedure. Continuous ECG, blood pressure and S_pO_2 were monitored throughout and he received oxygen by face mask during the insertion. The epidural space was identified with fluoroscopic assistance using loss of resistance to air with a wingless 16 guage epidural needle with a 7.5mm terminal orifice (to allow advancement and withdrawal of the electrode without danger of electrode damage). The needle tip was placed in the mid-line of the epidural space. The electrode was advanced under x-ray control so that its tip lay just to the left of the midline at C7 and a stimulation trial carried out (the wire has 4 electrodes (0,1,2,3) each of which can be positive or negative, on or off, allowing variable stimulation over a fairly wide area). The aim was to achieve stimulation (paraesthesia) over the area where angina was felt; this was achieved. When satisfactory stimulation was achieved a 2cm incision through skin was made caudal and cephalad to the epidural needle, a subcutaneous pocket created, needle withdrawn ensuring no movement of the electrode wire and the wire end attached to a subcutaneously tunnelled cable which emerged 10cm from the midline and attached again to the external hand held stimulator. After 4 days of trial stimulation the patient reported no episodes of angina and an increased exercise tolerance, so a permanent receiver was implanted subcutaneously. The patient was further instructed in how to use the stimulator.

RESULTS

Over a 36-week follow-up period the patient has had angina on a much reduced frequency and on one occasion it was found that the electrode wire had migrated cephalad, and after suitable withdrawal satisfactory stimulation had been re-achieved his angina was reduced again to its new

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much reduced state. His exercise tolerance has also increased. His frequency of hospital admission has also been reduced to 3 admissions totalling 9 days over an 8 month period.

COMMENT

The mechanism of pain relief by SCS is unknown.[8] One concern is that angina, (a potential warning signal) of impending myocardial ischaemia) would be masked. In a series of 50 patients with intractable angina in whom spinal cord stimulation was performed for 1-57 months, ten subsequently died due to acute myocardial infarction.[9] In nine spinal cord stimulation did not conceal precordial pain and in one no information about precordial pain could be obtained.[9] In other series continuous ECG monitoring has demonstrated less ischaemic changes during exercise in those with SCS and intractable angina when compared to a non stimulated group,[10, 11, 12] and furthermore, less ischaemia provoked by increased pacing frequency in those with concomitant pace makers in situ when compared to a control group.[13] When monitored electrocardiographically after adenosine infusion, decreases in left ventricular ejection fraction were less marked in the SCS group when compared to those unstimulated.[14] It does therefore seem that the effect of SCS is more than a simple masking effect and may actually improve myocardial oxygen supply thereby reducing angina.

SCS is expensive. The price of an electrode and receiver is around £5,300. However, it reduced suffering and hospital admissions in this case. Hopefully this case has highlighted potentially useful analgesic effects of SCS in a patient with intractable angina.

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Case Report

Primary breast lymphoma

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Primary malignant breast lymphoma is rare, accounting for 0.15% of primary breast malignancies in England[1] and between 0.18% and 0.53% in the United States.[2,3] This represents 2.2% of all extra-nodal lymphomas.[4] We report a 75 year old female in whom mammography failed to show any radiographic suspicious features despite the presence of a clinically obvious breast mass. We present the clinical, radiographic and histological features of a primary breast lymphoma with a brief review of the literature.

CASE REPORT The patient was a 75 year old female who presented one week after noticing a lump in her right breast. Clinical examination confirmed the presence of a discrete 2.0-2.5cm mass in her right breast with no overt axillary or supraclavicular lymphadenopathy. A mammogram was obtained but there was no mass lesion or radiographic abnormality corresponding to the clinically palpable mass. A segmental mastectomy was performed to remove the right breast lump.

Histopathological examination showed grossly a 2.2cm tumour with surrounding cuff of normal breast tissue. Histology showed the tumour to be composed of sheets of small round darkly staining cells with little cytoplasm. Immunohistochemical markers reacted strongly for lymphold markers whilst epithelial markers were negative. The tumour cells were positive for B-cell markers in keeping with a malignant non Hodgkin's B-cell lymphoma.

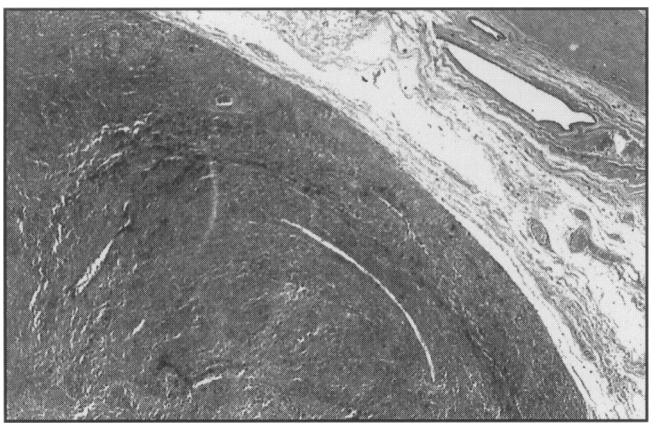

Fig 1a. Low power of fairly well circumscribed cellular tumour with adjacent breast tissue.

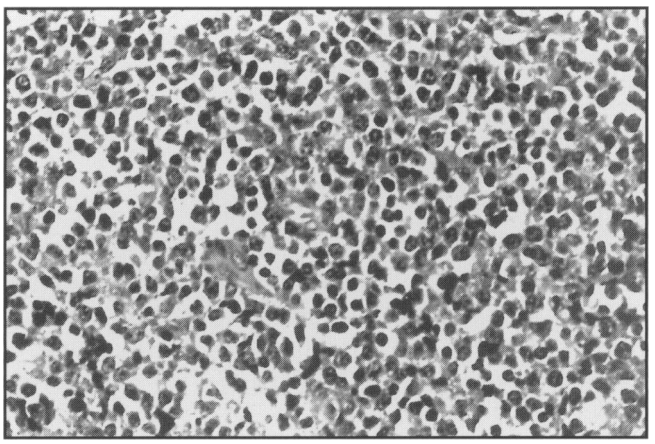

Fig 1b. High power of tumour composed of poorly cohesive cells with darkly staining nuclei and little cytoplasm. Immunohistochemical LCA stain positive for lymphoid tissue.

In view of this unexpected histological diagnosis she was fully investigated to rule out an extramammary focus of lymphoma: physical examination was otherwise normal; CT scan of

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thorax and abdomen was normal; bone marrow aspirate and trephine was normal. These investigations confirmed the criteria for primary breast lymphoma.

Following her original surgery this lady was treated by the regional oncology service with involved field radiotherapy to breast, ipsilateral axilla and supraclavicular fossa. Chemotherapy was not required. Follow up surveillance is planned but at present this lady is well and disease free.

DISCUSSION

Primary breast lymphoma is a rare tumour with an incidence of only 0.15% of primary breast malignancies in England.[1] This represents 0.7% of non Hodgkin's lymphomas and 1.7% of extra-nodal non Hodgkin's lymphoma.[5] Therefore it is important to look for extra-mammary evidence of lymphoma before a diagnosis of primary breast lymphoma can be made. To be considered a primary lymphomatous tumour, a close association between mammary tissue elements and the lymphomatous infiltrate is required and patients with pre-existing extra-mammary lymphoma do not qualify.[6] In this case excision biopsy specimen, computerised axial tomography and bone marrow aspirate satisfied these criteria.

Clinical presentation of mammary lymphoma is often indistinguishable from breast carcinoma, with a mass lesion being common. Bilaterality has been observed in some series[1-3, 7-9] but reports are few and series are often very small. Presentation with a second lesion in contralateral breast has also been reported,[9, 10] but this throws the initial diagnosis into question. Recent reports have linked the specific skin changes of lymphocytic lobulitis with primary breast lymphoma.[5] Some series suggest a bimodal age distribution but most recent reports suggest peak incidence in the 6th and 7th decades.[2, 8, 9]

Histological features in this case showed typical immunohistochemical staining for lymphold LCA marker while epithelial stains were negative (Figures la and 1b). B-cells predominate in this case as in most series; however mixed infiltrates are common.[7] Histological grade is suggested as a major prognostic indicator with high grade undifferentiated tumours faring worst accounting for a 5 year survival of as little as 49% in some small series.[2]

Treatment protocols often advise excision biopsy of the tumour both for diagnostic as well as treatment purposes. Management principles then are as for extra-nodal non Hodgkin's lymphoma with involved field radiotherapy to breast, ipsilateral axilla and supraclavicular fossa. Recent series suggest early use of combination chemotherapy may improve outcome[9] but series are small and treatment protocols often follow those for more common extra-nodal lymphomas. Often chemotherapy is reserved for evidence of disease recurrence or extension.

ACKNOWLEDGEMENTS

The authors wish to thank Dr R Harte for his advice on treatment in this case.

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Case Report

Hypoparathyroidism – Presenting 40 years after thyroid surgery

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Hypoparathyroidism after thyroidectomy is a well recognised post-operative complication. Usually however it occurs in the immediate post-operative period. We report here a patient in whom the diagnosis of hypoparathyroidism was made 40 years after thyroid surgery.

CASE REPORT

An 81-year-old woman was referred by her general practitioner with a three-month history of fatigue, generalised muscle weakness, anorexia, insomnia and depression. She also complained of an unpleasant metallic taste in her mouth occurring over the same length of time. Her symptoms were severe enough to give the patient concern as to whether she could maintain her independent lifestyle.

Apart from a thyroidectomy carried out in 1957, and bilateral cataract extractions in 1988 and 1990, she had had no other illnesses. She was not receiving any drugs.

On examination a thyroidectomy scar was evident. She had a mild proximal muscle weakness, a positive Trousseau's sign and a positive Chvostek's sign.

Serum calcium was 1.25 mmol/L (normal range 2.10-2.60 mmol/L); phosphate 1.97 mmol/L (normal range 0.8-1.55 mmol/L); alkaline phosphatase 134u/L (35-120 u/L); magnesium 0.61 mmol/L (0.70-1.03 mmol/L); T4 27.9 pmol/L (7.6-19.7 pmol/L); T3 1.9 pmol/L (1.5-3.0 pmol/L); TSH 0.02 mU/L (0.4-4.5 MU/L); parathyroid hormone <10 ng/L (10-50 ng/L). Full blood count, renal function and autoantibody screen were normal. An electrocardiogram showed a prolonged QT interval of 568 msecs.

The patient was treated with intravenous calcium (75 mmols over five days) and intravenous magnesium (5 grammes over 24 hours). She was stabilised on 1 gram of calcium daily, calcitriol 250 mgs twice a day, and bendrofluazide 5 mgs daily.

Her symptoms resolved as her calcium level improved and she regained her independence. At the time of discharge 10 days later, her calcium level was 1.9 mmol/L and TSH was 0.01 mU/L (0.4-4.5 mU/L). This persistent thyroid abnormality has continued throughout the follow-up period. 16 weeks later T4 was 15.1 pmol/L and TSH 0.02 mU/L, and 20 weeks later T4 was 16.2 pmol/L and TSH 0.01 mU/L. A TRH stimulation test gave a TSH of 0.02 mU/L; 0.05 mU/L, and 0.04 mU/L at 0.20 and 60 minutes respectively. The serum calcium remains within the normal range (2.58 mmol/L) on replacement therapy. In more recent weeks the patient has experienced new clinical symptoms, in particular episodes of sweating. Treatment with carbimazole has been introduced.

DISCUSSION

Hypoparathyroidism after thyroid surgery usually occurs within days. There are only a few case reports in the literature of a long latent period before presentation.

Lehmann and Leidy[1] reported the case of a 59 year old woman admitted to hospital with a tonic-clonic seizure, due to hypocalcaemia secondary to hypoparathyroidism occurring 38 years after thyroidectomy. In 1995 Bellamy and Taylor[2] reported a case of a 47 year old woman presenting

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with seizures as a result of hypoparathyroidism due to thyroid surgery 36 years earlier.

Billis, Montgomery and Clarke[3] published a paper on partial hypoparathyroidism following thyroid surgery and commented there was no direct relationship between symptoms experienced by patients and calcium deficiency. The reported symptoms are similar to those in our patient. However, our patient was hypocalcaemic on admission and responded to calcium replacement therapy, suggesting that her symptoms were related only to her recent calcium level. She had no previously-documented serum calcium measurement. Thus we are unable to say when in the post-operative period she became hypocalcaemic. Her response to calcium therapy would also make thyrotoxicosis an unlikely cause of her presenting symptoms. These resolved while her thyroid function test abnormalities persisted.

Our patient's latency period of 40 years is the longest between thyroid surgery and the presentation of hypoparathyroidism of which we are aware. Also she presented with vague symptoms related to hypocalcaemia rather than the florid symptoms present in the reported cases. It highlights the value of a serum calcium measurement in elderly patients presenting with vague symptoms.

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Centenary Meeting, Department of Epidemiology and Public Health, The Queen's University of Belfast, 26-28 September 1996

The Department celebrated its centenary in the Autumn of last year when a scientific meeting and social programme took place to mark the event. The Department was set up in the Autumn of 1896 when a part-time lectureship in Sanitary Science was established and held Jointly by a Professor Letts who was a chemist and Dr Henry Whitaker, the second Medical Superintendent Officer of Health for Belfast. Subsequently, there was a full-time lectureship in Hygiene and also in Sanitary Science before the Department was renamed Public Health in 1921. The Chair in Public Health was held by James Wilson who had qualified with First Class Honours at Queen's in 1905. The Department occupied two laboratories and some offices on the first and second floors of the old Anatomy building on the main QUB site; the building sadly was demolished in 1969. Professor Wilson made a notable contribution to research on infectious diseases. He was also Bacteriologist to the Belfast Water Commissioners.

Subsequently the Department was entitled Social and Preventive Medicine, and moved to the Royal Victoria Hospital in 1954. Professor Alan Stevenson held the Chair from 1948-1958 when he became Director of the MRC Population and Genetics Unit in Oxford: he died in 1995. Professor John Pemberton took over from Alan Stevenson in 1958 and developed the Department's interest in cardiovascular epidemiology. An early prospective study of the development of coronary heart disease in General Practice was successfully mounted. In 1965-66 a study of fatal myocardial infarction in Belfast paved the way for the introduction of Mobile Coronary Care by Frank Pantridge. During Professor Pemberton's time, other departments grew out of the general discipline of Public Health at Queen's: Medical Statistics, Medical Genetics and General Practice. He retired in 1976 at the same time as Professor Peter Froggatt, who had held a personal chair in the Department, became Vice-Chancellor. The Department of Social and Preventive Medicine changed its name to Community Medicine in 1976 and became the Department of Epidemiology and Public Health in 1989.

The present Head of Department is Alun Evans, Professor of Epidemiology who is involved in a number of international studies and is currently the Chairman of the WHO MONICA Project Steering Committee and Co-ordinator of the final analyses of the Project through a major grant from the European Commission. He is also involved in cardiovascular genetic epidemiological studies with France through the ECTIM and PRIME studies. These studies have identified major susceptibility genes for cardiovascular diseases. Dr Frank Kee, Honorary Senior Lecturer in Public Health Medicine, is involved in these genetic studies and Dr John Yarnell is Senior Lecturer in Cardiovascular Epidemiology with a special interest in haemostatic factors and cardiovascular disease. Dr Anna Gavin runs the recently established Northern Ireland Cancer Registry and Dr Paul Darragh has an interest in accident epidemiology; they are both senior lecturers in Public Health Medicine. The Biostatistical wing of the Department is strong in modelling and survivorship analysis through Dr Gilbert MacKenzie and Mr Derrick Bennett; and Dr Chris Patterson has a special interest in the epidemiology of diabetes. The Department is in transition again: it became the Division of Epidemiology on 1st October 1996.

ALUN EVANS

CURRENT WORK OF THE DEPARTMENT

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Much of the current work of the Department now involves international collaboration, the subjects encompassing biostatistics, cardiovascular and

cancer epidemiology, studies of accident prevention and health promotion, health services research in cardiology and the genetic epidemiology of cardiovascular risk and diabetes.

- The ECTIM project, a collaboration between four WHO MONICA centres (Belfast, Lille, Strasbourg and Toulouse) and INSERM (Paris), first reported an association in 1992 between a deletion polymorphism of the angiotensin converting enzyme gene and myocardial infarction (MI) risk. The role of a common polymorphism of the angiotensin II receptor gene in modifying this risk has now also been studied. DNA from 613 cases and 723 controls was assessed to determine the presence of an A → C transversion at position 1166 of the AGT_1R gene. The odds ratio for MI associated with the ACE DD genotype was 1.05 for subjects without the AGT_1R *C* allele, 1.52 in *AC* heterozygotes and 3.95 (1.26 -12.4) in *CC* homozygotes (test for trend $p<0.02$). Fifteen per cent of individuals carry both the DD genotype and at least one C allele and thus these results could have clinical implications for the prevention and treatment of coronary heart disease. The ECTIM study extension to women in Belfast has just finished recruitment: the study is being repeated in Glasgow.

- Using data from the Northern Ireland colorectal cancer register for 1990/91, we have assessed the association between colorectal cancer and levels of community deprivation. Electoral wards were grouped into quintiles of the population after ranking of their Townsend individual deprivation "scores". Incidence ranged from 22.5 (for quintile 1) to 29.9/ 100,000 (quintile 5) for men but the trend for females was less regular: 18.4, 23.8, 27.3, 26.5, and 23.9/100,000 for quintiles 1 → 5 respectively (ie from the most "affluent" to the most "deprived" fifths of the population). After adjusting for age and sex in Poisson regression, there was a significant association between total colorectal cancer incidence and levels of community deprivation, the rate ratio for the most deprived quintile of the population (compared to the least) being 1.28 (95% CI. 1.06 -1.53). As others have shown that such associations as these are not explicable entirely on the basis of the distribution of known risk factors, further research is needed to determine plausible mechanisms for the association.

- Previous attempts to assess what determines urgency for bypass surgery have not accounted for clinicians' opinions about the efficacy of surgery for particular types of patients. We have investigated these using clinical judgement analysis. Fifty "paper patients" were assessed by 33 clinicians to determine their urgency and priority for surgery. A Clinical Judgement Analysis employed linear regression models to reflect the impact of clinical and non-clinical "cues", such as smoking habit and angina severity, on urgency and priority decisions. Overall, the decision models had high explanatory power, accounting for, on average, 73% and 82% of variance of urgency and priority decisions respectively. The relative impact of the demographic cues on urgency and priority was substantially less than that of the clinical cues. The influence of factors such as smoking habit or body weight was generally independent of the doctors' beliefs about the efficacy of the operation. In light of this the basis of the rhetoric on the *pros* and *cons* of according priority to particular types of patients needs to be re-examined.

- Although childhood insulin-dependent diabetes mellitus (IDDM) is known to have a strong genetic component, environmental factors are also implicated. So far such environmental risk factors as have been identified are weak. The available immunological evidence points to the existence of a long pre-diabetic phase when the destruction of insulin-producing cells of the pancreas has begun, but the disease is not yet clinically apparent. This has led to an increased interest in potential risk factors operating in the perinatal period and in early infancy. The Northern Ireland Children's Diabetes Study is a case control investigation of environmental risk factors focusing on this early period. Northern Ireland is one of nine European centres collaborating in this project which will provide data on 1,000 cases and 2,000 controls. Data handling and analysis of the Project is based in Belfast.

THE BRADFORD HILL LEGACY

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Sir Austin Bradford Hill's criteria for causality are re-visited in the context of assessing the

effects of potential risk factors for CHD in longitudinal epidemiological studies.

The problem of quantifying the *independent* effects of competing risk factors in multifactor regression analysis is addressed briefly and reliance on pure significance testing is criticised.

The need to consider more substantive modelling issues is illustrated by analysing further data from the prospective '1202 middle-aged men' study of CHD incidence conducted in Belfast by Greig *et al* (1980). Multi-factor analyses at 5 and 7 years of follow-up confirm the findings of the Pooling Project (1979) in relation to the weakness of the association between CHD incidence and the three major factors: cholesterol, cigarette smoking and hypertension. These results lead to the conclusion that such data are virtually useless for the prediction of the disease status of individuals on study.

Curiously, the fact that it was possible to delineate relative risk successfully seemed to satisfy most epidemiologists working in this area at that time. There was an apparent reluctance to accept that some important risk factors may have been missed and this failure led to a decade (the 70's) of delay in alternative hypothesis elaboration, formulation and testing. It also led to over-optimism about the likely beneficial effects of population-based multi-factor primary intervention studies which were conducted up to the mid eighties. Since that time a number of additional hypotheses have emerged – on fibrinogen, anti-oxidants, and genetics – and the challenge for epidemiologists, today, is to learn how to assess the influence of these risk factors more efficiently than hitherto.

We argue for closer international co-operation in the organisation of international prospective studies, for better hypothesis elaboration – especially in relation to clarifying the multivariate dependencies between competing risk factors (e.g., do current genetic hypotheses purport to explain existing relationships or are they more ambitious?), for greater attention to success in predicting absolute risk, for less weight to statistical significance in single samples and for a standard paradigm in which results can be validated.

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CORONARY HEART DISEASE AND CORONARY RISK FACTORS: ARE MEN AND WOMEN REALLY DIFFERENT?

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There is a shortage of data on coronary disease and risk factors in women, particularly from studies which included both sexes using the same definitions and criteria. Lacking the same amount of data from the two sexes, commentators seem to have been almost equally biased between assuming that men and women were the same, and assuming that they must be different.

Two studies incorporated into the long-term programme of the Cardiovascular Epidemiology Unit in Dundee are helping to answer this question. Registration of coronary events in Glasgow for the WHO MONICA Project (as also is being done in Belfast) produced 3991 events in men over the first seven years 1985-91 and 1551 in women between ages 25 and 64. If the results are standardized for age side by side comparison can be made of the circumstances, management and outcome in the two sexes. For a very large number of comparisons the results are remarkably similar, but there are also differences which the large numbers are big enough to reveal, which are subtle but of profound significance. Women victims are more socially isolated and socially deprived, are more often affected when at home, tend to call the GP rather than an ambulance and take slightly longer to reach hospital, where their presentation is more complicated than for men, although investigation and management are very similar. It has long puzzled investigators that more women died after arrival in hospital than do men and the Glasgow MONICA data confirm this, but they also show something previously not realized, and now confirmed across the international MONICA Project, that more men die suddenly outside hospital. In our data the differences are equal and opposite so that the proportion below age 65 dead by 28 days is equal at 50%, but men and women get there by different routes.

Our risk factor data are based on the Scottish Heart Health Study which surveyed 11,600 men and women between 1984 and 1987 and for which we have an average of 7.6 years of follow-up data, allowing us to compare the performance of 26 factors in men and women, as well as the

menopause in women, in predicting coronary risk. We have used risk ratios, dividing continuous data into fifths, but also looking at categorical data according to its classes and have produced a league table for each sex showing the rankings of risk ratios in descending order. Again there are remarkable similarities but subtle differences, but in this case numbers of end-points are not large enough for many of the differences to exceed the play of chance. However, it is clear that risk ratios for many factors are higher than in men, although absolute risk is lower. Lipid factor ranking is different in men and women, and most surprising is what happens to the Bortner score for "Type A Personality" which is of no predictive value in men in this study, but turns out in women to be paradoxically and perversely a significantly protective factor. Further follow-up data will help us to define these differences more precisely.

HAEMOSTATIC AND RHEOLOGICAL FACTORS AND RISK OF IHD AND STROKE

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Rheological variables, which increase blood viscosity and may promote IHD and ischaemic stroke, include haematocrit, plasma viscosity (influenced by fibrinogen and lipoproteins) and white cell count.

In the MRC Epidemiology Unit in South Wales two prospective studies of IHD were established in parallel in the late 1970s. The first study was based in Speedwell, Bristol and was focused particularly on HDL cholesterol (the HDL fraction had been rediscovered as a risk factor in 1975). The Caerphilly Study (Caerphilly, South Wales) was also based on a population sample of middle-aged men and was testing several additional hypotheses to those being tested in Speedwell. One strength of these studies is the common core protocol and the measurement of several putative risk factors by the same laboratories. This was true for the haemostatic risk factors measured in the first phase of both studies. A fibrinogen measurement using heat precipitation, plasma viscosity and white blood cell count were measured in one laboratory, and the Clauss coagulation fibrinogen, the thrombin time and the heparin-thrombin clotting time in another. The results at 5 and at 10 years of follow-up confirmed the importance of both fibrinogen and white blood cell count as independent predictors of risk of subsequent IHD. However plasma viscosity was correlated with fibrinogen (particularly with that measured by heat precipitation), and whether fibrinogen or viscosity (or both) is the truly independent risk factor remains to be resolved. Differential white cell count was measured in the second phase of the Caerphilly study and significant associations were found between incident IHD and neutrophil and eosinophil counts.

Results from the South Wales prospective studies have been confirmed by Scottish studies including Scottish Heart Health, Glasgow MONICA, Edinburgh Artery and WOSCOPS. Viscosity reduction can be achieved by risk factor reduction (smoking, lipoproteins, fibrinogen). The recent association of fibrinogen genotypes with arterial disease suggests that some fibrinogen elevation may precede the disease, rather than result from the disease or from stimulants of arterial inflammation.

Other haemostatic risk factors investigated for their association with IHD include; factor VII, factor VII/von Willebrand factor, plasminogen activator inhibitor (PAI), tissue plasminogen activator (tPA) antigen, and fibrin D-dimer. Results from Caerphilly and elsewhere will be briefly reviewed, and a case for selective antithrombotic prophylaxis presented.

RENOVASCULAR DISEASE

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Controversy exists regarding the relative importance of genetic versus environmental factors in blood pressure variation. Kidney disease is the commonest identified cause of secondary hypertension. Elevated blood pressure is a major determinant of progressive renal injury leading to end-stage renal failure (ESRF), irrespective of the initial etiology of the renal disease. Hypertension is twice as common in persons with one or two hypertensive parents and genetic factors may account for 30% of the variation in blood pressure in various populations. Multiple crosstransplantation studies in experimental models of genetic hypertension have indicated that the kidney is the organ conferring

predisposition to hypertension. There is clinical evidence from human renal transplantation that a similar relationship may exist. Patients with ESRF who receive a kidney transplant from a donor with hypertensive parents tend to have higher blood pressures than those receiving a transplant from donors with normotensive parents.

There are relatively few specific examples of human genetic mutations which confer a state of renal hypertension. The commonest monogenic defect associated with hypertension is adult polycystic kidney disease. Liddle's syndrome, a rare monogenic hypertensive disorder, illustrates the physiological consequences of unregulated and persistent distal renal tubule sodium channel activity. The polygenic contribution to human hypertension has proved more difficult to unravel.

DNA polymorphisms within genes encoding components of the renin-angiotensin system (RAS) have been associated with predisposition to renal disease. Equally intriguing are reports linking specific RAS gene variants with more rapid decline in glomerular filtration rate and resistance to the beneficial effects of ACE inhibitor therapy. More effective targeting of clinical strategies to retard progressive renal injury remains a longer term goal of such population-based studies involving candidate genes.

A fetal environment which results in low birth weight has been postulated as a determinant of blood pressure in adult life. The theory that maternal malnutrition causes fetal growth retardation and subsequent adult hypertension is supported by data describing hypertension in the offspring of female rats fed low protein diets during pregnancy. An hypothesis exists that intrauterine growth retardation results in impaired renal development; this has been termed "congenital oligonephropathy". A decreased number of nephrons leads to compensatory nephron hypertrophy with associated glomerular capillary hypertension. This is ultimately a maladaptive response leading to glomerular sclerosis and the emergence of hypertension.

Therefore in a subset of hypertensive patients the origins of raised blood pressure may be traced to environmental insults which decreased functional renal mass coupled to genetic variation in the regulation of renovascular responses to injury.

THE GENETIC EPIDEMIOLOGY OF CARDIOVASCULAR DISEASE

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Coronary heart disease (CHD) is a multifactorial disease with complex physiopathology; its frequency increases with age, is higher in males than in females, varies considerably among populations and is influenced by a large number of environmental and hereditary factors. A gene may be involved in the predisposition to CHD if it exists under functionally different forms which may affect regulatory or coding regions of the gene. This may induce variability of metabolic or signalling pathways and have beneficial or detrimental consequences. By contrast with the rare highly deleterious mutations causing monogenic diseases, the known variants predisposing to CHD do not confer a strong increase of risk but they are quite common and thus may have an important impact in terms of population attributable risk. Another consequence of the high frequency of predisposing alleles is that their simultaneous presence in an individual is not rare and may lead to a high risk of CHD.

The candidate gene strategy involves the following steps:

1. select a candidate gene (a gene which codes for a protein that may be involved in the disease process),
2. obtain sequences of coding and regulatory regions,
3. identify polymorphisms of the gene,
4. measure intermediate phenotypes, such as the plasma or cellular levels of the coded protein,
5. look for associations of the genetic polymorphisms with intermediate phenotypes and disease, evaluate experimentally the putative functionality of the variants.

Associations between CHD and polymorphisms of the ApoE, Apo(a), ApoB, MTHFR and ACE genes have already been demonstrated and a large number of genes coding for molecules involved in the local processes that may contribute to the pathogenesis of CHD are currently being explored.

The linkage (sib-pairs) and association (case-control) approaches are complementary. The first offers the most straightforward way to identify new genes contributing to risk, whereas the second is the best way to study candidate genes. In the near future, gene-gene and gene-environment interactions will be investigated in large studies of several thousand patients. Characterization of genes involved in the chronic and acute processes of CHD may considerably improve our understanding of the etiology and mechanisms of this disease.

THE ANTIOXIDANT HYPOTHESIS TEN YEARS ON

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Since classical risk factors such as smoking, hypercholesterolaemia and hypertension explain only part of the risk of coronary heart disease (CHD), other factors remain to be evaluated. Fruit and vegetable-rich diets have been strongly linked to a substantially lower risk of CHD (as well as to cancer) so an exploration of potentially preventive nutrients is timely. In the Optional Vitamin Study of the WHO/MONICA Project the plasma levels of essential antioxidants, such as vitamins C and E, in the populations of European countries contrasting in CHD mortality and fruit and vegetable consumption, were investigated. This study showed a strong inverse correlation between plasma antioxidants and the age-standardized mortality of CHD. *The Antioxidant Hypothesis* [Bibl Nutr Dieta 1986; 37: 53-91] postulated that a suboptimal antioxidant status is a hitherto underestimated risk factor for CHD which may be corrected by a "prudent diet". Subsequently case-control and prospective studies in various populations have shown an increased CHD risk with poor antioxidant status at the individual level. The data suggest that, for a country with relatively poor vegetable consumption, e.g. Great Britain, the risk factors rank as follows: vitamin E >> vitamin C ≥ ß-carotene. In some data total cholesterol and diastolic blood pressure have been ranked below vitamin E. All complementary data suggest "optimal" plasma levels to be approximately ≥50 µmol/l vitamin C, ≥30 µmol/l lipid-standardized vitamin E (alpha-tocopherol/cholesterol ratio ≥5.2), and ≥0.4 µmol/l ß-(≥0.5 µmol/l total) carotene. Relative risks are doubled at 25-50% lower levels of each antioxidant, and multiplicatively increased in combination. Within the last ten years the antioxidant hypothesis has gained additional support from data based on the evaluation of self-prescribed vitamin supplements in the USA and randomized double-blind intervention studies [J Nutr Biochem 1995; 6: 206-236]. A recent British study suggests a potential for secondary prevention in CHD patients by large doses of vitamin E.

In conclusion, the best chance to minimize the risk of the multifactorial CHD may be avoidance of classical risk factors and a concurrent improvement in micronutrient intake.

HOMOCYSTEINAEMIA

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Homocysteine is a sulphur amino acid produced by the demethylation of the essential amino acid methionine. It can damage vascular endothelium, affect both platelet function and coagulation factors, and may promote the oxidation of low density lipoprotein cholesterol.

Homocystinuria was first described in Northern Ireland in 1962 by Carson and Neill. It occurs when plasma homocysteine levels are sufficiently high for the disulphide homocystine to be excreted in the urine, and is associated with precocious vascular disease. It is caused most frequently by the inherited deficiency of the enzyme cystathionine B-synthase and also by rarer enzymatic defects. Recently, a common mutation present in perhaps seven per cent of the population, thermolabile methyl tetrahydrofolate reductase, has been associated with less severe hyperhomocysteinaemia.

Mild elevations of plasma homocysteine are also now known to be unequivocally associated with increased risk of atherosclerotic vascular disease of the coronary, cerebral and peripheral arteries. The European Concerted Action Project Case Control Study of 750 vascular disease cases and 800 control subjects indicates that a fasting level of plasma homocysteine above 12 umol/L (the top one fifth of the control distribution) doubles the risk of myocardial infarction, stroke or peripheral vascular disease. Over 40 case control and prospective studies confirm this relationship and indicate a strong, graded and independent

effect. Furthermore, a raised plasma homocysteine level interacts with smoking, hypercholesterolaemia and hypertension to further increase risk.

Plasma homocysteine levels are controlled both by enzymes such as cystathionine B synthase and by the intake of folic acid, Vitamin B12 and Vitamin B6, all of which modulate the enzymes controlling homocysteine levels. Homocysteine levels increase even at low "normal" levels of folate intake, and folic acid has a profound homocysteine lowering effect in most subjects. It may also counteract the effect of thermolabile methyl tetrahydrofolate reductase. A high fat, low folate diet, frequent in these islands, may be particularly atherogenic. Whether or not folate supplementation may reduce vascular disease risk is now a critical question, and one which will be addressed by randomized control trials in the near future. Only when such trials are complete will it be possible to determine with certainty whether the strong relationship between plasma homocysteine levels and vascular disease is one of cause and effect.

TEN YEARS AFTER CHERNOBYL, ADJUSTED MORBIDITY RATES AMONG EMERGENCY WORKERS

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The Chernobyl accident – being the largest radiation disaster in the nuclear industry – has changed radiation in large geographical areas. Even ten years after the accident the impact of its effects on the health status of the population is difficult to assess quantitatively and objectively in scientific terms. For scientific, logistic and political reasons, sufficient data are not available and available data are of limited validity. On the other hand, there is a definite need for optimising decisions and measures in order to minimise the health-damaging consequences of conceivable future accidents.

Here, the German-Russian collaborative research project *Effects of Radioactive Radiation on Health – Risk and Projection Models* tries to make a contribution. It aims at the provision of methodologies for estimations and projections that are optimal for the data available as well as for present scientific knowledge. Bases for this are mathematical models which differ from traditional statistics by assessing uncertainties in data and knowledge explicitly.

A first model of this kind has been designed for morbidity estimation based on the available data of the Russian National Medical and Dosimetric Registry. It tries to assess uncertainties in the data due to non-regular participation in the annual follow-up health examinations. In other terms: unobserved states in the process of disease registration are modelled to allow morbidity estimates that are stable with regard to random fluctuations in the empirical data. A first application of the model deals with morbidity dynamics for the period of 1986-1994 among emergency workers (clean-up workers) at the nuclear power plant. The results show for 12 major classes of disease groups that incidence rates (adjusted for this type of uncertainty) are lower than those observed in the registry. This difference is explained by accumulated morbidity effects due to missing observations in the follow-up examinations.

In addition the results confirm a striking fact. Among emergency workers – at least in the first years after the accident – the major concerns in primary health care were psychosomatic diseases where a direct dose-effect relation of radiation is not known. At present it is unclear whether further modelling will reveal results for a better understanding of such effects of a nuclear accident.

However, the model generally deals with the well-known problem of 'missing follow-up' in registry systems. Thereby, the application of the model may become of general interest. We believe that traditional statistical methodologies do not assess the full potential of information in such registry systems. This may be true at least with regard to health policy decision making. Here, problems and objectives are differently defined as in epidemiological research.

NEW DIRECTIONS FOR EPIDEMIOLOGY?

Russell V Luepker

Division of Epidemiology, School of Public Health, University of Minnesota.

Epidemiology is in its golden age. The borders of our research are expanding and are well-supported. Student applications for training programs are increasing and our students easily

find employment at universities, government and industry. The work that we do is increasingly appreciated by our colleagues, government and industry. The public seeks our pronouncements to make important decisions about their daily lives.

Yet all is not well. Recent editorials such as those in the Lancet "Do epidemiologists cause epidemics?" and in Science "Epidemiology faces its limits" question our methods, interpretations and even importance. In the United States, a "public interest group" supported by industry has been established to confront scientific studies which are deemed "harmful".

Epidemiologists have responded to these criticisms with disbelief suggesting that we only present the information and others are over-interpreting. Others blame the press for mis-reporting and sensationalizing scientific information. On further thought, some experts do wonder if epidemiologists occasionally over-interpret associations and risks. But we also need to do some self-analysis to understand these criticisms.

Dramatic advances in data collection, data handling and analyses have occurred. Those who analyse the data are frequently not those who collected the information. Powerful software reduces the time to perform an analysis to seconds where days were formerly necessary. All potential associations can be analysed and significant, though marginal, associations will be found. Our training programs have taught this technology to our students who have the sophistication of the latest biostatistical methods. They are increasingly detached from an understanding of the intricacies of data collection or the pathophysiology of the diseases that they study. It should not surprise us if interpretations are flawed.

Finally, while many blame the press for sensationalizing epidemiologic data, many of our colleagues actively seek press exposure. Either drawn by the bright light of public interest or a desire to impress political and funding agencies, they become entangled with the reporters' objectives to find stories which generate public interest.

This crisis in etiologic epidemiology is a real and serious one. It calls for more thoughtful and conservative interpretation of data, better training of young epidemiologists in the breadth of the field and more thoughtful relationships with the media. However, it is also true that etiolgical epidemiology may actually be reaching its limits and, while valuable, it is not the direction for future development of the field.

There are two other important areas of public health inquiry suitable to epidemiologic methods. The first is in disease surveillance. Measuring trends over time with careful methods has only recently become a legitimate and growing area of research. The importance of understanding not only the current status of the population health, but the directions that it is taking are of immense importance and benefit. This applies both to the general population, the traditional domain of epidemiology, but also to those who are ill: ie outcomes and health services research. Here, epidemiologic rigor and methodology have been lacking but are needed for the important decisions on allocation and deployment of medical resources.

The second important area is a reminder of our history. As the legend tells, John Snow not only documented and mapped the London Cholera Epidemic, he also removed the Broad Street pump handle, eliminating the source of the infection. Epidemiology, as the basic science of public health, must look beyond counting events and finding associations. It must be ready and willing along with other public health disciplines to suggest the alternatives and directions for solving revealed public health problems. Only with this approach will Epidemiology attain its mission to improve the public health.

The Ulster Medical Journal, Volume 67, No. 1, pp. 73-76, May 1998.

Book Reviews

The Royal Victoria Hospital Belfast. A History 1797-1997. Richard Clarke. Belfast: the Blackstaff Press, 1997. pp. xv and 304. ISBN 0 85640 601-5. Price £20.

This has been a vintage decade for commemorating Irish teaching hospitals. In Dublin alone seven general, and three specialist ones, have attracted histories, six of the former on their down-town closure and move to the Beaumont, Tallaght or St. James's complexes: only Sir Patrick Dun's has failed to find a chronicler. It is now Belfast's turn and this sensibly planned, beautifully produced, handsomely illustrated, and well-written volume marking the Royal's bicentenary is a superb memorial to what is, by any standards, one of the premier hospitals in these islands. There have been previous histories – by A. G. Malcolm (up to 1850), Sydney Allison (1850-1903), and the almanac approach of Robert Marshall (1903-1953), and numerous other publications on specific events, facilities, personnel and personalities, but this is the first attempt to 'tell the story' of the hospital over the whole two hundred years albeit in one volume.

Several factors have ensured its success. The book is beautifully produced. Richard Clarke is a gifted narrator trawling expertly through a welter of facts to select those essential to the story so that the narrative never lacks pace, clarity and focus yet preserves essential scope – remarkable given the wealth of source material. I have never, for example, seen the early period (1797-1850) described so lucidly and coherently, if necessarily briefly in only some 9,000 words. Only in the post-1948 period does the mushroom growth of facilities and personnel force the author, in describing the trees, to neglect something of the forest. Professor Clarke has a wide knowledge of and feel for the swing of local and medical history and this adds sensitivity and authority to the text and adds skilfully the dimensions of the external social, economic, demographic, medical and political factors which were so important as being the waters in which the hospital developed and swam. There are few digressive *cul-de-sacs* and only one (highly appropriate) foot-note to distract and detract from the main thrust of the narrative. It is necessarily less detailed and comprehensive than Allison's history (1850-1903) but it is better ordered and cohesive and freer from errors. The numerous illustrations are well-chosen, of high quality, and add value to the text.

The format of the book is well-conceived. The chapters are sequenced chronologically, and contain sections describing the physical plant, specialities, personnel, personalities, medical developments etc, with little overlap and then adequately cross-referenced, the whole ensuring precision and clarity. There are valuable appendices including a 30-page chronological list of all consulting staff from 1797 ('visiting' and/or 'attending' staff up to 1948) giving dates of death (and of birth of deceased) where known, of appointment to and leaving the staff, and of the specially practiced. Authorities consulted are listed but not referenced in the text: this may offend the scrupulous scholar but it lubricates the narrative. There is an adequate, name-based, index.

The author treats personalities sympathetically though not hagiologically – a charitable medical tradition especially prudent when many of those mentioned are living colleagues! Despite the plethora of facts conducive to inevitable errors I detected only one typographical one ('1914' instead of '1941' on page 160) and several of fact, most being unimportant. Thus, for example, Henry McCormac was appointed to the combined chair of physic at RBAI in 1837 not 1836, and was president of the faculty of medicine in 1840 and 1846, not 1840-1845 (p. 18); J. L. Drummond held two chairs in 1835-6, not 'three', since 'anatomy and medical physiology' was one chair, botany being the other (p. 21); it was The Queen's University *in* (not *of*) Ireland (p. 75); the 'appointed day' for the NHS 5th July 1948, not 'June' (p. 138); Sir James Black's honorary degree from Queen's is DSc and not MD (p. 151); it is the University of Michigan (which is at Ann Arbor) and not 'Ann Arbor University, Michigan' (p. 156); and – a gem for the pedant – the 'fenced-off area with waitress service' in the restaurant below wards 6 and 7 was not 'for senior staff' but was unrestricted! (p. 143). Other readers may find other errors but the paucity and triviality of those above speaks eloquently for the author's general attention to accuracy.

Professor Clarke and his publishers have produced a splendid memorial to a splendid hospital, a readable history not a dry chronicle and junk-house of indigestible facts still less the 'coffee table' book which its handsome appearance and lavish illustrations might at first suggest. Everyone interested in Ulster medicine and the Royal should have this volume on their bookshelves.

P FROGATT

Clinical Investigations in Gastroenterology. Second Edition. Malcolm Bateson and Ian Bouchier. Kluwer Academic Publishers. ISBN 0 7923 4391 3.

This small book sets out to review tests in gastroenterology. Reflecting current pre-occupations, the first chapter is devoted entirely to Helicobacter pylori and is right up to date, including details of how proton pump inhibitors mess up diagnostic techniques; an important consideration for a medical culture which tackles even trivial dyspepsia by prescribing sledgehammer therapy first and asking questions later.

The rest of the book is laid out partly by organ system and partly by important clinical entities like gastrointestinal bleeding and malabsorption. Endoscopy is prominent, and it is refreshing on the threshold of the 21st century to find a text which does not perpetuate the notion that the patient with dysphagia always needs a barium meal before an experienced endoscopist is called. On the other hand, a statement that colonoscopy reaches the caecum in "70% or more of examinations" would not impress those defining minimum standards for our cancer centres. There is a useful appendix covering antibiotic prophylaxis.

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is an excellent chapter on pancreatic function tests which we possibly don't do enough of. Northern Ireland clinicians, who have excellent immunology and gastrointestinal hormone backup, might have liked more detail on the antibody tests for coeliac disease and their limitations and on fasting gastrin, which is probably as useful and a lot less bother than a Schilling test for diagnosis of pernicious anaemia.

This book is probably not selective enough for use by trainees as a day to day text, but as a small, easy-to-read reference source it has few competitors. Kept on wards and gastroenterology units, it can be used to determine not only how to do a more esoteric test but also whether it is worth doing. With it to hand, one of the basic tenets of informed consent – that the doctor should know a bit more about the procedure than the patient – will surely be facilitated.

WILLIAM DICKEY

Gastrointestinal Emergencies 2nd Ed, Edit Mark B Taylor Williams & Wilkins Baltimore. Price £120.

This book aims to comprehensively deal with all possible emergencies involving the gastrointestinal tract. Each chapter is written by different contributors, most with multiple authors, who are renowned in their field. All but 3 of the 119 contributors are from North America.

At 1064 pages it is clearly not a convenient handbook for ready consultation in the event of an emergency.

It is an impressive textbook, which covers the whole range of emergency situations. It covers surgical as well as medical emergencies. Arguably some topics are included which do not immediately spring to mind as emergencies e.g. space-occupying lesions of the liver, ascites and non cardiac chest pain. Some topics which usually merit little mention, such as Boerhaave's syndrome (oesophageal perforation associated with vomiting) and typhilits (bowel infarction of obscure origin in neutropenic patients) are well described. The chapter on foreign bodies of the upper oesophagus is particularly well covered. Each topic is considered in detail, often with helpful practical points, and management is described well beyond the immediate emergency episode.

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The scope of the book is extensive, which unfortunately leads to occasional excessive detail in relatively rare conditions and inadequate discussion on some of the more common vascular problems. This begs the questions as to what group of clinicians would benefit from reading "Angiology in Practice"? I suspect it will be of most use to general physicians and cardiovascular surgeons. It should also be of benefit, although perhaps more as a reference text, to general practitioners, neurologists and interventional radiologists.

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Total knee arthroplasty has increased in use throughout the world dramatically in the past decade. In Northen Ireland we have seen primary knee arthroplasty grow from under one hundred in the late 1980's to a current total in excess of seven hundred and it is likely to expand to over 1000 in the next five years. With this enormous growth in primary knee joint replacement has come an increase in the requirement for revision arthroplasty. The numbers at present are still quite small but with the use of arthroplasy in younger and younger patients the numbers will inevitably rise. Because revision surgery affects a small group of patients, experience in techniques and best methods of treatment must necessarily be limited. It is therefore essential to learn from larger centres throughout the world and to amend our practise accordingly. This book gathers together the experience of a wide group of surgeons mainly from North America and condenses their views into an excellent review of our current knowledge in this field. The book is written in an easily readable format with chapters dealing with all the main aspects of this type of surgery.

The book has been divided into five main parts with sections dedicated to planning, technique and outcome and a dedicated

experience of the leading authorities in Cardiothoracic transplantation medicine and in this second edition he consolidates this.

It comprises four sections, eighty chapters and some eight hundred and twenty four pages.

This is a comprehensive and readable review of the "state of the art" in the field of Cardiothoracic transplantation and replacement of thoracic organs.

The contributors are predominantly from the United States and the early chapters reflect an American perspective on the medico-legal aspects of brain death and the assessment of potential Donors and Recipients. I would recommend to a general audience the sections on the historical aspects of heart and lung transplantation. These give you a sense of how exciting and hazardous the early days of transplantation were. Physicians with patients who are recipients or potential recipients should find the middle sections on the clinical aspects of transplantation particularly useful in their practice.

For the cardiovascular surgeons it is a textbook worth dipping into, especially if preparing for the final part of the FRCS. There are excellent reviews on operative technique and post-operative management, and numerous illustrations of surgical anatomy. Technophiles will find the last section on current and future advances in thoracic organ replacement a delight and hospital administrators will become decidedly queasy at the potential costs.

In summary, the book is an excellent review which will probably grace the shelves of some well endowed hospital library and perhaps the occasional office of a physician with an interest in transplantation.

M J D ROBERTS

Angiology in Practice. Edited by A M Salmasi and A Strano. Kluwer Academic Publishers Group. pp 526. £188. ISBN 0 7923 4143 0.

The development of angiology, as a specialty in its own right, has tended, until recently, to be a European phenomenon. This book appears to be a joint venture between the United Kingdom and Italy with the authors, almost exclusively, coming from these two countries. The classification, epidemiology, pathophysiology and clinical manifestations of a wide range of arterial and venous diseases, are addressed in 35 chapters.

The scope of the book is extensive, which unfortunately leads to occasional excessive detail in relatively rare conditions and inadequate discussion on some of the more common vascular problems. This begs the questions as to what group of clinicians would benefit from reading "Angiology in Practice"? I suspect it will be of most use to general physicians and cardiovascular surgeons. It should also be of benefit, although perhaps more as a reference text, to general practitioners, neurologists and interventional radiologists.

The main advantage of this text is that it reminds clinicians of the systemic nature of cardiovascular disease. "Angiology in Practice" presents a wide range of conditions, not usually found in a single text, thus making it an asset to most post graduate libraries.

PAUL BLAIR

Manual of Infection Control Procedures. N N Dainani. Foreward by Professor G A J Ayliffe. Greenwich Medical Media Ltd. ISBN 900151 286. £22.50.

The first thing to be said about this book is that the title is somewhat misleading: it is neither solely a manual of infection or, deals only with procedures. In fact, it covers a wide range of infection control practice and contains a wealth of useful and helpful information. The author covers, largely from a UK perspective, the organisation of an infection control programme, isolation policies, disinfection, problems with specific pathogens, prevention of infection and precautions for health care workers, etc. Much of what is contained can be adapted for local use and there are a number of helpful tables, including those which contain the incubation periods for a variety of infections, often very useful in daily practice.

There are a number of useful line diagrams, usually adapted from other publications, e.g. figure 3.1 which outlines the resistance of micro-organisms to disinfectants. Indeed, the author could have made more use of diagrams and tables as the layout is largely textual in nature. In a book which contains a wealth of information and advice it is not surprising that there are one or two errors or points of disagreement. Figure 5.4 which describes the antigen and antibody response in HIV infection is incorrect as the responses are juxtapositioned and one might disagree with the recommendation that the operating theatre should be routinely cleaned with disinfectants; a good detergent clean is adequate in most instances.

The comprehensive nature of this book, the sensible approach of the author and the wealth of information contained therein ensures that this book will be considered by Microbiologists, Infection Control Nurses and other health care workers such as physiotherapy staff as a resource in their clinical practice and during training.

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section to the problems of infection. While it is primarily of interest to the revision surgeon it should I feel be compulsory reading for anyone undertaking total knee replacement. It is an excellent reference text, well illustrated with high quality radiographs, excellent photographs and line drawings. It describes an easy classification for bone defects in the distal femur and upper tibia at the time of operation and correlates these to outcome.

The section on the infected total knee arthroplasty sets out a comprehensive guide to dealing with this most perplexing problem, detailing the options and setting out a reasoned approach to the surgical alternatives.

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