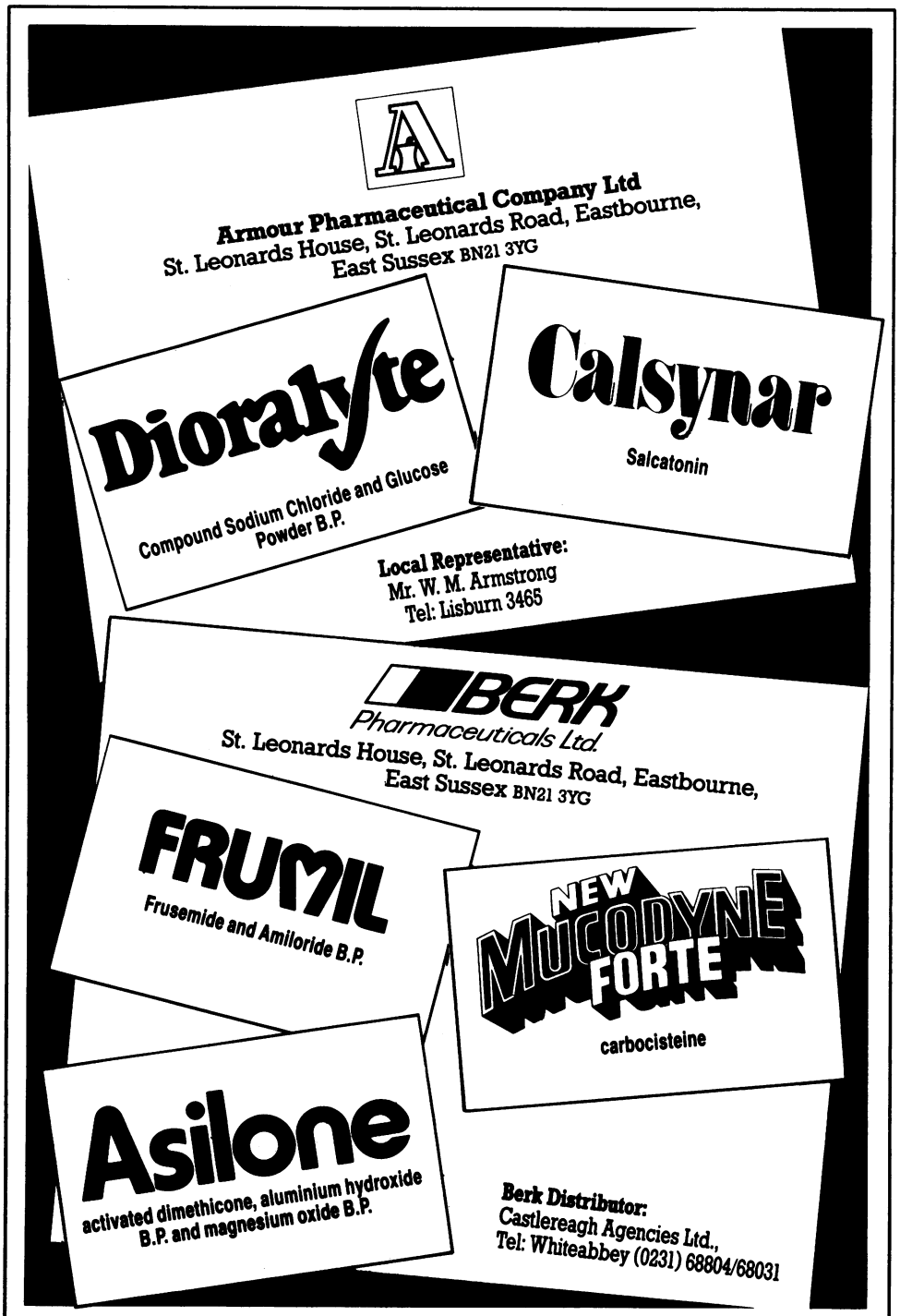
VOLUME 54, No 1. APRIL 1985

ISSN 0041-6193

THE ULSTER MEDICAL JOURNAL

Published on behalf of

THE ULSTER MEDICAL SOCIETY



The Ulster Medical Journal

THE ULSTER MEDICAL JOURNAL

NOTICE TO CONTRIBUTORS

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

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

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

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

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

6. References should be restricted to those really necessary and useful. This journal uses the 'Vancouver' style (see British Medical Journal 1982; **1**: 1766-1770 and Lancet 1979; **1**: 429-430). Text references are numerical. Each reference should include:

   i) a list of all authors when six or less (when seven or more only the first three should be listed followed by *et al*).
   ii) the title of the article.
   iii) the title of the journal (abbreviated to the form published by Index Medicus).
   iv) the year;
   v) volume number:
   vi) first and last pages.

   eg
   McCoy GF, Dilworth GR, Yeates HA. The treatment of trochanteric fractures of the femur by the Ender method. *Ulster Med J* 1983; **52**: 136-141.

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

7. Orders for reprints must be made directly to the printers when the author returns the proofs. Reprints must be paid for by the author, the cost can be obtained from the printer in advance.

8. Editorial communications should be sent direct to the Editor who will be pleased to advise on the preparation of manuscripts if requested.

   Fellows and Members of the Ulster Medical Society receive the Journal free. Individuals may subscribe directly (see back page). The journal contents are covered by *Current Contents: Clinical Practice, Index Medicus, Excerpta Medica* and *Science Citation Index.* This publication is available in microfilm from Xerox University Microfilms, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

Clinical chemistry in diagnosis and treatment. By Joan F Zilva and P R Pannall. 4th ed. (pp 539. £9.00). London: Lloyd-Luke, 1984.

This is the fourth edition of what has become the standard chemical pathology text for medical students and junior hospital staff. In addition, the text has in the past proved invaluable to students studying for the Institute of Medical Laboratory Technology's special examination in chemical pathology.

The current edition has been extensively revised due to advances in knowledge and practice since the previous edition in 1979. Some may feel that the text requires expansion — in the chapters on endocrinology, for example — but the authors claim, and I feel rightly so, that, if the size and scope of the book were increased, its original purpose as a text for medical students would be defeated. This problem is partly overcome by direction to recent specialist articles at the end of each chapter.

Many of the common problems met by junior clinicians in practice, such as those of electrolyte and acid-base balance, have been discussed in detail. Many chapters contain appendices which give details of treatments for specific clinical problems. Several chapters have been included which stress the integrative roles of the chemical pathologist in the laboratory and the clinician on the ward, and a chapter on the rapidly expanding field of drug monitoring has been included. KDB

Mnemonics and tactics in surgery and medicine. By John J Shipman. 2nd ed. (pp 339. £6.50). London: Lloyd-Luke, 1984.

I have always found that mnemonics are of little use in learning medicine and I have never used them in teaching. It seems to me that it is more difficult to remember the mnemonic than to remember the facts that it points to. The use of mnemonics also supposes an approach to learning medicine by memorising 'lists' of causes, complications etc. I think that this approach is incorrect, and that students should be encouraged to understand the general pathological condition and the disturbed physiology. The reasons for the causes and complications will then become apparent and learning lists by rote will not be necessary.

It was with these thoughts in mind that I approached this book. I noted firstly that it is a second edition, so presumably there are those who find useful the approach that I abhor. Then I noted that the author says in his preface that physiology and pathology are invaluable. He explains that 'the easiest way .... is to understand and to work out a logical sequence' and he then says that mnemonics might help in this understanding process. Maybe he is right, and if you are one of those who find them useful, here is a veritable feast.

There are 414 mnemonics recorded here. Some of them are as difficult to remember as the original list; for instance the features of the Stevens-Johnston Syndrome are memorised by remembering DAMASCUS! There are also some mnemonics which are very similar. The causes of the retention of urine (p 143) is PASSING BIG DUTCH CAPS, and the second list of causes of diarrhoea (p 80) is PASS BIG DUTCH CAPS. Similarly, the features of hereditary spheracytosis (p 204) is BUGGARS, whereas the causes of a swollen joint (p 152) is IT'S A BUGGAR. I see a cause for confusion in some poor student!

If you like this sort of thing, then this is the most comprehensive manual on the market. There are also four excellent cartoons. WO-S

Acknowledgements

The Ulster Medical Journal acknowledges the generous contributions from the following bodies, without which it would not be possible to continue publication:-

Royal Victoria Hospital Medical Staff Committee, Royal Group of Hospitals Free Funds, Belfast City Hospital Free Funds, Ulster Hospital Dundonald Medical Staff Committee, Queen's University Belfast Grant from Senate Funds and the Northern Ireland Council for Postgraduate Medical Education.

THE ULSTER MEDICAL SOCIETY

Whitla Medical Building
97 Lisburn Road
Belfast 9

If you are not a member of the Ulster Medical Society, we would appeal to you to give the question of joining your consideration. The Society has been in existence since 1862 (and is the direct descendant of the Belfast Medical Society founded in 1806), and has always been active in keeping its members interested in the advances in medical science. Meetings are held at intervals of a fortnight during the winter months, and papers are contributed by members and distinguished guests. Facilities are provided for doctors to meet informally afterwards, and have a cup of tea. *The Ulster Medical Journal, the official organ of the Society, is issued to all Fellows and Members free of charge.* The Society is now housed in its own rooms in the Whitla Medical Building of Queen's University at 97 Lisburn Road (replacing the Whitla Medical Institute which had to be vacated in 1965).

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

If you do not wish to become a member of the Society, will you consider entering your name as a subscriber to *The Ulster Medical Journal?* The subscription is £5.00 per annum, payable in advance to the Honorary Treasurer.

J A WEAVER, *President.*
P M REILLY, *Hon. Secretary.*
S A HAWKINS, *Hon. Treasurer.*

MEMBERS **£5.00** (A Member is one who is less than seven years qualified. He or she will automatically become a Fellow seven years after qualification and be liable to the higher subscription rate).

FELLOWS—1. (a) Annual subscription of Fellows **£8.00**; (b) husbands and wives who are both Fellows will be entitled to pay a combined subscription of **£10.00**; 2. Annual subscription of retired Fellows. Any Fellow who, by reason of retirement either through age or illness, is no longer engaged either in practice or in salaried employment, shall be entitled, on application, to pay an annual subscription of **£5.00** only, and provided that such Fellow has previously paid to the Society a subscription at the current rate for an uninterrupted period of at least ten years, or during such time has been in practice or service abroad.

All Fellows and Members of the Society who have paid subscriptions for 40 years or alternatively having been a Fellow or Member for 20 years and reached the age of 65, or more, shall on application be exempt from any further subscriptions.

TIES—Ties bearing the crest of the Society on a background of navy, maroon, green or brown may be obtained from the Honorary Treasurer.

To THE HONORARY SECRETARY,
ULSTER MEDICAL SOCIETY.

. . . . . 19 . . . .

Dear Sir,

We nominate for $\frac{\textit{Membership}}{\textit{Fellowship}}$ *of the Ulster Medical Society:—*

Name of Candidate . . . . . .

Postal Address . . . . . .

. . . . . .

Year of Qualification and Degrees . . . . . .

. . . . . .

Signature of Proposer . . . . . .

Signature of Seconder . . . . . .

EXCHANGES: Exchange journals and all relevant correspondence should be addressed to:

ULSTER MEDICAL JOURNAL,
QUEEN'S UNIVERSITY MEDICAL LIBRARY,
INSTITUTE OF CLINICAL SCIENCE,
GROSVENOR ROAD, BELFAST, BT12 6BJ,
NORTHERN IRELAND.

BOOKSELLERS: All correspondence, orders and payments for institutional and private subscribers, through booksellers, should be sent to:

THE HONORARY TREASURER,
ULSTER MEDICAL JOURNAL,
c/o. QUEEN'S UNIVERSITY MEDICAL LIBRARY,
INSTITUTE OF CLINICAL SCIENCE,
GROSVENOR ROAD, BELFAST BT12 6BJ,
NORTHERN IRELAND.

SUBSCRIPTIONS: Individuals who are not members of the Society wishing to take out a direct subscription should send a banker's order for £5.00 payable to the Ulster Medical Society (Northern Bank, Shaftesbury Square, Belfast), 'Ulster Medical Journal Account', to:

DR. S. A. HAWKINS,
HONORARY TREASURER, ULSTER MEDICAL SOCIETY,
DEPARTMENT OF MEDICINE,
INSTITUTE OF CLINICAL SCIENCE,
GROSVENOR ROAD, BELFAST BT12 6BJ, NORTHERN IRELAND.

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

The Ulster Medical Journal, Volume 54, No. 1, pp. 1-19, April 1985.

John Henry Biggart 1905-1979 — A portrait in respect and affection

John A Weaver

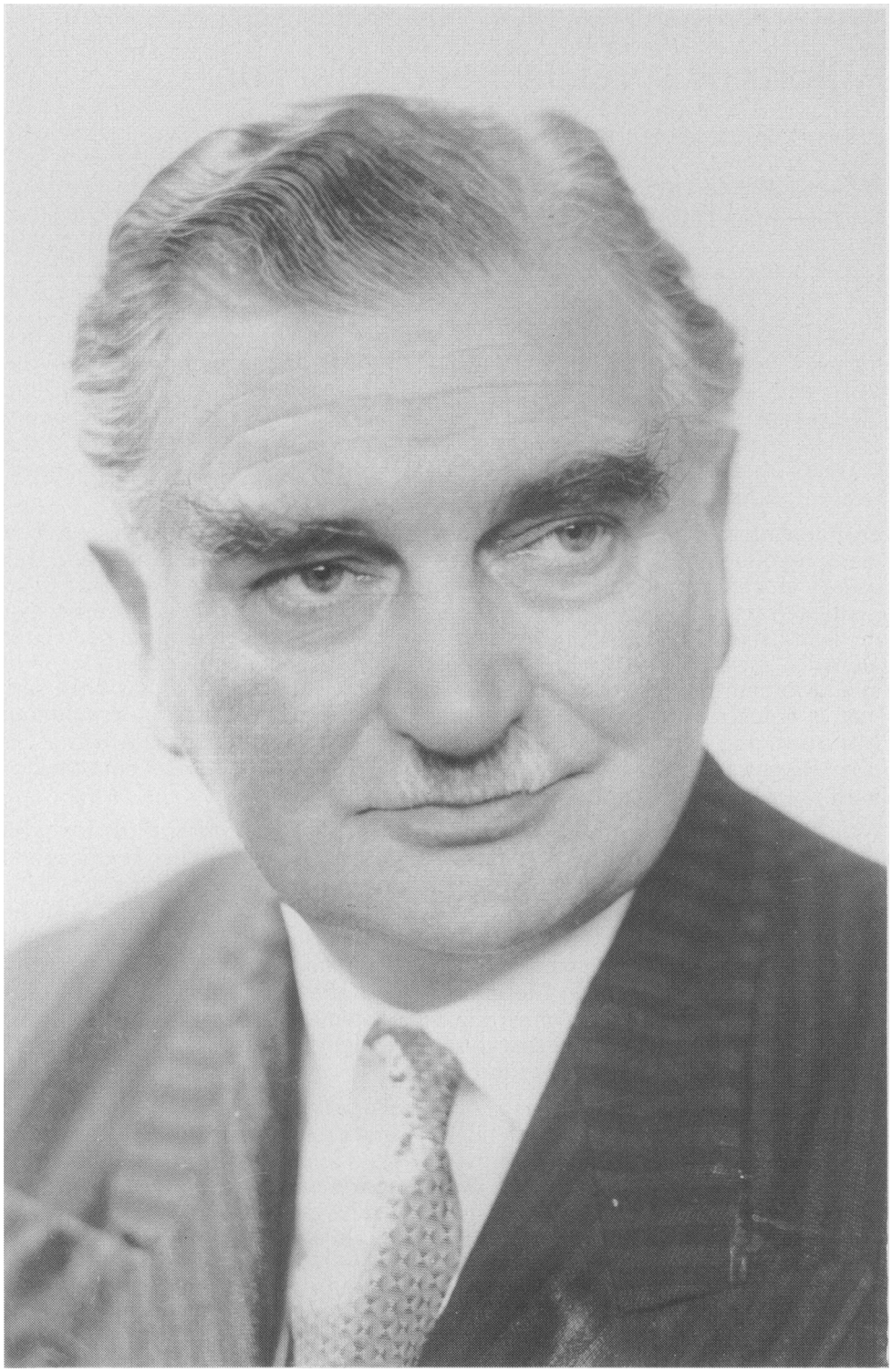
Presidential Address to the Ulster Medical Society,
1st November 1984.

John Henry Biggart was born on 17th November 1905 in Belfast in a house a few hundred yards from Queen's University. He was to be associated with the University as student, medical graduate, Professor of Pathology, Dean of the Medical Faculty, Pro-Vice-Chancellor and Pro-Chancellor, in all for half a century or more. He is arguably one of the major influences in the development of the Queen's Medical School on a par with other significant figures — McDonnell, Redfern, Whitla.

My intention is to describe his life in respect and affection, an affection shared by many here tonight because he accepted them into the medical school and was usually a benevolent father-figure throughout their undergraduate and post-graduate careers — more than a father-figure, an epitome of the philosophy that medicine should be a cultured calling, and he was ever a standard-bearer for Queen's University medicine. The emphasis on respect and affection does not intend to hint that others might take a more critical approach. It has been said that, in order to escape criticism, one should do nothing, say nothing, be nothing. None of those attributes was John Henry Biggart. One possible criticism of Sir John Biggart is that he held too much personal power for too long. Leave that for now — I will return to it.

I have been fortunate in my task by being granted access to personal papers of Sir John Biggart, and I am most grateful to Lady Isobel Biggart for her kindness and help. Lady Biggart is very well, or perhaps her own words portray the justifiable scepticism of a pathologist's wife: "The doctors tell me I am very well"; to which she added: "They have been very nice to me at the Belfast City Hospital". I joined with her in praising that excellent hospital, but no further test of our mutual sincerity was undertaken. In the course of an afternoon's conversation in the family home in King's Road, which she does not intend to leave, we talked about much that I will say. Her regretted absence tonight is encapsulated in "I am too sentimental an old thing at heart to come".

I have just referred to Sir John, and I have a difficulty in repeated reference to him as to what name I should use. To many he was simply "John Henry". To others "Harry" (and he was "Harry" to his family). He was personally amused at having once been called "a black bastard". Dr Desmond Burrows recounts that when he was working in the Pathology Department it was his task to approach the clinicians to present a case at the pathology meeting. He was sent to request a rather strong-willed physician "would he show a case?" When he came back to John Henry's office, Dr Burrows reported that the physician would not show a case. John Henry asked "Why?" Dr Burrows replied, "He won't show it". John Henry persisted. Dr Burrows repeated, "He won't show it". John Henry still questioned,



"What did he actually say?" Reluctantly, Desmond Burrows said, "He said he wouldn't do it for that black bastard".

Sir John opens his personal biography notes with a declared intent that they are "for my own delectation and to place on record for myself those episodes which bubble up through the morass of memory. So my journal is for me alone, for my enjoyment, but if others care to read some name, some place, may evoke for them, too, fond memory". I accept that as my licence for quoting from the papers. The journal begins: "I was born on the 17th November 1905. At that time my father was principal of a school in Ennis, Co. Clare. He had previously been principal in the school of Stranocum, Co. Antrim, and it was there that he met my mother. At that time he lived a bachelor life in the teacher's residence which, because of its architecture was known locally as the 'salt box'. It still exists and still justifies the description. Still, though I was conceived in Co. Clare, my mother, also of Antrim stock, came back to Belfast, and so I was born in the house of my aunt on the Stranmillis Road. My father later returned to Northern Ireland and we settled in the old Unitarian Manse near Templepatrick. Here the Rev. Robert Campbell had dominated his parish, and fathered Robert and John, who were subsequently to make their mark in the development of medicine in Northern Ireland. John was to build the Samaritan Hospital. Robert as a surgeon at the Children's Hospital in Queen Street is commemorated in the Campbell Oration".

Sir John Biggart's account continues, "And so I spent my youth in South Antrim. Yet my affection and my family ties were always with the northern part of the county. On the Biggart side my ancestors had come from Ayrshire in the early fifteen hundreds. My mother's family were Gaults from Ballynure".

About his childhood, Sir John wrote, "My affection is still to North Antrim, and its cliffs and rough seas. These North Antrim people were a curious but attractive mixture of the people of the glens and those Scots who had made their perilous way by Rathlin and the Isles to the land of rugged cliffs, who had made their way past Fair Head to Murlough Bay or Ballycastle. In 1921 my parents moved from their beloved Co. Antrim to Co. Down — near Ballygowan. They had been appointed to a mixed school. Of those early years in Ballygowan it is curious the memories that remain — Sunday school at 10.30 am, church at 12 noon, Sunday school at 3 pm and church at 6.30 pm — repeating the whole of the 119th Psalm, the whole of the Shorter Catechism with proofs.

"It is a curious thing that in a small provincial area like Ulster there should be such a diversity of living. While I lived in the village of Ballygowan, our house looked out over the village square, and from our windows one could overlook the whole comings and goings of the community. Yet perhaps the charm of the village life was the intimacy of it all. The country came through it and about it and pure undiluted country was only a few yards away".

Sir John continues, "I recall those to whom I owe a great debt — my Presbyterian and bachelor parson the Rev. W.K. McLernon. I remember the day he announced the creation of a lending library in his small village church. The establishment of the church library led to my having read most of Scott, of Dickens and of Thackeray by the age of 15. Thus was established a habit of reading which has been my solace: come riot, come bomb, come frayed tempers at committees, or frustration in one's work, one hour of deep immersion in one's books brought back peace and understanding. Gradually the novel has ceased to be of interest

and had been replaced by history, biography and the volume of George Eliot essays which I first read some fifty years ago. I found inscribed on its fly leaf 'In omnibus quietum quaesivi, sed numquam inveni salve in angulo cum libro' — 'In all things have I sought peace, but never have I found it save in a corner with a book'. Fifty years ago and perhaps the only prophecy which has remained true".

John Henry Biggart entered Royal Belfast Academical Institution at age 13 in 1918. "So I was accepted to Inst, went to James' the outfitters and duly appeared at school in the old black cap with its initialled monogram. Subsequent headmasters — not of local origin or tradition — were to alter the simplicity of the school cap, but to me it was a badge of superiority which we flaunted on the top of the tramcars as they sped down Wellington Place". His attachment to Inst was, I suspect, life-long. David Baird entered first year Dentistry, requested to transfer to medicine, as did a fellow student. The latter came out after 15 minutes in John Henry's office looking very worried, "I don't know whether I am in or not". David Baird straightened up, adjusted his Instonian tie and went in. "So you want to change to medicine, my son. That will be all right". John Henry's years at Inst seem to have been happy. Setting out each morning on the 7.30 train from Ballygowan, homework checked by father before leaving. It was obviously a long day of involvement in academic work and rugby and the various school societies. The sum of achievement while at the Royal Belfast Academical Institution was considerable. The Sullivan scholarship in mathematics, the Hyndman scholarship in Latin and Greek, the Musgrave scholarship in French, the Blair memorial scholarship in Physics and Chemistry; at the same time playing for the First Fifteen and obtaining an Ulster Schools cap.

There is a regretful note about social life, for in his account he says, "Unlike Methodist College which was co-educational, we were ruled with spartan simplicity, but it was generally thought that we more than caught up with the Methody boys in our first year after school". Also, an interesting analysis of Dr Jones the headmaster which maybe tells us something about John Henry's own appreciation as to how power should be exercised. "A great headmaster, but to many of us his seat upon Olympus seemed too lofty, too far removed, and the mountain side upon which we sought to climb too steep for us to obtain that intimacy that we desired".

And so to Queen's University. I continue quoting from his papers. "In the early twenties Queen's was a lovely university. Scarcely two thousand students were in attendance and over coffee in the Union one talked with budding classicists, or modern linguists, with embryo lawyers, with chemists and physicists. In those days the medical school was the dominant faculty. In my time each successive president of the Students' Representative Society, of the Students' Union and of the University Debating Society was a medical student. Of course that was before the development of that modern anomaly — the student politician — often neither student nor diplomat".

Some insight into Sir John Henry Biggart's commonsense exercise of power as a disciplinarian when Dean of the Medical Faculty and also a member of the General Medical Council can be gleaned from his account of his own appearance as a student in front of the University Disciplinary Committee: "Students' Rag Day in 1926 ended with a fancy dress ball in the Great Hall and I, for my sins, was one of the organizing committee. At the end of the Hall was a settee of matrons — wives of professors and pro-chancellors — in all a most decorous group whom we

hoped to entertain with presentations of bouquets, boxes of chocolates and the occasional sacrifice of a dance. As a member of committee one felt a certain responsibility. Students had been collecting all day. They had survived attacks in the spinning mills and factories which they had dared to invade. Managers in the offices of our then more frequent distilleries had poured out generous portions of their wares hoping soon to be left in peace. Girl students had done honour to the night, and many Queens of Sheba added beauty and excitement. My first call was to remove a student who had decided that he did not like a portrait of a former, and no doubt eminent, professor of philosophy and had shown his dislike by boxing the portrait. Next was the student who insisted on spending the evening in the ladies' lavatory. Then there was the dental student, who took what was normally a colourful professor by the lapels of his evening jacket and enquired about the whereabouts of 'the bloody red shirt' that he usually wore. On the whole it was a series of minor accidents. Yet in the morning when I returned to Queen's the night had attained the dimensions of a catastrophe. Rumour grew apace. One Queen of Sheba was the cynosure of every eye — had her straps not broken at a critical moment in the cloisters and had she not fled naked to the waist to the women's quarters pursued by those still able to run. The Committee was hastily summoned before the Discipline Committee. So I was interviewed by all the learned Deans of all the Faculties, grilled about every possible misbehaviour of students in such situations — indeed it was surprising how fervid their imagination could be. It was only when the final interrogator, the Dean of the Faculty of Medicine, asked me, 'Biggart, did you see anything that a drop of drink couldn't explain?' that I realized that here at last was a man with his feet on the ground".

Accounts of pre-clinical life as a student read as follows: "One of our great treats was the lectures from the Professor of Anatomy (Thomas Walmsley) — a Scot with much of the mystique of the Celt. His lectures were a delight, for with consummate skill he wove together the hard facts of anatomy and the philosophy of his subject. Many years later when I became involved in trying to understand the hypothalamus I was still to remember the scientifically clear way that I had been introduced to the anatomy of the third ventricle. He was a great teacher, possibly because of his reserved personality, too far removed from the average student, but many of the best were sufficiently stimulated to take up his subject and eventually to attain chairs in many places. Yet possibly the credit should not all be his, for back from the USA in the early twenties came as his lecturer one of the great characters of medicine — one Dr Richard Hunter. He had taken up medicine after a period in business, after acting as an interpreter for the British troops in France and I think partly because his brother, a general practitioner, had died from pneumonia after an exhausting journey in the snow to succour a patient". Sir John had obviously great affection for Dickie Hunter. In the 1949 RVH opening address he refers to him as "sometime lecturer in anatomy, artist, medical author and circus impresario . . . " Dickie had just returned from the Johns Hopkins Medical School and had been greatly enthused. Though a life-long bachelor he was to us the ultimate authority on procreation. From semen to ovum to blastocyte, to embryo, to foetus, we followed his magic drawings and his wit. He had the great facility of knowing each and every one of his students and when I returned to academic life he fathered me like a son, introduced me to medical history, taught me something of art, and years later led me into a particular type of personal, rather than paper, administration. A great lover of life, he was eventually persuaded to give up anatomy. So he became Secretary to the

University, and as such I found him when I returned to Queen's. Whether as Professor or Dean I don't remember that we ever wrote a letter to each other. Every morning I dropped into his office about 8.45, and we talked of many things. What little business we had to transact was duly noted, put upon the agenda of the committee and eventually the required end result achieved". At a later stage in his writings, John Henry describes the continuation of his happy working relationship with George Cowie, Dickie Hunter's successor.

It should be recorded in relation to anatomy that Sir John Biggart was the first winner of the Symington Medal — and was probably the only winner of the Symington and the University billiards championship in the same year. Generations of students will recall his pathology lectures opening the first year of clinical work — always the question to the class, peering up from under deep eyebrows at the seated rows, "Who won the Symington in this year? Who won the Milroy?"

"Having surmounted the hurdle of the second professional examination we proceeded to hospital. Spending our mornings at the Royal Victoria Hospital we dashed back to the Union dining room and a full round of lectures at the University. For the first time we felt that we were really becoming engaged in our profession. I suppose for most of us it meant something to do with patients and their ailments, and we had a rather mystical conception of what the doctor could do. In hospital we were well taught and Cecil Calvert, Ian Fraser, Cecil Woodside and George McFadden introduced us to the elementary surgical problems. One of the greatest difficulties was the overcoming of the shyness of bodily and physical intimacy. The belle on the beach is one thing; the same belle sick in bed is another. Yet somehow or other, almost unconsciously, we slowly acquired the art of medicine. For the art is much more slowly acquired than the science, a fact which seems often forgotten in the drafting of curricula by modern educationalists. Our clinical studies continued. All the ward chiefs were part-timers, giving their services free to the hospital, and I suppose earning their eventual rewards by impressing the next generation of general practitioners. Even the clinical professors were part-time. We soon learned of course to distinguish between those who could and would teach and those who couldn't and wouldn't. On the whole, however, their record of endeavour was very good, in some cases better than that of some of their full-time successors". I include that quotation from Sir John, not in the present-day context of part-time but as a tribute to that generation pre-1948 who created the medical environment of the hospitals of this city. Not for nothing does the inscription above Ward 9, RVH, proclaim, "The Honorary Medical Staff". The attitude of Sir John to part-timers portrays a characteristic that, while he was wholeheartedly a full-time academic, he could see virtue in those who were not and there lies in that an example of an openness of mind.

Hospital undergraduate years merge into the houseman year at the Royal Victoria Hospital in Sir John's reminiscences: "Pa Kirk and urea as a panacea for all things; the 'Turk' percussing a chest having made up his mind what was wrong, he then proceeded to demonstrate, and demonstrate beautifully and convincingly, the sounds that ought to emanate from his diagnosis. S. Boyd Campbell took some interest in me throughout my student years — I never knew why, but thought it possible that because our families stemmed from North Antrim we should stand together. Certainly he befriended me all the days of his life and in return I was occasionally able to mollify his colleagues. For he was a man who put his case strongly but not always tactfully". Sir John quotes from Dr T.H. Crozier's tribute, 'Strong in his enthusiasms and prejudices, he was a man of inflexible

principles, always a warm-hearted, generous and loyal friend'. "He was one of the clinical examiners in my final. I was given a patient with some valvular disease of the heart, and in my diagnosis claimed that I heard the relatively rare Austin Flint murmur. The actual examiner was a lady physician whom I knew was somewhat deaf. She listened to the heart sounds, could or would not confirm my diagnosis and appealed to her co-examiner. He listened and did not deny the presence of my murmur, but somehow I have always felt that, when I saw the twinkle in his eye, he too was fooling my examiner.

"The senior physician was one Dr John Morrow and I was his house physician in Wards 1 and 2. At some stage he had had an infection of the palm of his left hand which left him with a stiff middle finger. In the discussion of a problem he had the habit of massaging his stiff finger, and one soon came to appreciate that such massage was the danger signal before an emotional explosion. There was a tradition in Wards 1 and 2 that Johnny always liked to score off his house physician. It was an advantage to miss some obvious finding or test. I think this tradition grew from the time when Dr Sidney Allison had been his house physician. He was most competent, wrote up progress notes on his patients each and every day, and poor old Johnny had no excuse to show his authority as chief. Yet on one occasion Allison slipped. In the case progress chart he had recorded that the patient vomited 'once or twice' yesterday. So Johnny read the note, began to rub his finger, and turning to Allison exclaimed, 'Did he vomit once or did he vomit twice? For Christ's sake let us have some investigation in this ward'.

"As the houseman year came towards its end, one began to wonder what was to be the next stop. The man and subject that attracted me most was Professor Symmers and Pathology. So one afternoon I went to visit him. It was the afternoon of his final lecture for he was due to retire but he kindly appointed me so that his successor had a fait accompli on his hands".

Later an obituary of Professor Symmers in the *Ulster Medical Journal* described that he spent his retirement years studying 'Virgil', also reminiscent of Sir William Whitla's study in his own retirement of the Book of Daniel and the Apocalypse. I had been dejected that such topics were not being pursued by any of my retired friends until this year when Dr Richard Womersley retired to study in classics at Queen's. Sir John continues, "My new chief was Professor Drennan — homing back from Otago. Whilst Symmers was the Platonist, who was quite as likely to take us to W.H. Hudson's 'Purple Land' as to expound on pathology, Drennan revitalised our pathological service. He was a charming and always considerate chief, somewhat shyly encouraging, but at the same time expecting results". (Professor Drennan died this year — February 1984 — at the age of 100 years.) "My two years as assistant passed, my research completed, and my thesis was successful. Because of the impoverished state of the University, technicians were few and far-between, though those which we possessed were kindly and competent and have been my good friends throughout my life. From them I learnt the current techniques of the day. So the average day was from nine in the morning until midnight — cutting, staining, counting. Then one walked a couple of miles home and found that a considerate mother had left a plate of cold ham, a potato or two in the pot, plenty of butter and possibly a raw onion".

Bert Russell, the doyen of laboratory technical staff, describes a life-long sustained personal relationship with Professor Biggart. As Professor Biggart was appointed to more and more committees, the time spent in the Pathology Department was less. Bert describes how he used to chide him and it was received

with equanimity. "Professor Biggart, you will soon be the *visiting* Professor of Pathology". Bert Russell pays great tribute to John Henry's technical ability. Professor Biggart was, in fact, elected a Fellow of the Institute of Medical and Laboratory Science. "He was a very competent technical person. He could stand shoulder to shoulder with you in routine pathological work, cutting and staining. He understood the technical difficulties; I remember having tried to use silver impregnation to stain for senile plaques in a case of dementia and, try as I might, it would not work. I went to him and said, 'I have had a lot of trouble with this case and I still can't get it to take up the stain'. 'Let us have a look', After a moment. 'It is not much good. Did you try such and such?' And again, 'Did you try that?' Then finally, 'I always wondered why you got so much success. It is very nice to see that you are human after all.'"

Sir John's papers continue: "Towards the end of my second year Professor Drennan suggested that I apply for a Commonwealth Fellowship, so without much hope I filled in the requisite forms and duly appeared in London for interview. I am afraid I felt very much the little country boy. I was greatly pleased when a few days later I was notified that I had been awarded a Fellowship to Johns Hopkins". On arrival at the Johns Hopkins, Sir John writes, "I was greatly thrilled by being nominated as Professor McCallum's personal assistant in working with Arnold Rich and in getting to know that most historic pathologist Poppa Welch". One is conscious of what a royal medical lineage is here, for Welch had worked with Cronheim, Ehrlich, von Recklinghausen and Koch. I was also intrigued when I was checking Welch's work in Europe in the *Dictionary of American Biography*: 'Welch, William Henry. Born Norfolk, Connecticut, 1850. Died Baltimore, Maryland, 1934. Pathologist, medical educator'. 'Medical educator'. Perhaps that phrase should not have startled me, but startle me it did. I suspect that in this country we have ceased to regard anyone as having medical education as the prime function of his career. Harry Biggart's life shows that 'medical education' was to him a very prime function. My own memory of pathology lectures by Professor Biggart is how frequently the work and opinions of this group of men — Welch, McCallum, Rich — were quoted. There was nothing particularly chauvinistic in this because by any standards these men were giants but it is possible to discern that the 'country boy' from Ulster had at a critical stage of his development, been offered images of conduct, of pathological competence, and of medical administration (in the case of Poppa Welch). He writes, "Rich was a great stimulus. We met in his home every Monday night at seven for a journal club. For an hour we had a musical interlude, Rich playing the violin, his wife the piano". An asset of the Commonwealth Fellowship was that they supplied additional funds to enable the Fellows to travel throughout the States in the summer vacation. Sir John describes leaving Maryland down to New Orleans, across Texas and Arizona to the Californian coast, calling at Los Angeles (for 1932 was also the year of the Olympic Games), then up to Canada, the Fraser River valley, the Yukon, across the Rockies to Chicago. He comments, "As the second year at Hopkins drew to its close there was much to worry about. Academic posts in Great Britain were not numerous. In April Professor McCallum asked me to stay at Hopkins. Before I had really considered this, my former chief, Professor Drennan, who had moved from Belfast to Edinburgh, wrote to offer me a lectureship in neuropathology, and so my problem was solved. I had always been interested in neurology, and the artistry of gold and silver impregnation had been an attraction when I first entered pathology.

"So on the 1st September 1933 I took up my post in Edinburgh. As well as lecturer in neuropathology, I was also pathologist to the Scottish Asylums Board and neuropathologist to the Royal Infirmary, and became closely associated with Norman Dott and his neurological team". Four years were spent in Edinburgh, a great deal in routine neuropathology, this specialized subject very much in its infancy at that time. "At the beginning of my fourth year in Edinburgh my small textbook on neuropathology was published. It had been an interim in the routine work, and was founded on the lectures which I gave to the candidates for the Diploma in Psychological Medicine. In the spring of 1937 Sir Hugh Cairns offered me an appointment at Oxford, but whilst I was considering whether I should accept, the Chair in my own university became vacant. My Edinburgh friends encouraged me to apply for Belfast. Normally I should not have thought of it. I was only 31 years of age. All the professors of pathology I knew were men of maturity — Symmers, Drennan, McCallum, Muir, Shaw Dunn, Matthew Stewart, and so on. Obviously in many ways I was incompetent for such a post. Though one had endeavoured to gain as wide an experience as possible, and to make good defects in one's knowledge, such defects were bound to exist. Eventually I found myself on the 'short list'. Two mornings after the appointments board interview as I booked our return to Edinburgh I ran into Dr Hunter, the secretary of the University, who told me I had been appointed".

John Henry in a conversation with Ingrid Allen, expressed "a passing regret" that he had not remained in neuropathology — perhaps a memory that Hortega took the Chair at Oxford. Dame Dorothy Russell had commented "What a pity young Biggart didn't take it". Dorothy Russell — very famous, very precise, an austere figure, and Ingrid described a sense of awe of her and difficulty in getting on any degree of terms with her because of her fame and character. John Henry said he had no difficulty, "you just had to treat her like a woman".

"So I came back to the medical school as a professor, and practically all my colleagues had been my teachers. Of course they knew — or thought they knew — all my foibles. On the other hand, I still had the respect for all of them that I had had as a student. I too knew their weaknesses, for students are ever quick to see through their teachers. The Department was woefully understaffed. So much of the teaching and a great deal of the practical service work of the hospital fell to the professor and senior lecturers". John Henry Biggart's early years as Professor of Pathology were magnificent because of the unhindered commitment to the subject and the absence of the multiple responsibilities of his later years. Desmond Montgomery, a student in his first classes — "the impression was of the arrival of a whiz kid". About the same time, Gerald Nelson, a junior in pathology — "One knew that one was being taught by a master". Dr Robert Marshall records in *The Royal Victoria Hospital 1903-1953* that in 1937 there were 700 biopsies and in 1953 there were 6,000 and necropsies increased from 200 to 1,000. The late Dr Sidney Allison, during his years as hospital archivist, penned the following unpublished account of John Henry and it has reference to these early years. Dr Allison writes, "My first meeting with him was in 1930 at a dinner. I had just come to Belfast and at the time he was working as pathologist at QUB. Later he returned to his alma mater as Professor of Pathology. He had spent over a year working at Johns Hopkins Hospital and I remember observing the almost 'tonic' effect which work in one of the foremost hospitals on the other side of the Atlantic had had on him. Proof of this was soon forthcoming when he published his important paper with Colin Campbell on the relation between existing diabetes

insipidus and lesions of the pituitary. An admirable thing Biggart did was to reorganise the clinical pathological conferences which had been held in the King Edward Memorial Building from 1931 onwards, usually under the chairmanship of Thomas Houston. Under Biggart's chairmanship, the plan of the meeting was for three or four cases to be presented and discussed between 4.30 and 6.00 p.m. The procedure was for the physician or surgeon to give an account of the clinical aspects of the case and then one of the pathology staff to project slides on the screen and detail the pathology, after which a short general discussion took place. Right from the first meeting these were an outstanding success. The same advantage was to be given to Cecil Calvert and myself in 1947 when once a month we assembled with the Professor and his assistants at brain 'cut-ups' where again the clinical features presented in a case during life were compared with the post-mortem findings. In my own special field, multiple sclerosis, it was of interest to find after the second World War, on application to the Schools of Pathology in Glasgow, Birmingham, Manchester and London, how few records there were of post-mortems in this disease, whereas, in Belfast, Biggart could turn up some 14 examples at that early date without difficulty. I have often thought with such an active, research-minded attitude as he possessed what he might have done had he remained in the scientific field, but in effect the pressure of administrative business, the coming of the health service and the shaping of the future of the Belfast School of Medicine were more than sufficient to take the place of personal research. This missing element in his later career, however, was brilliantly displayed by members of his department, John Edgar Morison and Florence McKeown. Indeed, there can be little doubt that burying himself in pure research would not have satisfied the urge within, as strong on the day of his retirement in 1971 as it had been in 1946, to take an active guiding part in the affairs of the school and in the destinies of the students, of which he never lost sight".

Desmond Neill recounts another example of John Henry's character, and it also refers to the lab. meetings. "An ability to do his 'homework'. With the passage of time, more case presentations at the lab. meetings had some discussion about biochemistry. 'Desmond, there is a case coming up with abnormal lipids. Write me half a page on that. Not too complicated. Something I can just let drop as the discussion develops'." Another story of Desmond Neill: when appointed as a basic grade biochemist at the RVH, he saw an advertisement for a senior grade biochemist in the Belfast City Hospital and went to see Professor Biggart to ask either his permission to apply or else act as referee. "Desmond, just you come back up to your laboratory and get on with your work. I have plans for you". (One would comment in passing that the plans seem to have had a degree of success).

It would be a recitation of facts known to most of this audience if I outlined John Henry's career from 1937 onwards. He was Professor of Pathology for 34 years and Dean of the Medical Faculty for 27 years. In order to record fully his portrait I reproduce the extract from *Who was Who,* 1971-1980.

John Henry Biggart was elected Dean of the Medical Faculty at its 271st meeting on 25th April 1944. Item 12 of the minutes reads: "On the motion of Professor Lowry, seconded by Professor Thomson, Professor J.H. Biggart was elected Dean for 1944-5. Professor Lowry moved and Professor Thomson seconded and it was carried unanimously that the thanks of the meeting be given to Professor J.W. Wilson for his services as Dean for 14 years". Professor Biggart remained Dean of the Medical Faculty for 27 years. I asked in the Faculty office was he ever concerned that this was too long. The reply was "No. He was proud

of it". I asked was he ever opposed for re-election. The reply was, "Who would have been so brave?" A temporary diversion in the story is to look at the history of the Deanship of the Medical Faculty. The second meeting of the Faculty was on October 21st 1851. Present — Burden, Carlisle, Gordon, Stewart, Ferguson. "A ballot being taken for the election of Dean of Faculty, Dr Ferguson was elected". But at a subsequent meeting on May 1st 1852, "The Dean having read the resolution of council respecting the election of Deans, Faculty proceeded to ballot — Dr Ferguson 3 votes, Dr Gordon 2 votes". This second election within seven months of the original election was presumably based on the Statutes of Queen's College (1849) that each faculty must elect the Dean annually.

The new Statutes of Queen's College of 1863 omit any reference to the constitution and powers of the Faculties: all the professors of the university are grouped in a single corporate body and I do not think there was a Dean of the Medical Faculty between 1863 and 1909. A book of Minutes starts 10th September 1891. The meeting is described as, "Meeting of medical Professors". The President of the College, Dr Hamilton, was in the Chair. There are Minutes of meetings through 1891 to 1907, the President often in the Chair, but Professor Cuming in 1896 and Professor Whitla in 1902. Professor Byers chaired meetings in 1903 interspersed with the usual chairman President Hamilton: an insight into relationships. 16th May 1907: "The President explained the objects of the meeting, then called upon Professor Sinclair, Senior Professor of the Medical Faculty of the College, to take the Chair which he vacated and then withdrew from the meeting". Recent fashion, from 1970, has been a Medical Deanship of 3 or 6 years and I had assumed this was a previous norm. Not so.

1910 — 1913	Professor T.H. Milroy
1914 — 1929	Professor Symmers
1930 — 1943	Professor W.J. Wilson
1944 — 1971	Professor J.H. Biggart

The statutes of Queen's University 1909 show that the office of Dean of a Faculty is extremely powerful, granted his autonomy depends on yearly election. He is the executive of the Faculty, in charge of the business of the Faculty meetings, a member of all committees of the Faculty, in charge of applications to the Faculty and degrees. In fact, a role that demands the exercise of power. As holder of the Dean's office, John Henry Biggart was endowed with the rare gift of a man who was able to make up his mind and that is an initial premise in any assessment of him. For instance, Sinclair Irwin on the RVH planning committee wanted to discuss some future planning which Sir John resisted being sent to a committee, "You compromise if you have too many people deciding on a committee".

What was achieved during the 27 years as Dean? The building of all the extensions of the medical school, first on the RVH site in the 1950s and on the BCH site in the 1960s. George Cowie describes the development of the preclinical departments alongside the BCH site — the previous place of the Deaf and Dumb and Blind Institute. Although the former building was of architectural interest, it was in bad repair so the only solution when Queen's acquired it was to clear the area for rebuilding. Sir Eric Ashby (later Lord Ashby) strongly favoured building a new Union and student residential and recreational facilities. Not everyone was in total agreement with this, particularly as the site seemed too small and two main roads — Lisburn and Malone — separated it from the main university. Sir Eric's plan was however adopted at several meetings of the Development Committee at which John Henry was present but said little, if looking unenthusiastic about the

project. He was challenged after one meeting as to why he did not voice his obvious opposition. He replied, "Sir Eric Ashby will be the Master of Clare in seven months time. We will later change the plan when he has gone and build the preclinical school there". Interestingly, in terms of relationships between people, George Cowie felt that Sir David Kerr and Michael Grant relied absolutely on John Henry and accepted all his advice, but that, though Sir Eric Ashby often consulted him, he consulted other people as well. The story of the preclinical building reminds me of a remark of Richard Womersley's: "He was tough and charming. He got things done". I do not wish to present John Henry in any way Machiavellian, except on occasion with the most altruistic motives for his medical school. Both his secretaries in Faculty office and Pathology testify almost in the same terms. "He was never devious. We never knew him to go back on his word". A former student said, "There were no side doors to John Henry". (I would like to express especial thanks to Mrs Colleen Jackson and Miss Thelma Tennis for particular help in this context).

More important than buildings was the expansion of the medical school in academic staff. Full-time chairs were established in the main clinical disciplines — in many of these instances the appointees were largely John Henry's choice and more often than not he chose well. Perhaps there was also an underlying intent to minimize any parochialism in the medical school. There was balance in his intentions. Professors Bull, Rodgers, Wate, Pritchard and Greenfield were not Queensmen, but Queensmen Professors John Gibson, Dundee and Archer were encouraged to come home again. As regards parochialism, Professor John Young had warned George Cowie when he was leaving Aberdeen to be "particularly careful in Belfast for many of the medical families were related" and he was further warned not to assume that the relationships were always on a hundred per cent friendly basis. John Dundee with Chestertonian insight says of John Henry. "During his time as Dean he didn't know the meaning of the word 'democratic' but nevertheless he was very much a democrat". He remembers John Henry reflecting on anaesthetics and the intention to create a Chair in the subject. John Henry reminisced on the time he had given anaesthetics when he was a medical student, and went on to reflect that he didn't see the need for a chair in the subject and then added "but if others want it, I will support it". Sir John's account, in his papers, "My first morning in surgery I was placed on the anaesthetist's stool and gave three anaesthetics for some abdominal complaints. One sat and watched the respirations of the patient and gauged the efficiency of the anaesthetic by the tension used on the abdominal retractors and the temper of the surgeon". The Chair in Anaesthetics was founded in 1958, the fifth such Chair in the United Kingdom among 25-30 medical schools. The Chair of Ophthalmology — one of our brightest jewels I would add — was certainly "plotted". The Institute for the Blind saw that its services, as originally envisaged, were not needed and a scheme was hatched between Harold Clokey, Gavin Boyd and John Henry to alter the rules of the charity and reorganize its various monies to fund a Chair in Ophthalmology. At the same time, an aged aristocratic wealthy patient of my own was also potentially involved, provided his will was worded as had been hinted, and I remember meeting John Henry on several occasions about that time when he would enquire solicitously, "How is Sir Charles?", although a degree of his ill-health might possibly have been advantageous to the founding of the Chair.

Lord Richardson wrote about John Henry and the GMC in May 1979. "He had been on the Council since 1951, and for those days when new members were kept in their places it was notable that he was elected to committees within two years of his arrival. He served on the Disciplinary Committee or on the Penal Cases Committee virtually without break from 1954 to 1970. He was on the Executive for 23 years. I think he must have been a happy man. His mouth was singularly untouched with bitterness, although it had a considerable firmness, and I always found his presence reassuring. He was a comfortable person to have around. His advice, not readily volunteered, not in committee coupled with loquacity, where his presence was characterised by silence, was, when it came, clear and well-defined".

To Sir John Biggart is attributed the concept of the joint appointment system between the Health Department and the University. It is not easy to ascertain with certainty his hand in the origin of this scheme but the Vice-Chancellor Dr Peter Froggatt writes, "At this time (1948-1950) John Henry was of course Dean of the Faculty and for all practical purposes ran everything to do with educational and academic medicine involving the University and the Hospitals Authority, at least in so far as Sir Eric Ashby (Vice Chancellor 1950-1959) would allow him — and sometimes that wasn't very far! However, as you probably know, John Henry kept no records and when he did write letters they were in hand and no copies were kept". Dr Froggatt forwarded the Senate Minutes of 1950 which set out the reasons for the joint appointment system as outlined by the Northern Ireland Hospitals Authority in 1949. I quote, "The arrangements in Great Britain are not regarded as the best possible procedure for the following reasons:-

1. The Boards have to accept as Honorary Officers persons in whose selection they have had no voice;
2. A person whose salary comes from only one source necessarily must always put first the interests of the body providing his salary;
3. Honorary Officers naturally feel that they are entitled to more freedom and greater privileges than salaried officers;
4. The possibility of friction and of incomplete co-operation between Honorary Officers and the Boards or between Honorary Officers and other Officers cannot be dismissed as unlikely or as fanciful".

As incoming President of this Society one is expected to be as gracious and sensible as personality allows — which isn't much. It is impossible to be all things to all men on the joint appointment system. Its virtues are as outlined and its vices are that it only gets a grudging acceptance from both sides of the marriage. NHS staff sometimes feel that the University derives excessive benefit and facilities; the University staff feel that their contribution to the NHS in time, energy and ability are underestimated. Would it be sufficiently gracious of me to say of the scheme that one hopes its virtues exceed its vices and also to reflect it is a two centuries old problem. (For reference read Dr Froggatt's account of the first medical school at Inst.).

In all I have said, I am conscious that one's account of Harry Biggart is relatively close in time to his life and that the excellent lives of Whitla and Thomson were written after a 20-year interval which creates a clearer perspective. I accept that, but my personal ambition in the task is not to cast the cold eye of the passing horseman. Harry Biggart's role in life was large, the nature of the man was even larger, and I wish to convey that in the way it has been conveyed to me by many

previous students. Dr George Mogey of the GMC, a former student, wrote, "He became the father figure of Queen's medical and dental graduates; many of them continued to write to him from all over the world often seeking his help and advice. He would say, 'Don't come to me when the trouble has broken around you. Come when the clouds are on the horizon'."

I hope that I will make this portrait of him more complete by recounting incidents in which he was involved — anecdotes which should illustrate his personality better than any formal account of his achievements. He was a compassionate disciplinarian. Dr Jimmy Riddell remembers a fellow student summoned to the Dean's office for admonition. "You have been reported to me by the Dean of Residences". "Yes Sir". "Make sure it doesn't happen again". The culprit was never sure whether the crime or the reporting by the Dean of Residences was not to happen again.

A friend of mine did very badly in the Pathology examination and was before the Dean and lectured at length about this lamentable performance, but finally interrupted John Henry saying, "Well, Sir, if it is as bad as that I will give up and go back to my father's grocery shop". John Henry replied, "Don't be so hasty, We are not putting you out yet. There are very few like you left around here". (One student, allowed to sit the 2nd MB five times — against every precedent — later became a very famous and distinguished surgeon). Denis Gough remembers in the early 1950s Basil Gray becoming engaged while still a medical student — unusual in those days — and John Henry during his 12 o'clock lecture on that day interjecting a few strictures about the disadvantages of early marriage to a doctor's career, which, as far as the class could see, was directed to another student, Wilson Clark, whom he had mistaken for Basil Gray. This was rather unusual because he had a startlingly good memory of all the undergraduates over many years. He met Randall Hayes at his year's reunion dinner. "Ah, Hayes, did you get into medicine through your academic ability or rugby?" "Both Sir", was the reply, and after several seconds John Henry said, "We didn't do too badly did we?" (Anyone who entered this medical school between the early 1940s and 1970 will doubtless endorse that).

Dr Froggatt and Professor Roddie relate that, when interviewing possible applicants, he would ask about their sports activities. The reply might be, "I played in the Irish schoolboys' tennis team". John Henry, "What else?" "Stroked the first eight and played interprovincial hockey". "What about rugby?" "Sorry, had no time for that". John Henry would note down opposite the applicant's name, "No sports". A visiting American academic in another department expressed a specific wish to see John Henry before leaving. "I want to talk to him about selecting students for medicine. I hear he has no difficulties. We have terrible trouble back home. We do it on a computer. The smart guys take the course all right, but can't relate to people. The others flunk out". The excellent — the very excellent — secretary of this Society, Dr Philip Reilly, son of a medical family, went to Clongoes — didn't do science, but got first place in geography and history in Irish Leaving Certificate. Knowing John Henry's humanities background at Inst. one suspected he might have been tempted to take him. "Reilly, we will take you in. If you fail your first year exams we will kick you out". (Philip's memory of the interview was, "You certainly knew who was in charge").

In Sir John's papers he writes about his own children taking up medicine. "Both, in spite of living in a medical household, eventually chose to become doctors. Indeed it is a curious thing that, in spite of the full life of medical practitioners and

of the frequent complaint that their services are financially undervalued by the community, their offspring so often strive to follow in father's footsteps. Previously they were welcomed to the parental medical school but today, though they may be starry-eyed, embellished with the requisite characters, and often well informed of the difficulties of the profession, the impersonal selections of UCCA often lead to disappointment. Admission systems are very fragile, and often produce surprising results. Protagonists of this or that system are often militant in their advocacy, but in later years I always insisted on an interview. It is difficult to tabulate what one learns at an interview, but one hoped that one was always sufficiently subjective to appreciate something of the character of the candidate". Perhaps in fairness I should allow a subsequent Dean his comments on medical student selection. Professor Ian Roddie in the last month has written in *The Lancet*, "My trouble with interviews is that I tend to prefer people who think like me and share my interests, especially girls, though in my less egotistical moments I know that to cast all future doctors in my image would not be good for medicine".

One recalls the weary cynicism of Daniel Coit Gilman, first President of the Johns Hopkins, who opined that the medical student was the one too weak to work on the farm, not clever enough to be a lawyer and too immoral to put in the pulpit.

David Hadden must have found Sir John on an 'off' day, when he was deputed as BMSA representative for his year to approach the Dean for more recreational facilities for medical students — for instance, a sitting-room to retire to between lectures. The reply to David was that if things were as bad as that he would arrange for extra stools in the Pathology Museum so that everyone could have a place to go between lectures. (And extra stools were indeed provided. So truly it was an 'off' day from his usual mood).

In daily contact with a multitude of colleagues, staff, undergraduates, he was uniquely friendly and a conversationalist without any condescension, but always a strong sense of humour. Professor Frank O'Brien remembers, "I got on well with him" — thought it was because "I was a Southern Catholic". He had the impression that John Henry was never particularly attracted to the more rigid tenets of Ulster Protestantism — while being solidly "pro-British" — and felt that he was essentially a liberal and very reasonable man. In one passing encounter, Frank O'Brien said to him, "Isn't it a remarkable thing that most of the artistic endeavour in Ireland is the prerogative of Catholics?" John Henry said, "Let me think about it". And a few days later took up the conversation, "Maybe you are right, it is all due to the Mass — the influence of the incense and the vestments".

John Henry met a classmate of mine, Miss Mona McQuitty (now Dr Mona Harley) a few days after her medical jurisprudence paper. A question was, "Discuss the medico-legal problems of a 16-year-old boy having had intercourse with a 13-year-old girl". "Miss McQuitty" he said, "You wrote a very good answer. In fact, all the girls in the year did well, but I will have to enlighten the young men in the year".

He was indulgent of his own junior staff. The following story illustrates this but, more especially, his instant and undiluted authority on any matter. Sam Nelson recounts, "In early 1965 I was making arrangements to go to the USA. I applied to the University for a scholarship and awaited anxiously. One morning Miss Tennis contacted me, 'The Dean wants to talk to you. Can you come to his office in about a half-hour's time?' The conversation was as follows:

John Henry: 'Morning Nelson'.
Nelson: 'Morning Sir'.
John Henry: 'You want some money from the University?'
Nelson: 'Yes Sir!'
John Henry: 'What are you going to do in America?'
Nelson: (Explanation about research into donor selection for transplantation).
John Henry: 'How long will you be away?'
Nelson: 'One year, Sir, perhaps two'.
John Henry: 'And you intend to come back to Belfast?'
Nelson: 'Yes Sir'.
John Henry: 'That will be all right about the money then'.
Nelson: 'Thank you, Sir'.
John Henry: 'That will be all'.
Nelson: 'Thank you, Sir'.

Next morning, Sam Nelson had the cheque for the full amount of the scholarship.

Dorothy Hayes had a similar interview in which she was advised not to go to America because the funds she was being offered were insufficient to maintain her (I suppose in the manner to which she was accustomed). The interview ended unsatisfactorily but she met the Dean a few days later — simple one sentence, "I have arranged that the University give you another £700. I knew you could never manage on that amount".

Sir Lucius O'Brien's toothache illustrates his pragmatic qualities. George Cowie recalls, "John Henry in the early days tolerated — perhaps did not actively support — the need for developments in the Dental School. (In defence of this attitude, availability of money was the problem). A member of the Senate, Sir Lucius O'Brien got toothache, sat for two hours in a corridor in the KEB where the dental department then was, described the conditions as "squalid and a slum" at Standing Committee. John Henry who had not altogether been either positive or negative on the need for improvement prior to this, agreed that something had to be done. As George Cowie says, "When it came to the crunch he didn't dig his toes in".

A distant relative of John Henry had a medical student son who did badly in his exams and John Henry was approached to see if he would offer any advice to the young man. The only comment was, "Work! Work!" — Oslerian advice. "Work" — the master word in medicine as Osler put it, "Though a little one, the master word looms large in meaning. It is the true philosopher's stone which transmutes all the base metal of humanity into gold".

I will not quote from well-known papers of Sir John's, such as 'Parergon', but there are two small neglected guest editorials on medical education in the *Ulster Medical Journal.* About medical education in 1962 he opens with, "Things are not what they used to be" and ends with "The complete doctor is adept in the science, proficient in the art, sincere in the ethic and embellished with the culture of medicine. How, inside the framework of a relatively fixed curriculum and within a fixed period of time is this ideal to be attained?"

The 1963 editorial ends, "In our own school we have all endeavoured to lead the student to the belief that in spite of all its apparent fragmentation of the advances

which sometimes come here and sometimes there, there is but one medicine and one medical problem — the sick patient". These words represent, I think, his statement of faith with regard to medical education.

Sir John Biggart chaired his last meeting as Dean — the 489th meeting — on 29th June 1971. He wished Professor Froggatt all success as Dean and urged him to endeavour "to preserve the entity that is the Faculty of Medicine". John McKnight described him in his later years with regard to the Postgraduate Council as not particularly initiating anything new but always being a great source of strength and protection, and he particularly remembers the string of people who came to ask his advice. The success of the Postgraduate Council owes much to his senior statesman involvement. I am not going to attempt to emulate the magnificent obituary tributes of Dr Froggatt or Professor Roddie. Lord Richardson wrote about John Henry's death which occurred on 21st May 1979 while attending a GMC meeting in London. Lord Richardson said, "His mother and father both died suddenly and it was his wish that this should happen to him. He got his wish, as he did in many things, so I believe, and we must be grateful for that". I intend to say no more that would constitute an emotional tribute except to repeat John Henry's quotation used at the time of Sir Thomas Houston's death, "Cease not, till day streams to the West, then down that estuary, drop down to peace".

The portrait I have of John Henry has been created for me by many people — almost universally in total affection. Any dissenting voices were dictated by a view that he was at times too determined and too powerful, but when I analyse the various stories about him, that determination and power were always clothed in the velvet glove of dedication to medical education in general, and to this medical school in particular, and to the highest and enduring concepts of medicine as a caring vocation, in which the student, the doctor, would "be embellished with the culture of medicine". This medical school that was the vision of James McDonnell was eventually established by Queen Victoria "in or near the city of Belfast in the province of Ulster in Ireland". The Queen's medical school was never likely to be blessed by its geography or the cursed history of this island, but it was to be blessed by the loyalty and service of many devoted sons. In the almost 150-year history of Queen's no one controlled the destiny of the medical school for so long or so totally as Harry Biggart — and no one controlled it to such purpose.

ACKNOWLEDGEMENTS

My admiration goes to Lady Isobel Biggart. My thanks to many, some mentioned in the text, but others provided much background mood. Particular thanks to Dr Denis Biggart; Dr Hugh Caldwell, Archivist, RVH; Mr Leslie Stuart; Mr Norman McMullan and the staff of the RVH Photographic Department and to Miss May Weller for truly excellent secretarial help.

BIGGART, Sir John Henry, Kt 1967; CBE 1948; DSc, MD; FRCP, FRCPath; Director of Institute of Pathology, Queen's University, Belfast, 1948-71; Dean of Faculty of Medicine, 1943-71; Professor of Pathology, 1937-71; Pro-Vice-Chancellor, 1967-71; Pro-Chancellor, 1972; *b* 17 Nov. 1905; *s* of John Henry Biggart and Mary Gault; *m* 1934, Mary Isobel Gibson, Knock, Belfast; one *s* one *d. Educ:* Royal Belfast Academical Instn; Queen's Univ., Belfast; Johns Hopkins Medical Sch. MB (Hons) 1928; MD (Gold Medal), 1931; DSc 1937; MRCP 1952; FRCP 1957; FCPath 1964; Hon. FRCPI 1969. Commonwealth Fellowship, Johns Hopkins, 1931-33; Pathologist to Scottish Asylums Board, 1933-37; Lecturer in Neuropathology, Edinburgh Univ., 1933-37; Regional Dir, Blood Transfusion Service, 1936-46. Robert Campbell Orator, 1948; Mem., University Senate, 1948; Chm., Laboratory Services Cttee, Hospitals Authority, 1948-54; Chm., Medical Education and Research Cttee, Hospitals Authority, 1950-64; Gen. Med. Council, 1951; Gen. Dental Council, 1959; Chm., Standing Med. Adv. Cttee, Min. of Health, NI, 1967-73; Council, Brit. Empire Cancer Campaign, 1968; Council, Coll. of Pathologists, 1968; Chairman: NI Council for Postgraduate Med. Educn, 1971-79; Irish br. Council, GMC, 1971-: Belfast Home for the Blind, 1972-; Marie Curie Beaconfield Home, 1969-; Age Action Year (NI) 1976; Vice-President: NI Mental Assoc.; NI Br., British Empire cancer Campaign; Pres., NI Muscular Dystrophy Assoc., 1972-; Hon. FRCGP, 1971; MD (*hc*) Dublin, 1957; Hon. LLD QUB, 1971; Hon. DSc NUI, 1973. *Publications:* Text Book of Neuropathology, 1936; papers on general and nervous pathology in Brain, Jl Pathology and Bacteriology, Ulster Med. Jl, and Johns Hopkins Bulletin. *Recreations:* reading, writing, gardening, music. *Address:* 64 King's Road, Belfast. *T:* Belfast 653107. *[Died* 21 *May* 1979.

The Ulster Medical Journal, Volume 54, No. 1, pp. 20-29, April 1985.

The changing face of medicine

Annual Oration at the opening of the 1984-1985 teaching session, Royal Victoria Hospital.

Derek S Gordon

A teaching hospital like the Royal can hardly be said to have one Founder. But James McDonnell, more than any other, deserves the title of Founder of this Hospital. In 1827 he delivered an address to students entering the Belfast General Hospital in Frederick Street, later to become known as the Belfast Royal Hospital. Since then, at the beginning of each academic year, a member of the staff has welcomed the students entering their clinical training. This year it is my honour to address you, and to welcome you, on behalf of the medical staff of the Royal.

I hope that you read Tolstoy. His novels are marvels of sustained imagination. I think that *Anna Karenina* is perhaps the greatest of all novels. The opening sentence reads: 'All happy families are alike but an unhappy family is unhappy after its own fashion'. I am sure that you have already seen enough of the Royal to sense that relationship between staff and students which bears the hallmark of a happy family. You will enjoy the time you spend in the wards of the Royal. That is where you will learn the art of medicine.

You may wonder how a hospital of such singular character came into being. In 1896 a fund was started to build a new hospital to commemorate the Diamond Jubilee of Queen Victoria.[1] The architect was Mr Henman from Birmingham;[2] McLaughlin and Harvey started building in 1899. When the staff of the Belfast Royal Hospital moved from Frederick Street to the Grosvenor Road their hospital acquired a new name — the Royal Victoria Hospital. King Edward VII and Queen Alexandra opened the hospital in 1903, the building having been completed in four years. The Royal then had 204 beds; now it has 1,099. The staff for all duties then was 121; now it exceeds 5,500. Doctors in 1903 formed 30% of health workers; now they form only 8%. Here we take our first look at the changing face of medicine — cost. The hospital budget in 1908 was £20,000. When the NHS started in 1948 the beds had increased to 538 with a budget of £236,000. Today's budget with just over 1,000 beds is £45 million. In 1948 the entire Northern Ireland Health Service cost £2.5 million; now it costs £650 million. A private bed in 1946 cost 7 guineas a week; a bed in the Royal today costs over £1,000 a week. If the price of petrol had risen at a comparable rate a gallon would now cost over £20; by the same reckoning a bottle of gin would cost over £100. We shall return to these figures later.

In 1903 the hospital not only acquired a new name, it became an architectural milestone in its solution of some old problems in hospital building. It was in the Crimean War that Florence Nightingale insisted that sick rooms needed ventilation. In 1854 she arrived at Scutari Hospital near Constantinople.

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

Wounded soldiers were pouring in from Sebastopol and Balaclava. She found that 400 patients in every 1,000 were dying from infections. The battlefield was dangerous; the hospital was even more dangerous. After she insisted on improved ventilation and sanitation the mortality from infection fell to only 22 per 1,000. She said 'It may seem a strange principle to enunciate as the very first requirement of a hospital that it should do the sick no harm even if it does them no good'.[3] Her views on hospital design became reflected in the pavilion style of architecture. War had been the stimulus for change.

The pavilion style dominated hospital building in the second half of the 19th century. The buildings were cold, draughty and isolated; they were inefficient to run and unpopular with staff. But they were healthier than their damp, airless predecessors. Why then were the wards of the new Royal Victoria Hospital designed and built as they are? They were built side by side with no space between them and with no opening windows.[4] At first the medical staff, which included Sir William Whitla, regarded the plan as outrageous — 'like a cross between a factory and a prison'. Light was to be admitted through clerestory windows in the ceiling; fresh air was to be driven through the entire building. The architect said 'It is a revolution in hopspital design'. Professor Banham of University College, London, wrote 'The Royal Victoria Hospital became a building air-conditioned for human comfort, and very likely the first in the world'.[5] The design also made for great efficiency.

The ventilating system starts in a building between the laundry and the mortuary. Huge fans, about nine feet across, made in the Sirocco Works, drive air along a tunnel under the main hospital corridor. Professor Banham writes 'The duct is one of the most monumental in the history of environmental engineering; a brick tunnel with a concrete floor over 500 feet long and nine feet wide, 20 feet deep at the input end and only six feet deep at the downstream end'. How many of you know as you walk along the main corridor that you could drive a double decker bus into the tunnel under your feet? In a Third Programme broadcast, later published in *The Listener* in 1967, Professor Banham said 'exploring the tunnel was like pot-holing'. The entrance is like 'one of the ante-chambers of Hell . . . a pit of utter blackness, filled with a roaring relentless gale of wind'. The air reaches the wards through openings in the walls and is vented through those pagoda-like turrets you see on the roof. As the nurses can tell you, the system is by no means perfect; but it was unique. Since the Royal introduced air-conditioning to the world there have been many architectural advances. Recent buildings on this site are refined successors to the main corridor part of the hospital.

New hospital buildings are important; but you will come to see that even in the newest and most expensive institutions, the strength of the hospital or medical school lies not in its wealth or buildings but in the quality of its staff. And the quality of the medical staff naturally depends on the quality of its students. If 'A' Level results are to be relied on, the future appears to be in safe hands. You seem to be unusually intelligent and industrious. Medical students have not always been held in high esteem. Dr William Drennan was an important figure in late 18th century Irish medical circles. He wrote of his Edinburgh undergraduate days: 'A student of medicine is a term of contempt, but an Irish medical student is the very highest complication of disgrace'. In the 19th century William Dale remarked that 'Drinking, smoking and brawling were the very rational occupations of the dissecting room'. After they qualified, the students seemed to change. At least Robert Louis Stevenson seemed to think so, when he referred to doctors as

'men who stand above the common herd'. But in this century, in spite of the distinction of British medicine in its own eyes and in the eyes of colleagues throughout the world, we as a profession seem curiously irrelevant in contemporary society. If you visit the National Portrait Gallery this year, you will see an exhibit of 500 portraits of leading figures in the 20th century. It includes politicians, industrialists, trade union officials, lawyers, pop singers; there are three physiologists, a few forensic pathologists, a portrait of Marie Stopes (an early expert on birth control) and one of Grantly Dick Read, the proponent of natural childbirth. But among these 500 portraits there is not one clinician or nurse. Tony Hancock is there but no Lord Moynihan, Lord Horder, Lord Smith or Geoffrey Jefferson. In Anthony Sampson's book *The new anatomy of Britain* (a survey of the influential groups in our society) he mentions the Health Service only twice. Firstly he says that 'in its treatment of women the Department of Health might almost belong to Islam'. Secondly he recalls that the Ombudsman found the Department of Health second only to the Inland Revenue Service in its record of maladministration. I know that you will find that medicine is a world-wide profession with a homogeneity not known in other professions. I hope that in your electives and after you qualify you will travel abroad and visit overseas hospitals where you will immediately feel at home.

The popularity of medical schools has never been greater. For many years throughout Europe students have come to medicine like the oysters flocking towards the Walrus and the Carpenter — 'And thick and fast they came at last, and more and more and more'. But not all will meet the fate of the oysters. The EEC encourages overproduction, not only of beef and butter and wine, but also of doctors. In 1975 the United Kingdom had 18,000 medical students; Italy had 179,000, France 102,600, Germany 40,000 and Belgium 14,000. In that year, 1975, one person out of every 312 in Italy was a medical student, in the Republic of Ireland the ratio was 1:1300 while the United Kingdom figure was 1:3000. The effects on education and career prospects are well-known.

The strength of British medical education is the degree to which the hospital service is available to you. In some European countries training is so theoretical that the student rarely comes face to face with the patient. In the British system a large number of doctors provide small group tutorials — a system impossible to copy in most other university faculties. Formal lectures play a relatively small part. The importance of learning the basics of medicine in the ward, by the bedside, cannot be emphasised too strongly. In addition to clinical diagnosis you will learn a professional attitude to patients and relatives. The doctors who teach you may not have been appointed because of their piety or moral respectability. However, it is in the tutorial system that the ethical dilemmas of medicine are best discussed — the treatment of the incurably ill, the prolongation of a life without hope, the saving of a young life with no prospect of normal development.

These dilemmas have increased with the development of high technology specialties. A degree of specialisation can be seen in most wards of the Royal; most of the wards have patients referred from all over Northern Ireland. There has been a fear that specialties might swallow beds better employed in a teaching hospital for traditional general medicine and surgery. General medicine and general surgery remain the backbone of hospital practice. A specialty service cannot provide an acute service for a community. However, specialisation represents a natural evolution or growth in medicine. To remove the specialties from the student's view would damage his interests. Specialties often furnish

admirable lessons in applied physiology. As long as the emphasis is away from technical affairs and operations, the discipline of a specialty is a valuable training ground.

In the 20th century, the most dramatic change in medicine has been the emergence of specialisation. I would like to tell you about the development of my own specialty, neurosurgery, and then to discuss some of the implications high technology specialisation holds for the Health Service. The early history of most forms of surgery is difficult to trace. However, some remarkable archeological and documentary evidence outlines the development of neurosurgery. Surgical interest in the head goes back to the neolithic period. Many skulls from that time are found to have defects. Some defects clearly resulted from operations on fractures. Many more skull defects were caused by the operation of trepanning or trephining.[6] The shape of the hole in a skull treated in this way confirms that it was not caused by disease; the ingrowth of new bone shows that the operation was seldom fatal. The surgeons in the Stone Age used a flint or obsidian knife; later, crown saws or circular saw bits, not unlike modern instruments, were devised.

Trepanned skulls have been found in every European country, in much of South America, Asia, Africa and the South Sea islands. We cannot say where this extraordinary procedure started, or even whether it diffused throughout the world from one centre or appeared independently in different countries. Trepanning was probably used for epilepsy, for psychiatric disorders and for the release of spirits. The practice continued till the 20th century in parts of Europe. Indeed it is practised today in rural Zimbabwe for psychiatric disorders. In that community psychosis is not considered to be amenable to European medicine.

The first documentary evidence on surgical practice comes from the banks of the Tigris, the Euphrates and the Nile: that is from Mesopotamia and Egypt. Here again the record is most complete on neurosurgery thanks to the papyrus found by Edwin Smith in Cairo in 1862. It was probably the work of Imoteph about 3000 BC. Here are found the first descriptions of the meninges, the lateralisation of brain functions, the significance of head injuries which tear the dura mater and the different types of spinal injury. Less severe wounds were regarded as 'wounds to be treated'. The early Egyptians knew the importance of elevating depressed fractures. However, certain kinds of head injury and spinal injury with paraplegia were 'wounds not to be treated'. The importance of prognostic indicators and the efficient use of resources were well recognised 5000 years ago.

Greek neurosurgeons, especially from the Hippocratic school at Cos, wrote about head injuries and trepanning for epilepsy. Detailed operative procedures were outlined. By the 1st century AD, Celsus of Alexandria appreciated the need to diagnose and evacuate extradural haemotoma. Neuroanatomical and neurophysiological studies continued from Alexandria to the time of Galen, 600 AD. But from the decline of the Roman Empire in the West and through the Byzantine period we see a gradual decay in medical knowledge which continued till after the Renaissance. For example we read how an Army doctor, Surgeon Wiseman, relied on the techniques of Hippocrates to treat head injuries sustained in the Battle of Worcester in 1651. He said that if the Hippocratic methods were not used 'Putrefaction will set in; then will follow convulsions, howlings and dispatch of the patient'. Hippocrates died 2000 years before the Battle of Worcester.

Surgical advances remained leisurely till 100 years ago. Indeed progress was impossible before the bacteriological studies of Pasteur, the introduction of anaesthetics by Morton, and Lister's work on preventing sepsis in surgery. In the 19th century a surgeon could encompass all surgical knowledge. As the 20th century dawned, it became clear that specialisation was inevitable. The story of modern neurosurgery is contained in the lives of three men — Macewen, Horsley and Cushing. Sir William Macewen was born on the island of Bute in 1848. He became Professor of Surgery in Glasgow. He was the first to operate on a brain abscess and a meningioma. He carried out the first laminectomy for spinal tumour in 1879. By the time Horsley performed his first laminectomy, sometimes erroneously described as the first ever, Macewen had performed the operation five times. Macewen was offered the Chair of Surgery at the Johns Hopkins University in Baltimore but he said that he turned it down because the supervision and training of nurses would not be under his direct control. A somewhat autocratic figure, he was known to the students as 'The Great I Am'. After Macewen died in 1924 he was forgotten for many years. He has no grave; his body was cremated. His reputation even now has been only partially rehabilitated.

The first dedicated neurosurgeon was Sir Victor Horsley who was born in 1857 and became Professor of Surgery in London in 1884. In 1886 Horsley performed his first craniotomy in the National Hospital for the Paralysed and Epileptic in Queen Square. This was followed three years later by the first laminectomy outside Scotland. He soon introduced pituitary surgery and by 1890 had performed 40 operations on the brain. Surgeons from all over the world visited his clinic. In 1900, still only 43, he was the undisputed leader in his specialty but he made little further progress. He became increasingly eccentric; his extreme views on alcohol led him to spend much of his time at temperance meetings. He had few friends, the few he had he lost because of his imperious manner. Horsley died of heat stroke in 1916 on active service in Mesopotamia; his grave can be seen just south of Baghdad. Although Horsley has been called the father of neurosurgery, many contemporaries, including Ferrier, felt that Macewen deserved that title.

Modern neurosurgery was conceived in Scotland; it had its infancy in England, but it came of age in the United States. How often has that sequence been repeated. This brings us to Harvey Cushing.[7] He was born in 1869 and became one of the most remarkable of all surgeons. His BA from Yale in 1891 was followed by an MD from Harvard in 1895. On the 10th of January 1893 a patient suffering from strangulated inguinal hernia died in the Massachusetts General Hospital while being anaesthetised by Cushing, then a 3rd year medical student. Students gave largely unsupervised anaesthetics till well into the 20th century. Cushing realised that no anaesthetic record was kept which might give warning of impending disaster. He and Codman, a fellow student, then introduced the first chart for monitoring pulse and respiration in the operating theatre. Seven years later, when he was a surgical resident, he introduced the Riva-Rocci sphygmomanometer to the United States and incorporated blood pressure recordings in the anaesthetic chart. Such charts soon became obligatory throughout the world.

In 1897 Cushing moved to Johns Hopkins, where he came under the influence of Halstead. Halstead had learned from Kocher in Berne the technique of slow meticulous operating which Cushing was to introduce to neurosurgery. The elder of the Mayo brothers had little patience with this practice. He said after a visit to Baltimore, 'It is the first time I have seen the upper end of a wound heal before the

lower end is sutured'. In 1900 Cushing studied with Sherrington in Liverpool, with Horsley in London and with Kocher in Berne. His experimental work in Europe led him to appreciate the relationship between systemic blood pressure and intracranial pressure, a phenomenon so important that it was named the Cushing reflex. International groups now meet to discuss intracranial pressure and cerebral blood flow. Here was the beginning of their studies.

Cushing returned to Boston in 1910 to open the neurosurgical department in the Peter Bent Brigham Hospital. The first of his classical monographs appeared two years later. He described states of hypo- and hyper-pituitarism, acromegaly and what came to be known as Cushing's disease. It was now clear how dwarfs and giants came to be. Then came the 1914-18 War. Always an anglophile, Cushing came to Europe in 1915 with the Harvard Ambulance; later he became Senior Neurosurgeon to the American Expeditionary Force. His methods reduced the mortality of cranial missile wounds from 50% to 28%. Honours were showered upon him. In 1919 he was made Companion of the Most Illustrious Order of the Bath for 'exceptionally meritorious service as Director of Base Hospital No. 5'. Queen's University Belfast awarded him an Honorary MD.

In 1922 Dandy of Hopkins introduced air ventriculography as a means of localising cerebral tumours. Cushing was slow to adopt the technique, partly because he was not on speaking terms with Dandy, but more because he felt that neurosurgeons would not become properly trained in neurology and would neglect a full neurological examination. A close parallel exists today with the CT scan which already threatens clinical judgement. Without clinical judgement in diagnosis, said Cushing, how can you expect clinical judgement in treatment? Cushing's technical innovations included the silver clip for occluding blood vessels, the adaptation of suction to neurosurgery and the collaboration with Bovie to give one of the most significant advances of the century, the high frequency diathermy unit. By 1932 he had operated on over 2,000 brain tumours.

Cushing's biography of Osler brought him a Pulitzer prize for literary ability, and he followed this with biographies of Galvani and Vesalius. Now, as he reached his 60th birthday, Cushing's health was failing. He had smoked 60 cigarettes a day since he was an undergraduate at Yale where he entertained his fellow students by turning back somersaults from the library steps with a cigarette in his mouth. He could hardly walk 100 yards, he played croquet instead of tennis, his leg pulses had disappeared and he developed a deep gastric ulcer. He retired from surgery aged 63 and became Professor of Neurology at Yale. Cushing was a great public figure, a writer and orator, on first name terms with Roosevelt. He worked steadily till his death in 1938 aged 70.

How do you compare a giant of one age with a giant of another? Schiller felt that 'whoever has satisfied the best people of his time has lived for all times'. Cushing laid the foundations. He had two chief surgical attainments. First, he reduced the mortality of craniotomy from 60% to 10% — he made neurosurgery respectable. Secondly, and quite simply, he trained all the neurosurgeons in the world between 1919 and 1932. In every civilised nation the Cushing legacy persists. With the development of new techniques and better anaesthetists his results were eventually surpassed. Keats, also a doctor, understood the inevitability of gradual progress when he wrote in *Hyperion* 'So on our heels a fresh perfection treads: born of us and fated to excel us'.

In specialisation, Britain lagged behind the United States. Even in 1939 there were only three full-time neurosurgeons in Britain. Otherwise what neurosurgery was being done was by a few general surgeons who acquired by chance or force of circumstances some interest in the subject. Some of you will remember that Barney Purce led the way in Belfast. The 1939-45 War brought this to an end. 'War is the only real school for the surgeon' said Hippocrates. It became clear by 1941 that head injury treatment for battle casualties was inadequate. One of the lessons of military surgery is that the basic lessons are forgotten after each conflict. In the 1939-45 conflict the need for neurosurgical teams was appreciated only after Cairns at Oxford studied Cushing's original records from the 1914-18 War which were specially sent over from America. By 1942 the Oxford centre with men like Hugh Cairns, and Cecil Calvert from Belfast, was training young surgeons to man mobile forward neurosurgical units. The results for head injuries were totally convincing. Immediately after the war this group of neurosurgeons returned to found neurosurgical departments in every British medical teaching centre. The Belfast department was started in 1947 by Cecil Calvert whose name is remembered with special affection in this hospital. His papers on missile wounds of the head and on fractures of the base of the skull are classics. Sister Bell ran the theatre with a firm hand; Harry Shepherd, radiologist, and Fred Bereen, anaesthetist, were also founder members, joined later by Alex Taylor and Colin Gleadhill.

The changes since 1947 have chiefly been those of high technology medicine — complex, expensive techniques giving rise to a new breed of skilled paramedical scientists. An unforeseen expansion in neurosurgery and radiology was mirrored by equally expensive changes in other specialties, especially in the fields of anaesthesia, intensive care, laboratory medicine, cardiac surgery and isotope scanning. The majority of the 34 referral specialties have developed in the Royal. The hospital has doubled in size since the birth of the National Health Service. The attractions and the intellectual satisfaction from clinical medicine have never been greater.

But a cloud was appearing on the horizon. The changes in medicine from specialisation were beneficial but the cost was already leading to the crisis in health care which now affects most of the Western World. Under the present Government, the cost is being counted; coincidentally new forms of management are being introduced. The changing face of medicine for the future will reflect the joint impact that expensive high technology medicine, the recent administrative changes and Government policy make on our clinical practice. Let us look for a moment at how hospital administration began. Its origins coincide to a large extent with those of the nursing profession. The Reformation deprived Britain of the system of hospital services which had been supplied by the religious orders. Even in the first half of the 19th century, hospital building and nursing were still primitive. In 1860 Florence Nightingale wrote 'Nursing was undertaken by those too tired, too weak, too drunk, too stolid or too bad to do anything else'. I mentioned earlier Florence Nightingale's profound influence on hospital architecture. I wonder how many people appreciate the part she played in hospital administration.[2] She lived from 1820 to 1910. Because of family disapproval her practical nursing experience started only when she was 31; she retired from active nursing just five years later on her return from the Crimean War. By then everyone knew how the Lady with the Lamp had saved so many lives in the Scutari hospital and how she founded modern nursing. But her years

of study and travel before she started nursing gave Florence Nightingale an unprecedented knowledge of hospital practice and administration. One of her treatises was on the design of sinks in hospitals.

In 1858 she had been elected to the Statistical Society. In 1860 she worked with Sir James Paget and Sir James Clarke to draw up a classification of diseases; this was the birth of medical records and medical statistics. Then forms were drafted which would 'enable us to ascertain the relative mortality of different diseases and injuries among the classes which enter hospitals in different countries and in different districts of the same country'. Here was the beginning of medical audit—now, 124 years later, still in its infancy in many specialties. For the last 50 years of her life Florence Nightingale had a mysterious illness; a physical cause was never found. She could be seen only by appointment and gave most of her interviews lying on her couch. Few important decisions on hospital policy or planning at Government level were made without consulting her. She was the acknowledged expert on both civilian and military hospital planning. She remained the confidante of statesmen such as Gladstone and Lord Salisbury. The award of the Order of Merit in 1907 came as a surprise to the nation because most people thought she was dead. When she died in 1910 aged 90, six army sergeants carried her to her grave.

In those days hospital budgets were small and usually came from public subscriptions. Administration was through a Board of Management, Medical Superintendent or Chairman of Medical Staff and the hospital secretary. The Matron, at Miss Nightingale's insistence, had charge of the School of Nursing. Part of this system exists today in the London post-graduate hospitals. However, for the rest of the Health Service, the Heath Government in 1973 did away with Management Committees and gave us the multiple tiers of administration which confront us today. Decisions became increasingly remote from where the work was actually being done. One is reminded of how, after the Flood, the people built a tower in the land of Shinar. The top of the tower was to reach into heaven. Genesis II tells us that the Lord became concerned. He went down and confounded the people by creating different tongues. Because of the confusion of speech, no man could understand his neighbour and the people were scattered. The tower was called the Tower of Babel or Babylon. Each administrative tier of the Health Service has a different tongue. Those in each tier can understand each other but between tiers incomprehension exists. In Belfast we have been fortunate in our relations with administrative colleagues. But although the individual administrator may be efficient, the system prevents his becoming effective. If Florence Nightingale were to take her lamp and walk down the corridors of the Health Service she would, in the words of Griffith, be searching for someone who is really in charge.

Rising costs and rising expectations undoubtedly make for administrative difficulties. There is no such thing as a free Health Service. For a while the nation thought that there was. Between 1948 when the Service started until a few years ago the Health Service appeared to encourage the view that care should be provided irrespective of cost. However, the Health Service now costs over £15 billion a year. Over £300 per person or £1,200 for a small family. Along with Defence it is the Government's top spender. This vast sum is 5.5% of the gross national product of the United Kingdom. Some western countries spend more, but all face a similar financial crisis, a crisis with dangers for our profession. Treatment is better; but as a profession we find ourselves victims of our own

success; we can't afford — no country can afford — everything that modern medicine has to offer. Shortage of money therefore constrains clinical freedom. In the days before expensive specialisation when treatment was cheap, harmless and based on clinical opinion, full clinical freedom was often possible. We could prescribe as we chose for all our patients. Even now a patient in a district hospital may cost only £300 a week; however, if he needs specialist treatment in this hospital the cost can exceed £3,000 a week. Spend an hour with an accountant from the Health Service and you will appreciate the saying that, if economics is the dismal science, accountancy is the dismal practice.

Now we have to consider what we can afford. Rationing of health care by waiting lists is not new. In some fields — for example cardiac surgery — very ill patients who could benefit from modern treatment may die while still on a waiting list. There is rationing by expense: some procedures such as renal dialysis, CT scan, bone marrow transplantation and some forms of intensive care are limited by shortage of expensive equipment or shortage of staff. The onus of choosing who shall be treated often rests with the doctor, although examples of imposed rationing are not unknown in the Health Service. In spite of these economic constraints, we as a profession must ensure that a free atmosphere exists, an atmosphere in which advances can be made. The history of medicine is the history of men and women, not the history of committees.

While we must accept that clinical freedom is constrained by cost, clinical autonomy must be preserved. By this I mean that the patient remains the doctor's direct responsibility without scrutiny or interference. The choice of treatment rests with the doctor and so does the co-ordination of the hospital services for the patient's management; in this regard close collaboration with the nursing staff is of course fundamental. With very few exceptions the doctor in the National Health Service is responsible as an individual for his individual patient. Don't take this for granted. Clinical autonomy has never been recognised in some countries and in others — for example, the United States — it is coming under attack. But direct unqualified clinical responsibility, whether in general practice or in hospital, underlies what is best in British medicine. Better for the patient than in most other countries.

How is clinical autonomy to be safeguarded? First, we should be prepared to accept some responsibility for the budgetary control of our departments. This will be possible with computer-controlled hospital information systems; it is already possible in x-ray departments and laboratories. Secondly, we should participate in hospital management including the deployment of hospital resources. Thirdly, clinicians should conduct a medical audit to review ward policies and to modify the management of patients. These measures should preserve the clinician's ability to protect the care of his patient.

I have strayed a little from the immediate concerns and interests of the new students. The way before you is challenging. But I am sure that you are persuaded that you are entering a life which will carry great responsibility but will give you great satisfaction. Medicine is not a job: it is a way of life. In this privileged career your motto might well be that of the City of Belfast. It is said to be derived from the 12th verse of the 116th Psalm. "Pro Tanto Quid Retribuamus?" It may be freely translated "What return shall we make for so much?"

I am indebted to Mrs Maureen Bell for secretarial assistance, and for typing the manuscript.

REFERENCES

1. Marshall R. Fifty years on the Grosvenor Road, Belfast: W & G Baird Ltd., 1953.
2. Henman W, Lea H. Royal Victoria Hospital, Belfast; its initiation, design and equipment. *J R Inst Br Arch* 1903; 3rd ser, **9**: 89-113.
3. Woodham-Smith C. Florence Nightingale. London: Constable, 1950.
4. Reid H. The Plenum ventilation system at the Royal Victoria Hospital. *J R Vict Hosp League of Nurses* 1984; **35**: 13.
5. Banham R. The architecture of the well-tempered environment. London: Architectural Press, 1969: 82.
6. Walker AE. A history of neurological surgery. New York: Hafner, 1967.
7. Fulton J. Harvey Cushing—a biography. Springfield: Charles C. Thomas, 1946.

The Ulster Medical Journal, Volume 54, No. 1, pp. 30-40, April 1985.

Fetal rights

The first Belfast Royal Maternity Hospital Perinatal Lecture delivered on 7th September 1984.

J H M Pinkerton
Professor of Midwifery and Gynaecology,
The Queen's University of Belfast.

When I was in the Royal Navy during the last War we were always told that we had no rights to insist on, only privileges to be earned. Nowadays, rightly or wrongly, it is generally accepted the world over that everyone has rights: the right to life, to liberty, and to the pursuit of happiness. What then of the human fetus? As far as happiness is concerned I suppose we can never know for sure whether a fetus is, or indeed can be, happy or unhappy. Nevertheless those who through the ages have pondered our existence in the womb, whether poets or philosophers, saints or scientists, have usually regarded intrauterine life as a little paradise and the expulsion at birth into the outer world as a trauma or even a tragedy. As Wordsworth put it:

> 'Our birth is but a sleep and a forgetting:
> The Soul that rises with us, our life's Star,
> Hath had elsewhere its setting,
> And cometh from afar:
> Not in entire forgetfulness,
> And not in utter nakedness,
> But trailing clouds of glory do we come
> From God, who is our home'.

Similar ideas were also expressed by the English metaphysical poets of the 17th century. One of these, Thomas Traherne, an unpretentious country parson, long before the controversies of the 19th century about the relative importance of heredity and environment in the shaping of the individual, had come to the conclusion that 'our misery proceedeth ten thousand times more from the outward bondage of opinion and custom than from any inward corruption or depravation of nature; it is not our parents' loins', he declared, 'so much as our parents' lives that enthrals and binds us. The pure and virginal apprehensions and the divine light wherewith I was born, are the best onto this day wherein I can see the Universe. By the gift of God they attended me into this world and by His special favour I remember them from now . . . '. Few of us I fear have been vouchsafed Traherne's special favour. Perhaps just as well! — for although most antenatal experiences may well be pleasant, birth itself is hardly likely to be so. In the late 19th century this intrauterine paradise of the poets and mystics was invaded and shared by somewhat improbable partners when Sigmund Freud and his school of psychoanalysts used the concept of an intrauterine paradise lost to explain the whole wide variety of human neuroses. For them, anxiety first afflicts the individual during the process of birth, partly as the result of its attendant physical hurts and discomforts and partly in consequence of the concomitant

change from a highly pleasurable environment in the womb to an extremely uncomfortable one in the cold, noisy, odorous, boisterous, often painful outside world. They taught that this birth-provoked anxiety was the first content of perception — the first physical act, so to speak, and throughout life, just as this birth-malaise underlies every subconscious fear, so every pleasure anticipates the re-establishment of the intrauterine primary pleasure, the return to that dark unconsciously remembered place of comfort and peace where, in the words of another cleric-poet, the Jesuit Gerald Manley Hopkins:

> '. . . no storms come,
> Where the green swell in the havens dumb
> And out of the swing of the sea'.

It may be that we obstetricians sometimes aggravate this natal suffering with our oxytocin and our prostaglandin, our forceps and our scalpels; and recent suggestions that babes would be happier and healthier if born spontaneously into warm water may have some scientific or at any rate some psychological basis after all.

With regard to freedom from the womb I suppose no one in his right mind and not seduced by a serpent would want to leave or be liberated from paradise. But unfortunately the womb is not always paradise and can become a death trap. All obstetricians and perinatologists would certainly take this view and at least one poet regarded the womb as a prison.

No doubt John Donne was influenced by his own unhappy circumstances at the time. Early in 1631 the ill-starred Charles I was still on the throne and Donne, then Dean of St Paul's, was in rapidly failing health. On February 12th at the beginning of the penitential season of Lent he was terminally ill and barely able to drag himself up to Whitehall to preach before the King's Majesty the famous sermon soon to be published posthumously as 'Death's Duell'. He died shortly afterwards and is commemorated in St Paul's Cathedral by a macabre statue depicting him wrapped in a winding sheet standing on a funeral urn, with his eyes closed, his cheeks fallen in, his nose sharp as a pen. In his last sombre sermon Donne spoke of birth as an 'exitus a morte, an issue from death'. 'For' said he 'we have a winding sheet in our mother's womb, which grows with us from our conception, and we come into the world wound up in that winding sheet for we come to seek our grave'.

There is no doubt that in obstetrics the fetus for its own good must not infrequently be released, freed prematurely from the potentially lethal environment of the uterus, e.g. in placental failure or antepartum haemorrhage. Over the past 20 years there have been notable changes in the technique and incidence of induction of labour. The use of castor oil or quinine has been superseded by synthetic oxytocin and prostaglandins which are much pleasanter and much more effective. Older methods, involving the insertion through the cervix of foreign bodies like seaweed tents and rubber tubes, which relied largely if not intentionally on infection for their efficiency have been replaced by simple anterior amniotomy. And today induction-delivery times are measured in hours, not days. Infections are rare and caesarean section when necessary can succeed safely where induction has failed.

Apart from improvements in methodology there has been a remarkable ebb and flow in the volume of active interference in late pregnancy. As with most forms of therapy, fashion has played a considerable part in the use of induction of labour,

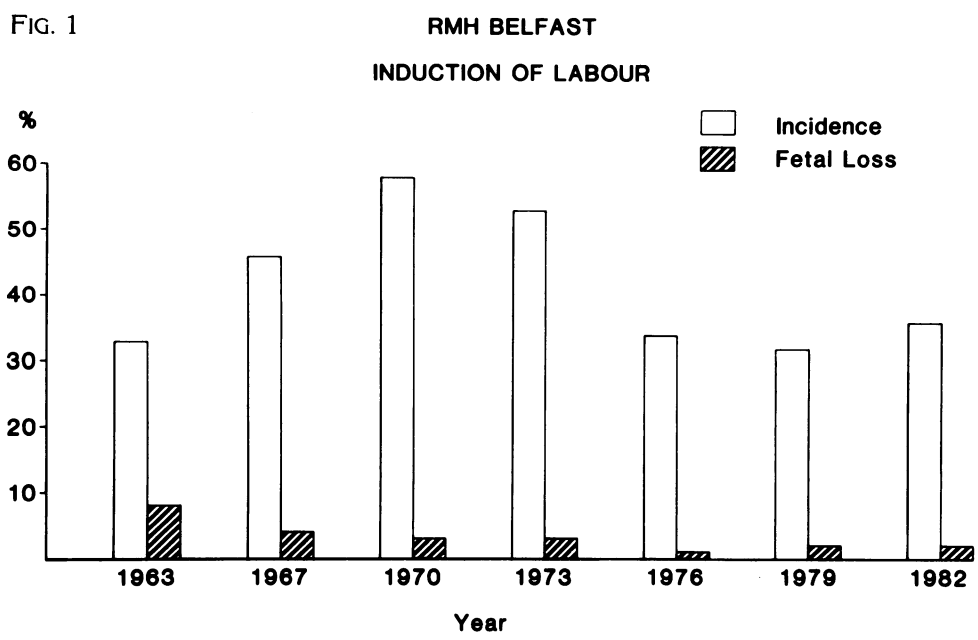
especially in the gray areas of obstetric practice. No one has any doubts today of the wisdom of premature delivery in placenta praevia with a mature fetus. And few would dispute the use of premature termination of pregnancy in severe pre-eclampsia or diabetes. With prolonged pregnancy perhaps there is less consensus. Even more controversial is induction of labour for mere convenience whether of the patient or her accoucheur. Of recent years, high rates of induction, especially for such reasons of convenience, have raised howls of protest from consumer associations aided and abetted by the media in search of a good controversy.

I must confess that during my active obstetric life I always leaned towards action rather than inaction, having grown up at a time when it seemed to me that too often the obstetrician was over-anxious to be seen to be doing the right thing. If something had to go wrong, let it be Nature's doing, not mine, was his prayer! But of course there are sins in obstetrics as in life generally of omission as well as of commission. Unfortunately the latter are more likely to attract social and legal censure than the former. It is what we do wrongly, rather than what wrongly we leave undone, that we are blamed for.

In 1963 the induction rate at the Royal Maternity Hospital (Fig. 1) was just over 30% associated with a total fetal wastage of nearly 7½% including IUDs and abortions at all stages of pregnancy. By 1970 the induction rate had reached an all-time high of nearly 60% whilst the fetal wastage had fallen to under 3½%. In other words, although the induction rate had nearly doubled, the total fetal loss had more than halved. Figures like this persuaded me at the time that our high induction rate was not only tolerable but even desirable, at least as far as obstetric indications were concerned.

There had meanwhile appeared in some areas of British obstetric practice a tendency to induce labour as a matter of convenience for patient or obstetrician

FIG. 1



or both. So far as I know, no definite harm had been shown often to have come of this, although the possibility of mistaken fetal maturity always existed and gave reason for pause to more cautious minds. Here in Northern Ireland the 'Troubles' beginning in 1968 and the civil strife occurring thereafter in Belfast, especially in the areas from which the RMH derived many of its patients, resulted in an environment unique in European obstetric practice in which unparalleled risks had to be faced if patients needed to be admitted urgently. We had lost one unfortunate mother with antepartum haemorrhage who insisted on going home and subsequently bled; whereupon local rioting prevented the ambulance from reaching her in time. We felt justified therefore in attempting to assess a technique which we called selective planned induction of labour, i.e. induction in normal patients after 38 weeks by which worried mothers were not hospitalised and separated from their families any longer than was absolutely necessary. Experience showed that fetal disaster due to unexpected prematurity was rare, but could be avoided with certainty only by the meticulous application of selective criteria, which unfortunately very significantly reduced the number of acceptable patients. However, provided these criteria were adhered to, no significant fetal or maternal harm ensued.

Probably our most important finding was the small proportion — only 16% — of patients who were suitable for selective planned induction if the necessarily strict criteria were adhered to. The reaction of the patients concerned is also interesting. It seems that each group liked what it got. Was it Lewis Carroll who said somewhere 'if you like what you get, you get what you like'? Certainly when asked before leaving hospital after their recovery 60% of the SPI (selective planned induction) group said they preferred induction, whilst 66% of the controls declared that they preferred spontaneous labour!

Since the time of this study some seven years have passed; local conditions have improved and obstetric fashions have changed, including the popularity of high induction rates. By 1976 in the RMH the incidence of premature induction of labour has fallen to under 40% with an associated total fetal loss of 1.2%. Between 1963 and 1976 the total fetal loss for the Hospital as a whole has also fallen, from 10.5% to 2.3%. The causes for this are complex and by no means entirely medical. But whatever they are the effect on fetal survival is gratifying.

Fetal happiness then, and fetal liberty, but what of fetal life itself? In the earlier decades of this century stillbirth and perinatal death rates had remained disappointingly high despite the advances that had been made in the prevention of fetal morbidity and mortality during the previous hundred years or so. The Belfast Lying-In Hospital, which was to become in due course the RMH, opened its doors to the women of Belfast in 1794, one of the 16 maternity hospitals founded in the British Isles during the previous 50 years or so. Obstetrics and especially the care of the newborn was at that time very much the province of the midwife. As a medical art, midwifery was regarded as barely respectable and certainly no occupation for a physician and a gentleman.

Even after obstetrics had become established in medical practice, it largely involved the management of abnormal cases such as APH or prolonged labour due to pelvic contractions. How little attention was paid to the wellbeing of the fetus at this time is indicated by the fact that in the Sydenham edition of William Smellie's excellent *Treatise on midwifery,* published in 1751, only some 28 pages out of 1171 are devoted to the problems of the newborn infant. This need

hardly astonish us as at the time most surviving infants were born without medical assistance. Where the latter was required the fetus was often dead or dying and the mother was also in dire straits. In such circumstances even the most humane and expert obstetrician was helpless on behalf of the fetus and only too often on behalf of the mother also. Malnutrition frequently resulted in pelvic distortion and consequently cephalo-pelvic disproportion. The only method available for the treatment of this in the early years of the 19th century was accouchement forcé, the forcible extraction of the child usually after destructive operations had reduced his bulk and rendered his removal possible. Caesarean section, so readily available today, was frequently fatal before the 20th century. The French obstetrician Pagot, writing in 1875, declared that 'the operation had cost the lives of all the unhappy ignorant women who had undergone it in Paris since the beginning of the century!'

Early attitudes to the unborn child and even the newborn infant are based, at least in western cultures, on Aristotle's views on embryological development. He taught that the newly conceived individual passed through vegetative and animal stages of development, only later becoming fully human. Such ideas are reflected in Hebrew and Islamic theology and later indirectly influenced mediaeval Christian thought on fetal rights, since obviously a vegetable or an animal does not command the same respect as a human being. The early Christian Church had evolved in a pagan society where slavery and brutality of all kinds were the norm and where the unborn child and the young infant were regarded as mere chattels of their fathers who had absolute rights over them. With no rights of their own, they were not infrequently done away with by abortion or infanticide on the flimsiest grounds.

The early Christians with their strong sense of the sanctity of human life condemned unconditionally all such acts of aggression against the human fetus at all stages of its development. Later on, unfortunately, the Church became bogged down in Aristotelian arguments about the time of ensoulment of the fetus, and the more important aspects of its humanity were lost sight of for many centuries. Thus by 1642 Sir Thomas Browne could still write 'we live, move, have our being and are subject to the actions of the elements and the malice of disease in . . . the womb of our mother . . . . In which obscure world the embryo awaits the opportunity of objects and seems to live . . . but in its roots and soul of vegetation'. He thus appears to regard the fetus in utero as something less than fully human 'still in its root and soul of vegetation' for, he continues, 'only after birth do we arise up and become another creature performing the reasonable actions of man'.

Nearly two centuries later, as we have noted, the wellbeing of the fetus was still very much a secondary concern in early 19th century practice. Nevertheless because the deliberate destruction of the living fetus was abhorrent both to the conscience of the obstetrician and to the law of the land it was of great importance to the accoucheur to be able to know for certain whether the fetus in utero was alive or dead. In the 1820s Laennec's new invention the stethoscope in the hands of physicians like the Breton, Kergaradec, and the Ulsterman, Ferguson, constituted a major advance towards the solution of this important forensic and ethical problem. Armed and skilful with the stethoscope the obstetrician, said Ferguson, 'who valued not only his good name but his conscience — was now able to pronounce with certainty whether the fetus be living or dead'. If the latter, then the child could in good conscience be fragmented and extracted. But if the

former, if the fetus still lived, the teaching accepted by Church, State and Society was that the operation must be delayed until after his death in order to avoid the 'great misprision' of fetocide which, if not murder as some would have it, was certainly manslaughter in the eyes of the law. At this stage then, the emphasis was on early diagnosis of fetal death to the advantage of the mother; for 'the obstetrician', as Collins of the Rotunda noted in the 1830s, 'sure that the child was dead, could with an easy conscience deliver her before she was exposed to the most torturing pain and not infrequently death, or — worse than death — extensive sloughing of the urethra'.

Later the emphasis was to shift from fetal death to fetal life and, in the 1840s, Sir James Simpson, advocating the use of forceps and intrauterine version instead of destructive operations in patients with disproportion, emphasised the value of fetal auscultation 'in determining the presence of life in the infant; which could be a contra-indication to delay and destructive operations and an indication . . . for early intervention on behalf of the living fetus'.

This shift in emphasis from early interference on behalf of the mother, the fetus having been shown by auscultation to be dead, to early interference on behalf of the fetus, the latter having similarly been shown to be living, was of the utmost significance in subsequent obstetric practice resulting as it did in considerable amelioration in fetal prognosis during the latter decades of the last century. Thereafter progress slowed and the neonatal death rate for England and Wales showed little change in the first 30 years of this century, remaining stubbornly over 35 per thousand. The main causes were trauma, infection and prematurity, each of which was responsible for about a third of neonatal deaths.

Even as recently as the 1930s and '40s, caesarean section was still regarded as much more hazardous for the mother and her infant and more difficult for the obstetrician than a high- or mid-cavity forceps delivery. When I first became Tutor in the RMH in 1944 I was allowed to attempt almost any kind of vaginal delivery on my own, but caesarean section had at least to be supervised, and was usually performed, by a consultant obstetrician. Gradually after the last War, however, with improved blood transfusion, antibiotic therapy, anaesthesia and surgical technique, caesarean section became progressively easier and safer than ever before and its more frequent use resulted in a very significant improvement in fetal morbidity and mortality. A less desirable result perhaps has been a tendency to abuse this operation, especially of recent years for fetal indications. But it should always be remembered that although the fetal results may be better where the operation is used in the management of conditions such as prematurity, multiple pregnancy and malpresentations there is undoubtedly a higher maternal risk. Nevertheless it must be conceded that the maternal and fetal results are both very much better in certain conditions when caesarean section is freely used. The most obvious example of this is of course disproportion. But, with the gradual disappearance of pelvic abnormalities due to nutritional causes, disproportion has become of much less importance than hitherto. Other conditions have, as a result, become relatively more important.

Placenta praevia is a condition in the management of which this hospital has played a leading part. When I began my obstetric career here in 1944, C.H.G. Macafee was a clinical lecturer in Professor Lowry's department. He was preparing for publication his classic paper on the conservative treatment of placenta praevia on which he had been working for seven years. His objective had been to lower

the fetal without adversely influencing the maternal mortality rate. The poor fetal results that characterised the treatment of bleeding from placenta praevia in the past were attributable to two main causes; first, prematurity — inevitable as long as the received doctrine was that there should be no expectant treatment of low-lying placenta; second, traumatic hypoxic vaginal delivery — also inevitable with manipulative procedures such as internal version and breech extraction or the application of Willetts forceps to the fetal head through an incompletely dilated cervix. The experience in the RMH (Fig. 2) for 1932-36 illustrates the risks involved: during that period nearly 3% of the mothers and over 50% of the babies died. As we see, this had altered little since Simpson's time a hundred years earlier. The next series from the Hospital shows the immediate improvement by Macafee's new attitudes to when and how these patients should be delivered. Between 1937 and 1944 the maternal mortality had been reduced nearly fivefold to 0.6% and the fetal mortality more than halved from 51.3% to 23.5%. A subsequent series reported by Grant in 1955 comprised 200 patients with no maternal deaths and a fetal mortality of 12%. It is noteworthy that the caesarean section rate in this series had increased from 39% to 76%. Subsequently reviews of these results of treatment in the RMH of placenta praevia show continuing improvement, the fetal mortality rate falling gradually to 3.5% in 1978-82. There have been no maternal deaths. Amongst the most important factors operating here are a high caesarean section rate and the help afforded by the paediatric service in managing the often premature infants of these mothers.

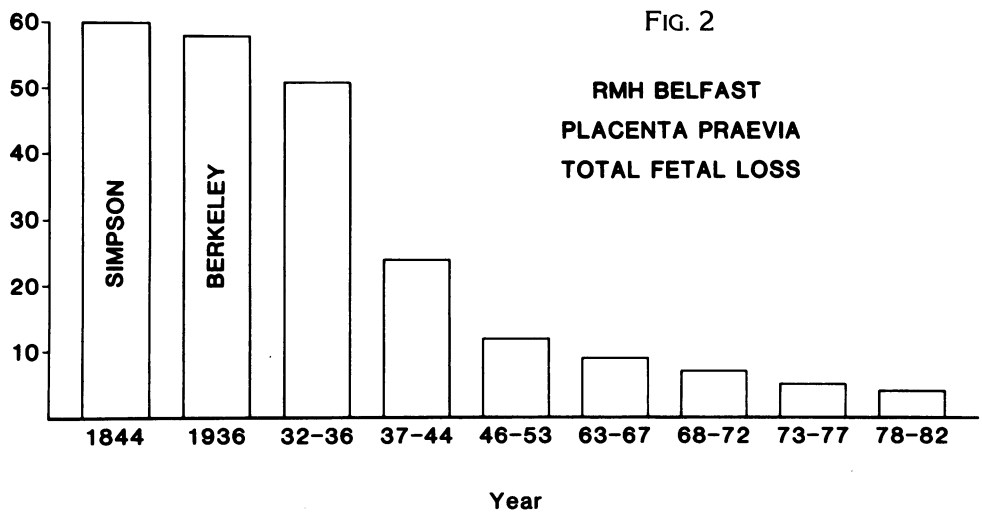


FIG. 2

Ladies and gentlemen, I believe that Macafee's work was a model of clinical research ranking high in the medical advances of this prolific century. It has been universally applied and has saved the lives of thousands of mothers and babies. I do not think he received the public recognition he deserved during his lifetime but I am glad that his colleagues in the Royal College of Obstetricians and Gynaecologists and in the Royal Society of Medicine awarded him their gold medals for his distinguished work.

It is interesting to recall that it was about this time that obstetricians were becoming increasingly dissatisfied with both their own and the paediatricians' efforts on behalf of the sick newborn. The obstetrician was often too preoccupied with the mother, who in such cases had usually been delivered surgically by forceps or caesarean section, to give the infant the immediate attention he obviously required; and the paediatrician was often not there at all! In any case neither of them was sufficiently skilled in the art and science of neonatal care, which was then in its infancy. Anaesthesia fortunately was increasingly becoming a highly skilled technology and the anaesthetist was always present — day and night — for operative deliveries, so what more natural than that the newborn be placed in his competent hands? As a result, neonatal morbidity and mortality diminished considerably but then remained static until the recent evolution of neonatology as a highly specialised, immediately available facility once more has improved the outlook for sick neonates.

Diabetes is another complication of pregnancy which, when I started obstetrics forty years ago, carried a very high fetal mortality. Here too, spectacular improvements have occurred over the years. Although these were less evidently determined by obstetric advances than was the case in placenta praevia, nevertheless improved obstetric techniques, especially a high rate of caesarean section, has played a part.

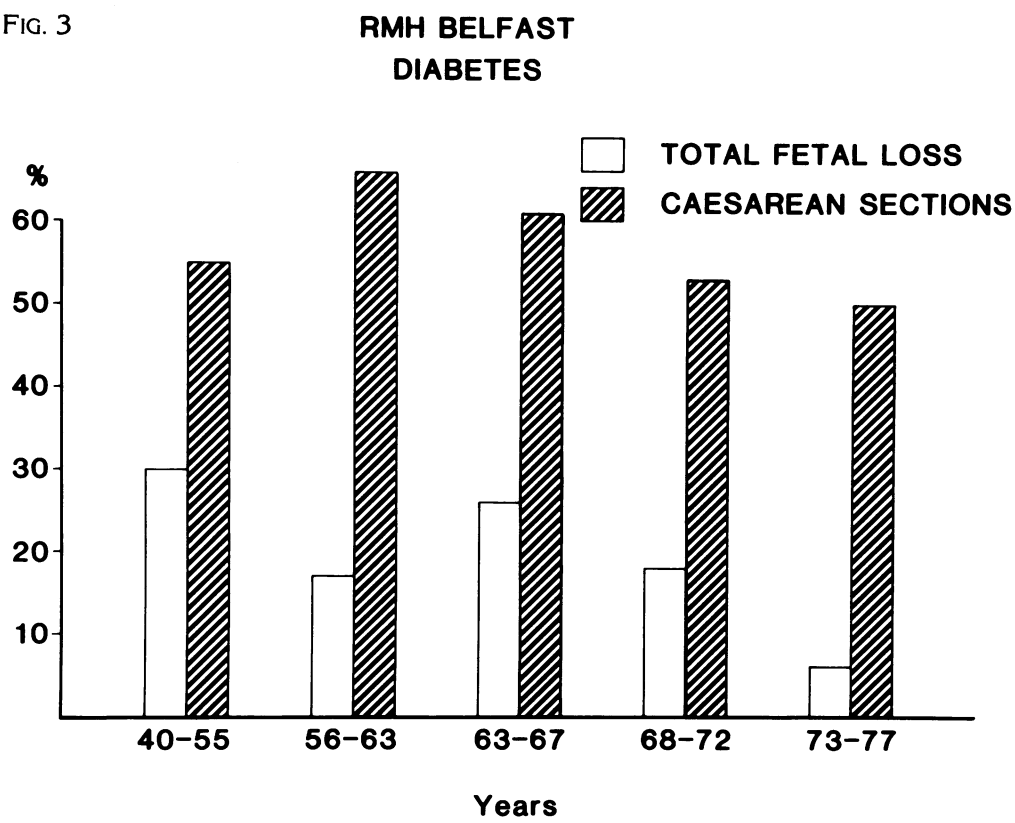
The Royal Maternity Hospital has for many years attracted most of the diabetic mothers of the province, and in 1956 Stevenson published the results in 119 diabetic pregnancies occurring during the 16 years from 1940-55. The fetal wastage overall was 30% and the caesarean section rate 55%. The latter figure, very high for that period, was the result of Peel's contention that the high fetal mortality rate, whatever its cause — whether poor diabetic control, pre-eclampsia or dystocia due to large babies — would be greatly reduced by terminating the pregnancies, often by a more extensive use of caesarean section at or before 36 weeks — it was generally recognised that many unexplained fetal deaths occurred during the last month of pregnancy.

In 1965 Professor Graham Harley reported on 115 babies born in the RMH from diabetic mothers during the eight years 1956-63. The results were greatly improved, with a fetal wastage nearly halved at 16.5%. The caesarean section rate was now 66% (85% in primigravidae). He emphasised the importance of good antenatal care and the avoidance of rigid schedules of time and method of delivery. Understandably he was not happy with 85% caesarean section in primigravidae! He also very properly pointed out the importance of good paediatric care of these premature and unpredictable newborns. Subsequently in 1964, after I had taken over from Professor Macafee, a special diabetic clinic with joint obstetrician-diabetician control by Professor Harley and Professor Montgomery was begun in the RMH with emphasis on the careful control of the diabetic condition of the mothers. Despite this, in 1963-67 the fetal wastage was 26.6% and in 1968-72 it was still 18.3%. I must emphasise that these statistics are derived from total raw figures including abortions, IUDs and fetal abnormalities. As with the placenta praevia data, no attempt at correction of any kind has been made.

By 1973 an increasing awareness was developing of the importance to the fetus of a maternal environment tending to the hypo- rather than the hyperglycaemia. As a result, maternal blood sugars began to be rigorously controlled at much lower levels than had previously obtained. The effect of this regimen was

apparent in the five-year figures for 1973-77 (Fig. 3) when the fetal wastage had fallen to 5.7% — a result that would have been regarded as unachievable 5 years before. At the same time the caesarean section rate fell to just over 50% from the previous high figure of 66%. This was the result partly of more efficient methods of induction and partly of a less slavish adherence to the idea of termination of pregnancies before 36 weeks.

FIG. 3



So far I have dealt, however sketchily, with the remarkable improvements in perinatal mortality over the past 40 years during which the perinatal mortality rate has fallen from 48 in 1943 to 14 in 1983. I believe that very soon mortality figures in the developed world will be as meaningless for the fetus as for the mother and reports will refer to individuals and small groups and concentrate much more on morbidity. I need hardly emphasise that this is very far from the truth as far as the underdeveloped or third world countries are concerned. And I believe that, in all our thinking about the future of maternity and child welfare services in the more fortunate parts of the world, we must increasingly bear in mind our less fortunate neighbours whose plight grows worse as ours grows better. Funds everywhere for everything except armaments seem to be in short supply; it is all the more vital that we learn to share such as are available. We can only ignore our responsibilities to the rest of the world at our peril.

But what of the future in our own countries? I believe that the general public has now accepted that babies are best born in properly equipped and staffed institutions. However, if we are not to give encouragement to the lunatic fringe who want babies to be born in domestic bathrooms or squatting in the public parks, we will have to make our hospitals pleasanter places to be in and to visit. Little expenditure will be needed for this once the idea is accepted. From the medical point of view the main problems that will confront the perinatologist in the next decade are low birth weight and abnormal babies. Unlike the third world, where low birth weight is largely due (through no fault of their own) to malnutrition, in the developed countries it is significantly contributed to by the the self-inflicted ills of unhealthy life-styles and the use of toxic substances like tobacco and alcohol. These are matters perhaps more for the priest and the paedagogue than the physician, but until such times as people in general learn to look after their own health, perinatologists and obstetricians will continue to be faced with the problem of what to do with the seriously underweight premature infant. We must never forget that modern medical care is a luxury, only possible if our industries and our agriculture produce enough excess wealth to provide more than the bare necessities of life: when shortages occur they affect the politically and physically weak first. The fetus in utero is bottom of the league in both these respects and in any fight for survival is liable to do badly, witness the incidence of legalised abortion in well-off countries today, mostly for indications that are rarely even remotely in the interest of the fetus and regrettably reminiscent of the late Roman Empire where they at least had the excuse that no effective contraceptive measures were available.

In planning the future — and it must be planned by us or someone else will do it — we must be careful not to insist too much on expensive high technology to be applied to seriously handicapped or underdeveloped babies. I believe our first duty at present is to those infants, at home and abroad, who have at least a reasonable chance of healthy survival, with a minimum expenditure at birth on intensive care which many disinterested people today regard, along with other forms of 'salvage' medicine, as approaching the point of diminishing returns, and therefore of low priority in competition for resources, financial and otherwise. In modern democratic medicine, many things that may be desirable are not expedient. Our first duty, bearing in mind that health and welfare costs continue to rise and that resources are finite, is to determine priorities. There is something absurd, some would say obscene, about a society which with one hand kills off thousands of normal but inconvenient fetuses and with the other hand expends scarce resources trying to produce artificial conceptions or to sustain seriously abnormal fetuses with little chance of anything approaching a reasonably normal existence. Even when such priorities have been agreed at government and professional level, they still have to be acceptable to society at large.

Experience with relatively simple matters, such as attempts at fluoridisation of drinking water or the use of safe and effective contraception, shows how difficult this latter process can be. Without doubt, universal knowledge is the key, and this depends upon education. There is nothing new in such ideas. Nearly a hundred and fifty years ago Charles Dickens described how the Spirit of Christmas-to-come 'sheltered in his garments two children, wretched, abject, frightful, hideous, miserable . . . No change and degradation, no provision of humanity in any grade has monsters half so horrible and dread. "They are man's", said the Spirit looking down upon them. "And they cling to me appealing from their fathers. This boy is

Ignorance. This girl is Want. Beware them both but most of all beware this boy for in his brow I see that written which is Doom unless the writing be erased. Deny it. Slander those who tell it ye! . . . and bide the end".' Today we seldom see these tragic children in our own streets, but alas they are still plentiful in the third world only a few hours' flight away. To impart knowledge costs money, but, Ladies and Gentlemen, 'if you think Education is expensive you should try Ignorance'.

The Ulster Medical Journal, Volume 54, No. 1, pp. 41-45, April 1985.

Refsum disease — the effect of diet

J H D Millar
Honorary Consultant Neurologist,
Royal Victoria Hospital and Claremont Street, Hospital, Belfast.

Based on a communication given at the 12th World Congress of Neurology, Kyoto, Japan, September 1981.

Accepted 1st January 1985.

A family suffering from Refsum disease,[1] the first to be discovered in the British Isles, was identified in 1957. One brother and two sisters were affected, while the second brother remained unaffected.[2] The propositus James (JS) presented in 1953 at the age of 21 with an acute polyneuropathy and raised CSF protein (4.5 g/l). His married sister aged 23 died a few weeks after she was first examined in 1957 from an acute encephalomyelitis with raised CSF protein (6.0 g/l). The younger sister Kathleen (KG) aged 20 had no complaints and the CSF was normal in 1957. All showed the characteristic features of Refsum disease, retinitis pigmentosa with night blindness and constriction of the visual fields, skeletal abnormalities and ichthyosis. James and Kathleen pursued a relapsing remitting course and slowly deteriorated despite treatment with steroids during relapses. At the request of Professor Daniel Steinberg now of the University of California, San Diego, they were transferred to the National Institute of Health at Bethesda, Maryland, USA, in 1966 for further investigation and dietary treatment. Their neurological state on admission to the NIH was described by Steinberg et al.[3] and their initial treatment and progress by Kark et al.[4] This paper is concerned with their further progress since their return to Northern Ireland in May 1970. They were then receiving a very strict low phytol, low phytanic acid fluid diet.

CASE 1

KG: Kathleen remained well, and she married in 1973. She was able to look after her house and go shopping. Her visual acuity was N12 N12 and weight 12 st. 1 lb. In 1975 a cataract developed in the right eye which was extracted in June 1976. Postoperative glaucoma developed and the visual acuity was 6/60 6/60. In August 1976 she complained of nausea and anorexia, probably due to acetazolamide treatment for glaucoma. Her weight fell to 8 st. 1 lb. Visual acuity was limited to hand movements. She developed a urinary tract infection with coliform organisms which was successfully treated. Blood pressure was 140/100 mm Hg. Twenty-four hour urine protein was 0.1 g in 1560 ml. Serum urea was 19.9 mmol/l, creatinine 250 μmol/l and calcium 2.24 mmol/l. Intravenous pyelogram showed small kidneys with blunted calyces suggesting chronic pyelonephritis. X-ray chest and electrocardiograph were normal.

In 1977 she was able to do her housework and read headlines in newspapers. There were no urinary symptoms apart from occasional nocturia. Unfortunately, major epileptic fits developed, without an aura or focal onset, and carbamazepine

treatment 200 mg daily was started. By 1979 the dosage of carbamazepine was increased to 300 mg daily because of continued fits. A left cataract was extracted. Her skull x-ray was normal but her EEG showed a sharp wave focus in the right parietal region. Serum urea had risen to 34.5 mmol/l and creatinine to 580 µmol/l. Serum calcium was 2.41 mmol/l and alkaline phosphatase 108 units/l.

In January 1980 she had a further fit. Her weight remained 8 st. 1 lb. She was able to smell only very pungent odours. Visual acuity in the right eye was limited to hand movements, left eye 6/36 and she could read with a magnifying glass. The right cornea was hazy, the left clear. She used a hearing aid. Power in the arms was good with slight weakness of dorsiflexion of the fingers. There was slight wasting of the forearms and small muscles of the hands. Power in the legs was good except for movements at the ankles, and co-ordination was good. Tendon reflexes were present, apart from the ankle jerks, and the plantar reflexes were flexor. All forms of sensation were normal. With the help of leg calipers she had a high steppage gait, slightly wide-based and ataxic but surprisingly good considering her poor vision and marked weakness at the ankles. The dryness of her skin was improved. A CAT brain scan was normal. The haemoglobin was 10.4 g/dl and urine culture was sterile. Serum urea was 35.9 mmol/l, creatinine 557 µmol/l, calcium 2.18 mmol/l and alkaline phosphatase 794 units/l.

In view of her deteriorating condition and rising plasma phytanic acid level (Figure) it was decided to try the effect of plasmapheresis. Between January and July 1980 11 two-litre exchanges were carried out. Following this she felt slightly better and the plasma phytanic acid level fell dramatically. Nerve conduction in the right ulnar nerve improved from 25 to 31 m/sec, and in the right median nerve from 28 to 30 m/sec. There was no change in renal function, serum urea 34.5 mmol/l, creatinine 639 µmol/l and calcium 2.07 mmol/l. Carbamazepine was continued at 600 mg daily.

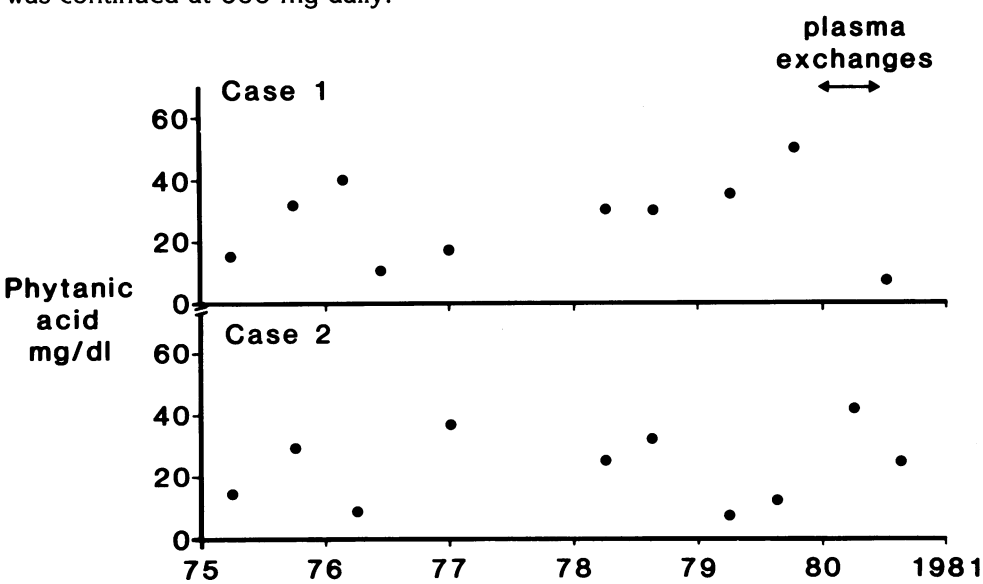


FIGURE. The figure shows the plasma phytanic acid values in Cases 1 and 2 from 1975. Prior to dietary treatment, the highest value in Case 1 had been 75 mg/dl, and in Case 2 had been 55 mg/dl. Note the fall in the phytanic acid value in Case 1 following plasmapheresis.

In spite of this initial improvement she steadily deteriorated. She was admitted to the South Tyrone Hospital on numerous occasions because of an increasing number of major fits and generalised myoclonus. In October 1980 blood pressure was 145/100 mm Hg, serum urea 36 mmol/l, creatinine 651 µmol/l, calcium 1.75 mmol/l and alkaline phosphatase 100 units/l. Haemoglobin was 11.5 g/dl. In January 1981 she was admitted with a pyrexial illness, vomiting, fits and myoclonus. Haemoglobin had fallen to 5.9 g/dl, but rose following transfusion to 13.6 g/dl. Blood pressure was 160/100 mm Hg. Visual acuity was limited to hand movements in the right eye and was 6/24 in the left eye. Myoclonus was unaffected by intravenous calcium or by cholecalciferol. A urinary tract infection with coliform organisms was treated with ampicillin. Carbamazepine 1000 mg daily and diazepam 15 mg daily were required for epilepsy and myoclonus.

She died on 14 February 1981. Autopsy was carried out by Dr R Lyness (A 36278). Both kidneys were markedly reduced in size. There was a large abscess in the upper pole of the right kidney measuring 6 cm in diameter containing green pus. The cortex and medulla were destroyed. In the lower pole there was a small cavity containing black brackish fluid. The cortex and medulla were reduced at the lower pole. The lower pole of the left kidney had a cavity containing black fluid and soft debris. The remainder of the cortex and medulla contained some small cysts. The pelves, ureters and bladder were normal.

Histological sections showed sclerosed glomeruli, and foci of chronic inflammation in the interstitium. The residual glomeruli and tubules were unremarkable on routine staining. More specific staining showed accumulated fat within the tubule lining cells and within the lumen of the tubules. There was a wide spectrum of fatty infiltration with some cells containing scant fine droplets and others disturbed by fat. One section showed a cyst filled with old blood. Other organs did not show this increase in fatty infiltration.

The brain showed no focal lesion to account for the epilepsy and myoclonus. Spinal cord sections showed loss of posterior column fibres with decreased staining of myelin. The posterior spino-cerebellar tracts showed a slight decrease in staining indicating some fibre loss. The anterior horn cells were normal and there was thickening of the anterior roots. Staining for fat showed increased thickness of the myelin sheaths, some of which took up the stain to give a purple hue. Peripheral nerve sections showed hypertrophy with hyperplasia and hypercellularity of the Schwann cell coating of the axons. There was an increase in the amount of endoneural tissue. Longitudinal sections showed a degree of myelination of fibres, and stains for fat showed increased amounts of lipid around the nerve fibres.

Cranial nerves:

I	Normal.
II	Section shows a loss of nerve fibres with increase of tissue in the endoneural space. The eyes have yet to be processed.
III-VII	Sections show slight patchy loss of myelinated fibres.
VIII	Sections show loss of nerve fibres and myelin, gliosis and corpora amylacia.
IX-XI	Sections show proliferation of the Schwann cells to give thickening of the nerve fibres.

The cause of the epilepsy was not clear. There was no lesion in the brain to account for the focal EEG abnormality, but no doubt the chronic uraemia and low serum calcium played a part.

The cause of death was septicaemia secondary to a coliform abscess in the right kidney. The chronic progressive renal failure was probably due to chronic pyelonephritis. What part the fatty deposits in the kidney (probably phytanic acid) had to play is difficult to assess, but, as similar changes had been found in the kidneys of another patient from a second Belfast family suffering from Refsum disease who died in uraemia,[5] further studies were performed. Thin layer chromatography was carried out by Dr D McCormick on lipid extracts of liver, kidney, heart, skeletal muscle and brain (frozen unfixed tissue). All these tissues except brain showed an extra fraction which migrated ahead of the main triglyceride fractions and which was absent from control samples, similar to the findings in the case mentioned above.

CASE 2

JS: James was very well when he returned from Bethesda in 1970. He could walk 5 miles but he was completely anosmic and deaf. His visual acuity was 6/60 in the right eye and he could only perceive light with the left eye.

In January 1974 he suffered a relapse following an attack of diarrhoea. Walking became worse and he was unable to walk outside without assistance although he could get around the house. He had difficulty tying laces and buttoning his shirt but could feed himself. There was slight weakness of extension at both elbow and wrist joints, weakness of all finger movements, particularly of the right thumb, and moderate weakness at both knee and ankle joints. Light touch and pain sensation was impaired in the hands and the lower third of both legs. Vibration sensation was absent in the legs but joint position sense was normal. All the tendon reflexes were absent apart from the biceps jerks. Cerebro-spinal fluid protein concentration was 3.40 g/l. Conduction velocity in the right ulnar nerve was 11.1 m/sec. and plasma phytanic acid was 13.9 mg/dl. Following a course of ACTH injections for several months, power and sensation improved considerably. CSF protein fell to 1.58 g/l and conduction velocity in the right ulnar nerve rose to 17.3 m/sec.

In December 1974 he felt well and had become married. A normal daughter was born in 1975. (Her plasma phytanic acid level was within normal limits in 1982). In 1980 he began to complain of a 'buzzing noise' in the head for which no cause could be found apart from deafness. He was able to walk 3 miles. Apart from weakness at the ankles, power and co-ordination of the limbs was very good. Sensation had improved except for persistent impairment of pain sensation in the lower third of both legs. All the tendon reflexes were present apart from the ankle jerks. Serum urea was 7.2 mmol/l and calcium 2.46 mmol/l. Urine contained a trace of protein but was sterile. Conduction velocity in the right ulnar nerve was now 30.8 m/sec.

Now, in 1984, James keeps well but complains still of the 'buzzing' noise in the head. His daughter is normal in all respects. His plasma phytanic acid levels have been satisfactory, varying from 12.1 to 33.0 mg/dl during the past two years. Serum urea is 13 mmol/l, creatinine 103 µmol/l and calcium 2.44 mmol/l. The unaffected brother is alive and well.

DISCUSSION

There is little doubt that the dietary treatment led to considerable improvement in mobility, power, co-ordination, sensation and peripheral nerve conduction in both cases. There was also marked improvement in the ichthyotic condition of the skin. It should be pointed out that Case 1 was in a wheelchair before the diet was started. However, no clear-cut improvement could be shown in the special senses of smell, vision and hearing in these two patients. Gibberd and Goldman[9] have also reported disappointing results for the special senses. Perhaps dietary treatment did not begin early enough. The striking improvement with ACTH treatment for the relapse in Case 2 in 1974 raises the possibility that disturbances of an immunological nature can occur in the relapsing remitting type of the disease.

Plasmapheresis has been successfully used in several cases of Refsum disease, reducing the level of plasma phytanic acid.[6, 7, 8] This treatment certainly caused a dramatic fall in phytanic acid level in Case 1 but could it have contributed to her death from renal abscess and septicaemia? Wing and his colleagues[10] state that one-third of patients undergoing plasmapheresis for renal disease will develop a serious infection. They also found that each plasma exchange removes approximately 30 per cent of serum immunoglobulin and 30 per cent of circulating complement components when plasma is replaced with fresh-frozen plasma and albumin in a 1:1 ratio.

ACKNOWLEDGEMENTS

It is a pleasure to thank Professor Daniel Steinberg, University of California, San Diego, for his continuing interest in this family and for carrying out the phytanic acid estimations. I also wish to thank Dr B T McNamee and Dr V H Patterson for permission to quote from their records.

REFERENCES

1. Refsum S. Heredopathia atactia polyneuritiformis. In: Peripheral neuropathy. Ed. Dyck PJ, Thomas PK, Lambert EH, Bunge R. 2nd ed. Philadelphia: Saunders, 1984; **2**: 1680-1703.
2. Ashenhurst EM, Millar JHD, Milliken TG. Refsum syndrome affecting a brother and two sisters. *Br Med J* 1958; **2**: 415-417.
3. Steinberg D, Vroom FQ, Engel WK, Cammermeyer J, Mize CE, Avigan J. Refsum disease — a recently characterised lipidosis involving the nervous system. *Ann Intern Med* 1967; **66**: 365-395.
4. Kark RAP, Engel WK, Blass JP, Steinberg D, Walsh GO. Heredopathia atactica polyneuritiformis (Refsum disease): a second trial of dietary therapy in two patients. In: Bergsma D, McKusick V, eds. The nervous system. National Foundation — Birth Defects, Original article series 1971; **7**: 53.
5. Allen IV, Swallow M, Nevin NC, McCormick D. Clinico-pathological study of Refsum's disease with particular reference to fatal complications. *J Neurol Neurosurg Psychiat* 1978; **41**: 323-332.
6. Penovich PE, Hollander J, Nusbacher JA, Griggs RC, MacPherson J. Note on plasma exchange therapy in Refsum disease. In: Kark RAP, Rosenberg RN, Schut LJ, eds. The inherited ataxias: biochemical, viral and pathological studies. New York: Raven Press, 1978: 151. (Advances in neurology; Vol 21).
7. Gibberd FB, Billimoria JD, Page NGR, Retsas S. Heredopathia atactica polyneuritiformis (Refsum disease) treated by diet and plasma exchange. *Lancet* 1979; **1**: 575-578.
8. Moser HW, Hayden B, Peyeritz RE, Ullman D, Murray C, Asbury A. Therapeutic trial of plasmapheresis in Refsum disease and in Fabry disease. *Birth Defects* 1980; **15**: 491.
9. Gibberd FB, Goldman JM. Clinical manifestations of heredopathia atactica polyneuritiformis. Communication given to the Association of British Neurologists, April 1984.
10. Wing EJ, Bruns FJ, Fraley DS, Segel DP, Adler S. Infectious complications with plasmapheresis in rapidly progressive glomerulonephritis. *JAMA* 1980; **244**: 2423-2426.

The Ulster Medical Journal, Volume 54, No. 1, pp. 46-52, April 1985.

High tibial osteotomy in degenerate diseases of the knee

G F McCoy, H K Graham, C J McClelland

Accepted 21st January 1985.

SUMMARY

Between 1970 and 1981, 64 patients underwent 77 tibial osteotomies for degenerate diseases of the knee at the Withers Orthopaedic Centre in Belfast. Records on 11 patients (12 knees) were either missing or inadequate, leaving 53 patients who underwent 65 tibial osteotomies for study. There were 23 males and 30 females, ranging in age from 23 to 75 years (mean 59.8 years). The predominant diagnosis was osteoarthrosis and the indication for operation in all cases was pain. With a follow-up of from two to ten years (mean 4.8 years), 39 knees were assessed as good, 15 as fair (improved, but still symptomatic), and 11 as failures. Patients with valgus deformity did worse than those with varus deformity. The importance of adequate pre-operative assessment is stressed, the operation itself is outlined, and the end result is seen to correlate closely with the degree of correction obtained.

INTRODUCTION

High tibial osteotomy is an operation of proven value for relieving pain and improving function in the degenerate knee. Despite the increasing success and acceptability of total knee replacement,[1, 2, 3] there is often justifiable reluctance to consider arthroplasty, especially in the younger patient with unicompartmental disease. As most of the patients considered suitable for this procedure have a good range of movements, arthrodesis is not an acceptable alternative. The predominant indications for high tibial osteotomy are pain, accompanied by valgus or varus deformity, and a reasonable range of movements.[4] The causes of the pain are not fully understood. Lloyd-Roberts in 1953[5] cited capsular fibrosis as the cause, but Phillips et al[6] felt the major cause was venous congestion. More recent work suggests that articular cartilage breakdown

Musgrave Park Hospital.

G F McCoy FRCS, Senior Orthopaedic Registrar.

H K Graham FRCS, Senior Orthopaedic Registrar.

C J McClelland FRCS, Consultant Orthopaedic Surgeon.

Correspondence to: Mr G F McCoy FRCS., Department of Orthopaedic Surgery, The Queen's University of Belfast, Musgrave Park Hospital, Belfast BT9 7JB.

products have a role. The pain is characteristically worse on standing or walking, as the deformity increases with weight-bearing. The fact that tibial osteotomy successfully relieves joint pain was reported by Jackson and Waugh in 1961,[4] and, subsequent reports[7, 8, 9, 10, 11, 12] have all indicated that the operation of tibial osteotomy is reliable in properly selected cases.

This paper outlines the experience of tibial osteotomy in Northern Ireland from 1970 to 1981. In Northern Ireland, prior to 1974, tibial osteotomy was the only alternative to arthrodesis in degenerate knee diseases. From 1974 onwards, this procedure was used more frequently for its more classical indications of the mobile varus or valgus knee with relative sparing of one compartment as an alternative to the Marmor unicompartmental replacement. All the osteotomies were performed above the insertion of the patellar tendon. Two types of osteotomy were employed, the wedge osteotomy (and a variation of this, the bracket osteotomy) described by Slocum et al,[13] and the dome osteotomy which we will describe. Patients were assessed pre- and post-operatively with regard to pain, range of knee movements, and mobility skills. Two to 10 years follow-up was available on all patients.

MATERIALS AND METHODS

From 1970 to 1981, 64 patients underwent 77 tibial osteotomies for degenerate diseases of the knee. Due to absence or inadequacy of records in 11 cases (12 knees), 53 patients (65 tibial osteotomies) were available for study. The diagnosis in 60 knees was osteoarthrosis, four of these being due to previous intra-articular fractures. There were five cases of rheumatoid arthritis. Fifty knees underwent bracket or wedge osteotomy, the osteotomy being held by staples (43 cases), plaster of Paris (9 cases), or Charnley clamps (3 cases). The technique of bracket and wedge osteotomy has previously been described.[13] Eleven knees (mostly occurring later in the series) had 'dome' osteotomy performed.

The patient is anaesthetised on a standard table, and a pneumatic tourniquet applied. The fibula is first obliquely osteotomised through a separate incision at the junction of its upper and middle thirds. The tibia is exposed through a curved or transverse incision at about the level of the tibial tubercule. The patellar tendon is isolated and raised clear of the anterior surface of the tibia, its attachment remaining undisturbed. The osteotomy is commenced using a series of one-eighth inch drill-holes parallel with the joint surface in the shape of a dome, the apex of which should ideally lie one centimetre below the joint line. A hand drill is used, preferably, because its superior 'feel' allows the posterior cortex to be perforated safely without undue risk to the vital structures at the back of the knee. The osteotomy is completed using an osteotome. Manipulation facilitates the displacement, which, in the case of a varus knee, should aim at 5° of over-correction (Fig. 1a and b). With a valgus knee, the correlation should ideally be to neutral. The 'step' seen in the post-operative x-rays (Fig. 2) is a good indication of the degree of correction obtained. Post-operatively, the correction is held in plaster of Paris until union is achieved. No internal staples are employed, so that, if the correction is not adequate, a further manipulation is possible. Union occurred in almost all cases in our series within 10 weeks.

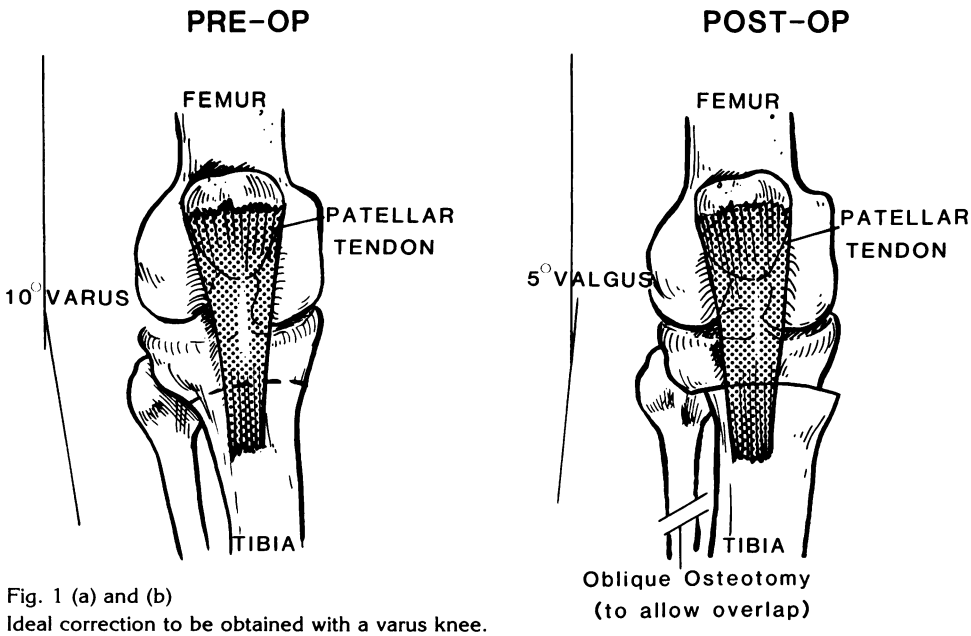


Fig. 1 (a) and (b)
Ideal correction to be obtained with a varus knee.

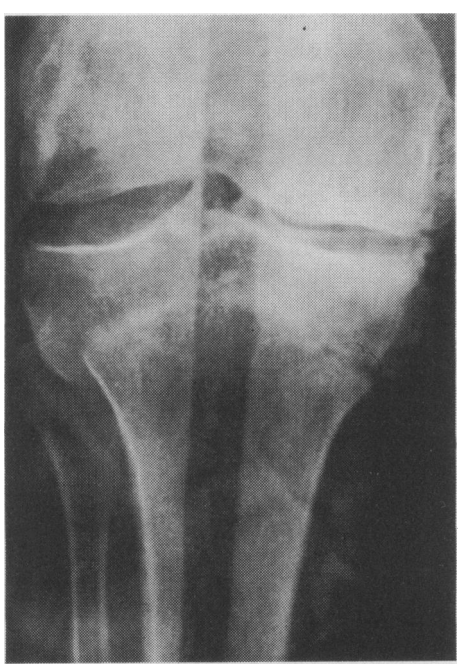


Fig. 2
Varus knee post-operative (in split plaster) showing the 'step' denoting good correction.

RESULTS

The 53 patients in our series (65 osteotomies) were followed up for between two and 10 years (mean 4.8 years). They were assessed both pre- and post-operatively with regard to pain, range of knee movements, and walking ability. The degree of pain relief achieved by this operation was very marked. Pre-operatively, 97% of patients complained of moderate or severe pain. Post-operatively, 82% of patients had only mild pain, or none at all (Fig. 3).

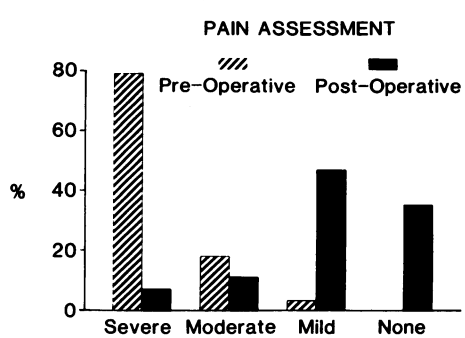


Fig. 3
Pain assessment pre- and post-operatively.

Similarly, there was an improvement in the maximum flexion achieved by the patient from 41% with flexion greater that 100° pre-operatively, to 61% with this degree of flexion post-operatively (Fig. 4). It is undoubtedly true that much of the improvement in the range of movement was due to the considerable pain relief achieved by the operation.

The improvements in symptoms with regard to pain and limitation of movement brought with them increased mobility as expressed by walking ability (Fig. 5). This improvement in mobility is not as dramatic as occurs with total knee replacement,[1, 14] but then, it is not expected to be so, and, in any case, a high percentage of these patients were fairly mobile pre-operatively.

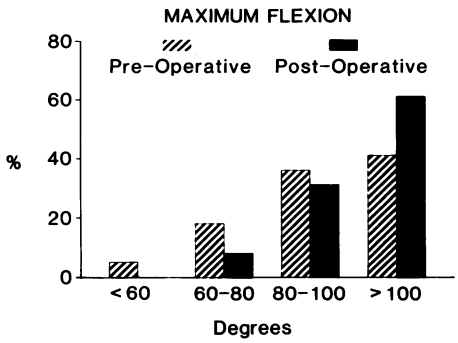


Fig. 4
Range of flexion pre- and post-operatively.

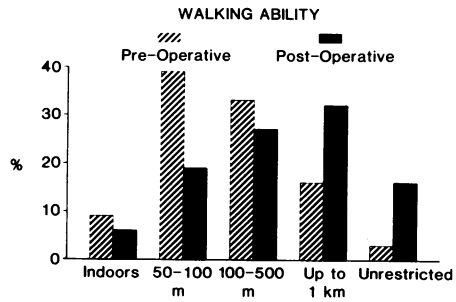


Fig. 5
Walking ability pre- and post-operatively.

Taking the overall results, 60% of patients had a good functional result and were prepared to consider a similar procedure on the contralateral side. Fifteen knees (23%) had only a fair result, in that, although improved, there remained significant symptomatology. Eleven knees (17%) must be considered as failures (Table). There were three early outright failures, two of these being arthrodesed and the other being converted to a knee replacement. A further three cases were considered for knee replacement at between one and two years from the original operation and have now all undergone successful arthroplasty. Three had persistence of pain and in two of these cases there was the added complication of recurrent valgus. Another knee assessed as poor achieved considerable improvement with regard to pain at the expense of greatly diminished movements. Finally, one osteotomy was complicated by persistent non-union, despite three attempts at grafting.

TABLE

Results of tibial osteotomies

	Good	*Fair*	*Poor/Failure*	*Total*
Valgus knees	6 (34%)	8 (44%)	4 (22%)	18
Varus knees	33 (68%)	7 (16%)	7 (16%)	47
Overall results	39 (60%)	15 (23%)	11 (17%)	65

Many knees were classed as fair on the basis of post-operative persistence of a moderate degree of pain. This was especially the case with valgus knees, where the deformity being corrected was often quite marked. Two knees finally rated as fair had delayed union, and one non-union (the latter requiring a bone graft). Three cases which were good results are now being assessed for total knee replacement at 6, 8 and 10 years respectively. Five patients remain symptom-free more than eight years after operation.

If the overall results are broken down according to whether the knee was initially valgus or varus, significant trends are noticed. With the valgus knees, only one-third achieved a good result. Forty-four per cent was only fair, and 22% were failures. This contrasts quite markedly with the results in varus knees. Here, 68% had a good result, with 16% in each of the fair and poor categories. The mean correction in the valgus knees was from 27° pre-operatively to 4.5° (valgus) post-operatively. Thus there was a tendency to correct only to physiological valgus and not to neutral. With varus knees the correction obtained was from 11.5° pre-operatively to 2.4° (valgus).

Complications were relatively few, apart from the three patients who required early conversion to either arthrodesis or knee replacement. The most serious complication was that of a patient with persistent non-union (despite three bone grafts). This patient has now achieved a fibrous union and is mobile on a stick. She has declined any further surgery. There were two cases of delayed union and one of non-union (the latter requiring a bone graft). In four cases wound problems delayed discharge from hospital, but the wounds eventually healed. There were no cases of common peroneal nerve injury or compartment syndrome.

DISCUSSION

Many authors report benefits achieved by tibial osteotomy in properly selected cases.[4, 7, 8, 9, 10, 15, 16, 17, 18] In our series, we were impressed by the overall improvement achieved, particularly with regard to pain relief. The improvement in range of movement and overall mobility was also quite marked. The operation of tibial osteotomy is, however, often compared in the patients' mind with that of knee arthroplasty. Any residual incapacity may then only be accepted with reluctance. It is important to explain the nature and expected outcome of the operation to the patients so that they do not entertain unrealistic expectations.

The precise estimation of the pre-operative deformity is mandatory,[11, 12, 19] since good results are closely correlated with accurate correction. Pre-operative arthroscopic assessment is probably unnecessary.[20] Coventry[21] considers bone-scanning to be most valuable in assessing the likely outcome and has shown that if both compartments are affected as demonstrated by increased uptake, then the result is likely to be disappointing. Several authors stress the importance of standing x-rays in evaluating the true deformity,[19, 22, 23] and a case from our own series re-emphasises this, where the supine deformity of 3° of varus increased to 7° on standing. With regard to pre-operative assessment, those patients whose deformity was marked generally did badly. Insall[19] maintains that with varus deformity exceeding 10° or valgus deformity greater than 15° tibial osteotomy is contra-indicated. It is certainly true that patients with marked valgus deformity generally do badly. In our series, all those with valgus deformity exceeded 15° on standing x-rays, and this probably contributed to the high incidence of indifferent and poor results in these cases. Agletti[23] and Coventry[21] both stress the

importance of over-correcting a varus to between 5° and 10° of valgus. In our series, this over-correction was apparent (mean correction obtained from 11.5° (varus) pre-operatively to 2.4° (valgus) post-operatively), and, this may, in part, explain the high percentage of good results (68%). Four of the seven bad results were not corrected to beyond neutral and consequently suffered recurrent varus. Our best result from an initially varus knee (symptom-free at 10 years) was over-corrected at operation to 10° of valgus.

The much poorer results with initially valgus knees merits close scrutiny. Shoji and Insall[24] maintain that with valgus knees correction to 5° valgus is indicated and that such deformity should not be corrected to neutral. Coventry[21] feels that some degree of over-correction may be necessary to prevent recurrent valgus. In our series, the mean correction with valgus knees was from 27° to 4.5°. There was therefore a tendency to correct only to physiological valgus and not to neutral, and a tendency to recurrent valgus was evident. Attempts to correct too great a deformity are likely to give a bad end result. Thus, those with valgus deformities of greater than 20°, or varus deformities exceeding 15° generally had indifferent or poor outcomes. Where the initial deformity lay between 15° of varus and 20° of valgus (on standing x-rays) the outcome was generally good. Marked pre-operative restriction of movements was noted to correlate with worse results, an observation noted by other workers.[4, 21, 23]

CONCLUSIONS

Despite the increasing success of total knee replacement in the treatment of degenerate diseases of the knee, there remain definite indications for tibial osteotomy. These are:

(a) Relatively young patients, with
(b) Painful, mobile knees, and
(c) Valgus or varus deformity in the range 20° valgus to 15° varus (weight-bearing) with relative sparing of one compartment.

Applying these indications, tibial osteotomy can offer long-term relief to many. It will often defer by years the time when arthroplasty becomes necessary, and in a good percentage of cases no further surgical treatment will be indicated.

We wish to thank our colleagues in the Withers Orthopaedic Centre for their help and co-operation in this survey, Mr Brendan Ellis and Mr Ronnie Wood for their illustrations and photography and Miss Lily McGuffin for the typing of the manuscript.

REFERENCES

1. McCoy GF, McLeod NW, Nixon JR, Lowry JH, Mollan RAB. The Richards (RMC) knee replacement. *International Med* 1984; **Suppl. 9**: 20-24.
2. Shaw NE, Chatterjee RK. Manchester knee arthroplasty. *J Bone Joint Surg* 1978; **60B**: 310-314.
3. Freeman MAR, Todd RC, Bambert P, Day WH. ICLH arthroplasty of the knee: 1968-1977. *J Bone Joint Surg* 1978; **60B**: 339-344.
4. Jackson JP, Waugh W. Tibial osteotomy for osteoarthritis of the knee. *J Bone Joint Surg* 1961; **43B**: 746-751.
5. Lloyd-Roberts GC. The role of capsular changes in osteoarthritis of the hip joint. *J Bone Joint Surg* 1953; **35B**: 627.

6. Phillips RS, Bulmer JH, Hoyle G, Davies W. Venous drainage in osteoarthritis of the hip. A study after osteotomy. *J Bone Joint Surg* 1967; **49B**: 301.
7. Wardle EN. Osteotomy of the tibia and fibula. *Surg Gynecol Obstet* 1962; **115**: 61.
8. Venemans J. Tibial osteotomy for osteoarthritis of the knee. *J Bone Joint Surg* 1962; **44B**: 956.
9. Jackson JP, Waugh W. Tibial osteotomy for osteoarthritis of the knee. *J Bone Joint Surg* 1963; **45B**: 618.
10. Torgerson WR. Tibial osteotomy in the treatment of osteoarthritis of the knee. *Surg Clin N Amer* 1965; **45**: 779.
11. Coventry MB. Osteotomy of the upper portion of the tibia for degenerative arthritis of the knee: a preliminary report. *J Bone Joint Surg* 1965; **47A**: 984.
12. Coventry MB, Bowman PW. Long-term results of upper tibial osteotomy for degenerative arthritis of the knee. *Acta Orthop Belg* 1982; **48**: 139.
13. Slocum DB, Larson RL, James SL, Grenier R. High tibial osteotomy. *Clin Orthop* 1974; **104**: 239-243.
14. McCoy GF, McLeod NW, Nixon JR. Experience with the Sheehan Knee Replacement. *Ulster Med J* 1983; **51**: 35-40.
15. Maquet PGJ. Osteotomy. In: Freeman MAR, ed. Arthritis of the knee: clinical features and surgical management. Berlin: Springer, 1980: 148.
16. Maquet P, Watillon M, Burny F et al. Traitement chirurgical conservateur de l'arthrose du genou. *Acta Orthop Belg* 1982; **48**: 204.
17. Vainionpaa S, Laike E, Kirves P, Tuisanen P. Tibial osteotomy for osteoarthritis of the knee: a five to ten year follow-up study. *J Bone Joint Surg* 1981; **63A**: 938.
18. Kettlekamp DB. Tibial osteotomy for unicompartmental osteoarthritis. In: Leach RE, Hoagland FT and Riseborough EJ, eds. Controversies in orthopaedic surgery. Philadelphia: Saunders, 1982: 5.
19. Insall J, Shoji H, Mayer V. High tibial osteotomy. *J Bone Joint Surg* 1974; **56A**: 1397-1405.
20. Keene JS, Dyreby JR. High tibial osteotomy in the treatment of osteoarthritis of the knee: the role of pre-operative arthroscopy. *J Bone Joint Surg* 1983; **65A**: 36-42.
21. Coventry MB. Upper tibial osteotomy. *Clin Orthop* 1984; **182**: 46-52.
22. Jackson JP, Waugh W. High tibial osteotomy for arthritis of the knee. *J Bone Joint Surg* 1969; **51B**: 88-94.
23. Aglietti P, Rinonapoli E, Stringa G, Taviani A. Tibial osteotomy for varus osteoarthritis knee. *Clin Orthop* 1983; **176**: 239-251.
24. Shoji H, Insall J. High tibial osteotomy for osteoarthritis of the knee with valgus deformity. *J Bone Joint Surg* 1973; **55A**: 963-973.

The Ulster Medical Journal, Volume 54, No. 1, pp. 53-57, April 1985.

Increasing demands on today's blood donors

W M McClelland

Accepted 1st January 1985.

SUMMARY

Recently in Northern Ireland there has been a rapid increase in demand for a variety of blood components. To meet this need a large proportion of routine blood donations must be processed at the Transfusion Centre. In addition, several blood components are collected direct from donors by apheresis techniques. Apheresis is currently restricted to the collection of components from highly selected donors, but in future this method is likely to be employed for collection of some routine components. This changing pattern is placing increasing demands on many of our blood donors.

INTRODUCTION

Blood transfusion plays an essential role in modern medicine and surgery. Loss of blood, whether occurring spontaneously or as a result of trauma, can be rectified by the transfusion of whole blood or of concentrated red cells, a practice so well established as to be taken almost for granted. More recently, transfusion of particular components of blood has been introduced as part of the management of a wide variety of diseases. Indeed, whereas the usage of red cells has been increasing slowly, the demand for many of these other blood components has shown a dramatic upsurge in recent years. This upward trend applies particularly to platelet concentrates, fresh frozen plasma, and various plasma products, especially Factor VIII, albumin and certain immunoglobulins.

This development has placed additional burdens on the Blood Transfusion Service which is responsible for the production of components from donated blood. The huge increase in demand for blood components means that a high proportion of blood collected is now processed at the Transfusion Centre laboratories. The Table summarises the issues of blood and the major blood components to hospitals in the Province from 1974-84. It can be seen that, although blood donations have not been increasing during the past 6-7 years, the usage of most blood components has increased dramatically during the same period. The exception is cryoprecipitate, the usage of which has decreased as this product is gradually replaced by freeze-dried concentrates of Factor VIII for the treatment of haemophilia A. As in the rest of the UK, a proportion of the Factor VIII concentrate used in the Province has had to be imported, and the sudden increase in supply during the past year reflects a determined drive to become self-sufficient in this product.

Northern Ireland Blood Transfusion Service, 98 Durham Street, Belfast BT12 4GE.
W M McClelland MB, MRCPath, Director.

TABLE

Issues of major blood components from NIBTS — 1974-1984

Year	*Blood donations* (Units)	*Platelet concentrates* (Units)	*Fresh frozen plasma (220 ml/unit)* (Units)	*Factor VIII concentrates* (International Units)	*Cryoprecipitate (80 IU Factor VIII/pack)* (Packs)	*Albumin solutions (20 gm albumin/bottle)* (Bottles)
1974	54,251	882	NIL	NIL	6,982	342
1976	61,882	1,916	NIL	NIL	10,629	1,533
1978	64,219	3,166	NIL	120,000	10,560	2,027
1980	66,401	5,614	1,574	140,000	5,080	3,265
1981	64,135	6,689	2,396	240,000	3,473	3,331
1982	63,310	8,288	4,943	35,000	3,253	4,561
1983	62,428	9,042	4,883	337,000	2,516	9,551
1984 (projected)	66,500	12,500	6,500	1,300,000	3,000	10,300

The projected 1984 figures are based on those available for the first nine months of the year.

Albumin solutions include plasma protein solution, salt-poor albumin and prior to 1982, dried plasma.

To meet the demand for Factor VIII concentrate (and other fractionated products), huge quantities of fresh frozen plasma must be collected, currently equivalent to approximately 35,000 donations per annum. This in turn depends on acceptance by clinicians of plasma-reduced blood for the majority of patients who require red cell transfusions. The production of purified plasma products requires special facilities and since 1982 fresh frozen plasma from Northern Ireland has been sent to the Protein Fractionation Centre, Edinburgh, for processing and purification.

While most blood products can be harvested from ordinary, single-blood donations, for some components there are advantages in direct collection from the donor. This applies particularly to those components which are found only rarely among the blood donor population, e.g. certain specific antibodies, or to those which are difficult to obtain in sufficiently high yield from conventional donations, e.g. granulocytes.

The main purpose of this article is to describe the organisation and precautions involved in setting up specialised blood donor panels for the collection of blood components and to highlight the increasing demands being placed on these donors.

APHERESIS

Collection of blood components direct from the donor is carried out by a procedure called apheresis which allows harvesting of the desired component while the remainder of the blood is transfused back to the donor. Apheresis procedures have a number of advantages over ordinary donations for the collection of components. Thus, not only are larger yields per donation obtained, but, because there is no significant red cell loss, donation can be carried out more frequently (commonly at monthly intervals). Apheresis can be carried out either manually, using a centrifuge to isolate the required component, or mechanically, using specially designed cell-separator machines. A large number of the latter devices which can be used to collect plasma, platelets or white cells direct from donors are now available. Although these procedures have become well established and are considered safe, they do tend to be fairly prolonged and entail having a needle in situ (one or both arms) for periods varying from 45 minutes to two hours. Furthermore, certain types of donors (e.g. anti-D donors) require to be immunised with foreign material prior to apheresis.

During the past few years, many apheresis donors have been recruited by the Northern Ireland Blood Transfusion Service. These donors are organised into separate panels according to the products obtained from their donations, i.e. cells (granulocytes and platelets), anti-D and other specific immunoglobulins.

GRANULOCYTES AND PLATELETS

Granulocyte transfusion is sometimes indicated for infected patients who are also severely granulocytopaenic. The need for transfusion arises particularly during the treatment of leukaemia when severe bacterial infection, which is unresponsive to antibiotics, is present. To make it effective, a large quantity of white cells is required, and this is best obtained by cytopheresis, using the automated cell-separator machine sited in the Royal Victoria Hospital.

Platelets are normally obtained from single donations, but certain patients who become refractory to random-donor platelets may require platelets from HLA-matched donors. Invariably very few of the latter will be available, so the most practical method of collection is by plateletpheresis.

Granulocytes and single-donor platelets may be donated by relatives of the patient concerned. However, in many cases, suitable relatives cannot be found, and so in 1980 the NIBTS began recruitment of a panel of volunteers from the blood donor population. In order to avoid exerting undue moral pressure on existing donors, recruitment was carried out by a general publicity campaign rather than by a direct approach to individuals.

A code of practice for the organisation of such a panel of volunteers has been produced [1] to which strict adherence is required. Before being admitted to the donor panel, each potential volunteer is provided with a detailed explanation of the procedure, including full information about any risks involved. The approval of their general practitioner is also sought. Those wishing to proceed are then asked to sign a consent form. Most donors are under the age of 40; this is desirable, as donors over this age should, according to the code of practice, have an ECG and chest X-ray prior to each donation. The donor panel has at present about 200 members who provide around 60 donations of granulocytes and/or platelets per year.

ANTI-D IMMUNOGLOBULIN

The almost complete prevention of haemolytic disease of the newborn by the routine administration of anti-D immunoglobulin to all Rhesus (D) negative women soon after the delivery of a Rhesus-positive baby (introduced in 1968) has been a major medical advance. The success of this programme has, however, led to some problems in providing the raw material (human plasma containing high titre anti-D) from which the immunoglobulin is produced. Until recently, the major source of this plasma were those Rhesus-negative women who had produced anti-D as a result of pregnancy. Now that this is successfully prevented in most cases, such individuals have become very few in number. The only alternative source is Rhesus-negative men who volunteer to be deliberately immunised with Rhesus-positive blood, so as to stimulate the production of anti-D antibodies. A panel of such donors has been recruited during the past year by the NIBTS in order to meet the demand for anti-D immunoglobulin in the Province.

As with cell donors, no direct approach was made to recruit existing donors to the panel, and fully informed written consent was obtained before admission. Since the procedure involves the administration of repeated small blood transfusions (1-2 ml), rigorous precautions must be taken to ensure that potential side-effects, e.g. hepatitis, production of unwanted red cell antibodies, etc., are prevented. Thus, in addition to the usual pre-donation tests, blood used for immunisation is selected from a specially accredited donor panel. The latter have exhaustive and frequent tests carried out to ensure as far as possible that their blood will not transmit any of the hepatitis viruses. Furthermore, the donor and recipient are matched for all clinically significant blood groups apart from the Rhesus (D) antigen. This is to ensure that no unwanted red cell antibodies are produced which might increase the difficulty in obtaining compatible blood, should the individual ever require a blood transfusion. To this end, volunteers are also encouraged to wear an identity bracelet which is designed to alert their medical attendants to the importance of transfusing Rhesus-negative blood.

Once the anti-D titre has reached a sufficiently high level, plasma is collected by apheresis at about monthly intervals. Approximately 300 plasma donations per annum are required to meet current demand in the Province. This plasma is sent to the Protein Fractionation Centre, Edinburgh, for processing to anti-D immunoglobulin.

The future may see the introduction of antenatal prophylaxis of all Rhesus-negative women (to cover the occasional cases where antibodies are produced during pregnancy).[2] This practice has been shown to be effective, but while clinically desirable, it would cause an enormous (fourfold) increase in the requirement for anti-D and consequently for many more immunised donors. Some would argue that the risks to donors (although extremely small) do not justify the relatively small benefit which might result.[3]

OTHER SPECIFIC IMMUNOGLOBULINS

In addition to anti-D, a number of other specific immunoglobulins are produced from human plasma containing a high titre of the appropriate antibody. These include anti-Hepatitis B, anti-Tetanus and anti-Varicella Zoster, etc., which are used for post-exposure prophylaxis in certain 'at risk' individuals. Suitable donors of the source plasma are extremely scarce, so, when identified by prior laboratory testing, they are encouraged to donate by plasmapheresis. About 300 such donations are collected per annum.

FUTURE DEVELOPMENTS

As indicated above, apheresis procedures have been used hitherto on a small scale, being restricted to highly selected donors. This is likely to change in the near future as the demand for blood components (e.g. platelet concentrates and certain plasma products) normally collected from random donors, continues to increase rapidly. Since the demand for red cells is growing much more slowly, it seems logical to augment supplies of these non-red cell products by the adoption of apheresis procedures on a proportion of the normal blood donor population. Until recently, the methods available have been too cumbersome, slow and costly to allow apheresis to be used on a large scale. Devices are now being produced which make this much more feasible and have already been used for the collection of large volumes of platelets and plasma in some centres.[4]

It is likely that the demand for blood components will continue to increase at least in the short-term. Eventually, it is quite possible, as developments in genetic engineering enable some of these products to be synthesised, that the requirement for blood components of human origin will decrease.

REFERENCES

1. Department of Health and Social Security. A code of practice for the clinical use of blood cell separators. (Working party report). London, 1976 (CSWP 26).
2. McClelland WM, McLoughlin KG. Prevention of Rhesus(D) immunisation — some causes of failure in Northern Ireland. *Ulster Med J* 1980; **49**: 148.
3. Tovey LAD, Stephenson BJ, Townley A, Taverner J. The Yorkshire antenatal anti-D immunoglobulin trial in primigravidae. *Lancet* 1983; **2**: 244.
4. Robinson EA. Single donor granulocytes and platelets. *Clinics in Haematology* 1984; **13**: 185.

The Ulster Medical Journal, Volume 54, No. 1, pp. 58-60, April 1985.

Ampicillin resistance in *Haemophilus influenzae*

A C Lafong

Accepted 21st January 1985.

SUMMARY

Fourteen out of 150 (9.3%) consecutive strains of Haemophilus influenzae *isolated on culture of sputum in the Bacteriology Department, Belfast City Hospital, during 1982/83 were found to be ampicillin-resistant (ß-lactamase-producing). Susceptibility testing to other antibiotics of these ampicillin-resistant strains showed that cefuroxime, cefotaxime, gentamicin, and amoxycillin with clavulanic acid were reliable alternatives. Other useful alternatives included tetracycline, trimethoprim and co-trimoxazole. Erythromycin was of limited usefulness.*

INTRODUCTION

Haemophilus influenzae is an important pathogen in acute infective exacerbations of chronic bronchitis. Until 1974, ampicillin had been regarded as the drug of choice for treating these infective episodes as it was effective against virtually all strains of *Haemophilus influenzae.* Since then many reports have shown an increasing incidence of ampicillin-resistant strains of *Haemophilus influenzae* in the United Kingdom.[1,2] A large survey of antibiotic resistance in the United Kingdom in 1981[2] showed that 6.2% of 1,841 strains were resistant to ampicillin. The percentage ranged from 0% in Stockport to 11.5% in Portsmouth. The usual mechanism of ampicillin resistance in *Haemophilus influenzae* is the production by the organism of an enzyme, ß-lactamase, which inactivates the drug.[3]

To determine the current situation in Belfast, a study was carried out during the winter of 1982-83 on strains of *Haemophilus influenzae* isolated from sputum.

METHODS

One hundred and fifty consecutive strains of *Haemophilus influenzae* isolated from sputum were collected. Each strain was seeded on to a nutrient agar plate and V, X and XV factor discs were placed on the surface. The plates were then incubated in air at 37°C for 18 hours. Organisms that grew around the XV disc only (i.e. both X and V factor-dependent) were confirmed as *Haemophilus influenzae.* All the strains identified as *Haemophilus influenzae* were tested for ß-lactamase production using the chromogenic cephalosporin substrate method.[4]

Belfast City Hospital, Belfast BT9 7AB.
A C Lafong MB, BCh, Senior Registrar, Department of Bacteriology.

Each strain was seeded on to a Diagnostic Sensitivity Test (DST Oxoid) chocolate agar and filter paper discs containing ampicillin, tetracycline, erythromycin, trimethoprim, sulphonamide, co-trimoxazole, amoxycillin with clavulanic acid, cefuroxime and cefotaxime were applied to the surface. After incubation at 37°C for 18 hours, the zone sizes around the discs were measured and compared with a control-sensitive strain.

RESULTS

Of the 150 strains collected, all satisfied the conditions set out above for identification and were confirmed as *Haemophilus influenzae.* Fourteen out of the 150 strains (9.3%) produced ß-lactamase and were designated ampicillin-resistant. Tables 1 and 2 respectively show the resistance of the 14 ampicillin-resistant and 136 ampicillin-sensitive strains of *Haemophilus influenzae* to 10 antibiotics. Resistance was defined as a zone size smaller than for the control by more than 3 mm.

TABLE 1

Resistance of 14 ampicillin-resistant (ß-lactamase-producing) strains of Haemophilus influenzae *to 10 antibiotics*

Antibiotic	*No. Resistant (%)*	*Antibiotic*	*No. Resistant (%)*
Ampicillin	14 (100%)	Co-trimoxazole	3 (21%)
Tetracycline	3 (21%)	Gentamicin	0 (0%)
Erythromycin	6 (43%)	Amoxycillin/ Clavulanic acid	0 (0%)
Trimethoprim	2 (14%)	Cefuroxime	0 (0%)
Sulphonamide	3 (21%)	Cefotaxime	0 (0%)

TABLE 2

Resistance of 136 ampicillin-sensitive strains of Haemophilus influenzae *to 10 antibiotics*

Antibiotic	*No. Resistant (%)*	*Antibiotic*	*No. Resistant (%)*
Ampicillin	0 (0%)	Co-trimoxazole	8 (6%)
Tetracycline	8 (6%)	Gentamicin	0 (0%)
Erythromycin	54 (40%)	Amoxycillin/ Clavulanic acid	0 (0%)
Trimethoprim	5 (4%)	Cefuroxime	0 (0%)
Sulphonamide	8 (6%)	Cefotaxime	0 (0%)

All strains were sensitive to gentamicin, amoxycillin with clavulanic acid, cefuroxime and cefotaxime. A small percentage of the ampicillin-sensitive strains was resistant to tetracycline (6%), trimethoprim (4%), sulphonamide (6%) and co-trimaxazole (6%). Among the ampicillin-resistant strains this percentage was greater: 21% for tetracycline, 14% for trimethoprim, 21% for sulphonamide and 21% for co-trimoxazole. A significant percentage of both the ampicillin-resistant strains (43%) and the ampicillin-sensitive strains (40%) was resistant to erythromycin.

DISCUSSION

This survey showed that 14 out of 150 (9.3%) strains of *Haemophilus influenzae* isolated from sputum were resistant to ampicillin — a figure roughly similar to that obtained at some other centres in the U.K. Disc sensitivity testing of the 150 strains of *Haemophilus influenzae* to other antibiotics showed that cefuroxime, cefotaxime, gentamicin, and amoxycillin with clavulanic acid were 100% effective. Tetracycline, trimethoprim and co-trimoxazole showed good activity against the ampicillin-sensitive strains but were less effective against the ampicillin-resistant strains. These antibiotics could be useful alternatives in patients who are allergic or have other side-effects to ampicillin or amoxycillin. Trimethoprim may be preferable to co-trimoxazole because it has been shown to be equally effective in adults with chronic bronchitis or with pneumonia, has fewer side-effects (rashes, nausea etc.) and is marginally cheaper. Tetracycline has been used for many years in the treatment of bronchial infections and still has a place today. Erythromycin was only moderately active against both the ampicillin-resistant strains (43%) and the ampicillin-sensitive strains (40%) and it has only a limited place in the treatment of lower respiratory tract infections due to *Haemophilus influenzae.*

In view of the trend towards increasing ampicillin resistance among isolates of *Haemophilus influenzae,* other alternative antibiotics are likely to be of increasing importance for the treatment of infections due to the organism. They should be considered for patients not responding satisfactorily to ampicillin or amoxycillin or when these β-lactamase-producing strains of *Haemophilus influenzae* are isolated on culture of sputum. In very ill patients in hospital, parenteral therapy with one of the newer cephalosporins such as cefuroxime or cefotaxime would be the drug of choice especially before the results of sensitivity test are available. Gentamicin could also be used but it is a potentially toxic drug with a narrow therapeutic margin and it has no useful activity against the pneumococcus which is the other important pathogen in acute infective exacerbations of chronic bronchitis. Among the oral agents, amoxycillin with clavulanic acid is the drug of choice, although trimethoprim, co-trimoxazole and tetracycline may also be useful.

I wish to thank Dr T S Wilson, Dr W P Ferguson and Dr N McCracken for helpful comments and Miss M Maxwell for secretarial help. Amoxycillin with clavulanic acid is marketed as "Augmentin" (Beecham).

REFERENCES

1. Howard AJ. Ampicillin resistance in *Haemophilus influenzae. J Antimicrob Chemother* 1977; **3**: 535-7.
2. Philpott-Howard J, Williams JD. Increase in antibiotic resistance in *Haemophilus influenzae* in the United Kingdom since 1977: report of study group. *Br Med J* 1982; **284**: 1597-9.
3. Bell SM, Plowman D. Mechanisms of ampicillin resistance in *Haemophilus influenzae* from respiratory tract. *Lancet* 1980; i: 279-80.
4. O'Callaghan CH, Morris A, Kirby SM. Novel method for the detection of beta-lactamases using a chromogenic cephalosporin substrate. *Antimicrob Agents Chemother* 1972; **1**: 283-8.

The Ulster Medical Journal, Volume 54, No. 1, pp. 61-64, April 1985.

Surgical experience in necrotising enterocolitis: a report of nineteen cases

S R Potts, W I H Garstin

Accepted 30th January 1985.

SUMMARY

The results of operative intervention in 19 neonates with necrotising enterocolitis are reported. There were four deaths (21%) in the series. Intestinal perforation was the indication for surgery in 10 cases (55%), three of which did not survive. Timing of operative intervention can be optimised only by close liaison between experienced physicians and surgeons in order to avoid intestinal perforation.

INTRODUCTION

Necrotising enterocolitis occurs in approximately 2-5%[1] of premature and low birth weight babies. In the past decade the survival rate has increased from 20-30%[2] to 70-80%[3,4] with improved intensive care and parenteral nutrition facilities. Whilst approximately two-thirds of cases are treated medically and do not require surgery, it is accepted that laparotomy is indicated for intestinal perforation, obstruction and gangrene.[5] Other possible indications for surgery in necrotising enterocolitis, such as a clinically deteriorating abdomen or falling platelet count, are not agreed upon.[3,5] It is the purpose of this report to review the experience in the Royal Belfast Hospital for Sick Children over the past four years and thereby clarify the indications for laparotomy.

MATERIALS AND METHODS

The hospital records of all newborn infants undergoing laparotomy for necrotising enterocolitis between January 1980 and January 1984 were inspected. Gestational age, birth weight, sex and anoxic factors at birth were recorded. The age at onset of necrotising enterocolitis was noted. Exact details of feeding were difficult to determine from the records and were omitted from the study. The indications for surgery, the length of medical treatment, operative findings and procedures were tabulated. Pre-operative abdominal x-rays were inspected to establish or confirm the indications for surgery. The postoperative course was followed for signs of food intolerance suggested by loose stools and failure to thrive. The development of intestinal stricture was also recorded.[6]

The Royal Belfast Hospital for Sick Children.
S R Potts FRCS, Senior Surgical Registrar.
W I H Garstin FRCS, Surgical Registrar.
Correspondence to: S R Potts FRCS, The Royal Belfast Hospital for Sick Children, Belfast BT12 6BE.

RESULTS

There were 19 cases in the series (11 boys, 8 girls). The clinical profile is presented in Table 1. Nine babies were over 3 kg at birth. The same nine infants were over 38 weeks' gestation. Three babies were under 1500g at birth and were among five infants under 32 weeks' gestation. Examination of the data in Table 1 revealed no subgrouping, in that birth weight and gestational age did not relate clearly with the age at onset of necrotising enterocolitis or the length of medical treatment prior to surgery.

TABLE 1

Clinical profile of 19 cases of necrotising enterocolitis

Birth weight (grams)	*No. of cases*	*Gestational age at birth (weeks)*	*No. of cases*	*Age at onset of necrotising enterocolitis (days)*	*No. of cases*	*Length of medical treatment (days)*	*No. of cases*
< 1000	1	28 – 30	3	0 – 2	4	0 – 2	6
1000 – 1500	2	30 – 32	2	2 – 4	6	2 – 4	5
1500 – 2000	3	32 – 34	1	4 – 6	3	4 – 6	1
2000 – 2500	1	34 – 36	2	6 – 8	4	6 – 8	1
2500 – 3000	3	36 – 38	2	8 – 10	0	8 – 10	2
3000 – 3500	3	38 – 40	9	10 +	2	10 +	4
> 3500	6	40 +	0				

Table 2 shows the sites affected by necrotising enterocolitis. Intestinal perforation occurred in 10 cases (55%). In one case, two perforations were present in the terminal ileum. All perforations were diagnosed pre-operatively. Other indications for surgery were obstruction in three cases (17%), and peritonitis in five cases (25%). Six cases developed 11 intestinal strictures, all within three months.

TABLE 2

Sites affected by necrotising enterocolitis

	Stomach	*Jejunum*	*Ileum*	*Ascending colon*	*Transverse colon*	*Descending colon – rectum*	*Total colon*
Distribution of disease	1	2	6	11	11	10	8
Sites of perforation			5	1	3	2	
Sites of stricture				4	4	3	

The clinical features of the four who died are presented in Table 3. Although all four had resections these were not massive and were comparable with the five cases undergoing intestinal resection which survived. Three of those who died had anoxic factors at birth: (a) antepartum haemorrhage; (b) oxygen therapy

following caesarean section; (c) foetal distress during breech delivery. The significance of anoxic factors at birth in the study group was not possible to assess, but all cases demonstrated some stress factor.

TABLE 3

Clinical features of non-survivors from 19 cases of necrotising enterocolitis

Birth weight (grams)	*Gestational age (weeks)*	*Site of disease*	*Indication for surgery*	*Resection (intestinal)*
1700	29	Jejunum and ileum	Perforation	Yes
1700	31	Descending colon	Perforation	Yes
3500	39	Total colon	Perforation	Yes
2700	33	Ileum and ascending colon	Peritonitis	Yes

DISCUSSION

Necrotising enterocolitis is diagnosed on clinical and radiological grounds. The classical sign on abdominal x-ray is intestinal pneumatosis but this feature is not present in every case.[7] The heavy reliance on clinical criteria therefore makes the true incidence of the disease difficult to ascertain. The authors, however, estimate that the cases reported in the text represent approximately 20% of the total cases occurring during the same period in Northern Ireland. Most cases of necrotising enterocolitis can and should be treated medically. It is beyond the scope of this paper to discuss this disease in depth but the features exhibited are typical of those reported worldwide.[3, 4, 7, 8]

The outstanding feature of this report is that intestinal perforation was the indication for surgery in 10 cases (55%). In this situation intestinal perforation carries a 50% rate of intestinal resection, 30% mortality rate plus attendant morbidity indicating that earlier operative intervention when possible could improve the outlook in certain instances. None of the four deaths (21%) was related directly or indirectly to surgery or anaesthesia per se (the senior author was personally involved with these cases), therefore there should be no reluctance in submitting a fully resuscitated newborn infant to exploratory laparotomy in the face of a deteriorating clinical situation. It must be remembered that a pneumoperitoneum may not develop after perforation[7] as a consequence of efficient nasogastric suction producing a paucity of intestinal gas. In addition, a small perforation may be sealed with or without resultant abscess formation. Acute perforation in fulminant disease is impossible to avoid in all cases but intestinal perforation must not be regarded as an inevitable development in a baby which has been closely watched throughout the progress of the disease. Laparotomy will not disadvantage a properly prepared child and offers the opportunity to exteriorise the bowel and divert the substrate for the infective process away from the site of the disease.

The authors wish to thank Mr B T Smyth, Mr V E Boston and Mr S Brown, Consultant Paediatric Surgeons, for their advice in compiling this study.

REFERENCES

1. Thomas DFM. Pathogenesis of neonatal necrotising enterocolitis. *J R Soc Med* 1982; **75**: 838-840.
2. Santulli TV, Schullinger JN, Heird WC, et al. Acute necrotising enterocolitis in infancy: a review of 64 cases. *Pediatrics* 1975; **55**: 376-387.
3. Kosloske AM. Necrotising enterocolitis in the neonate. *Surg Gynecol Obstet* 1979; **148**: 259-269.
4. Burrington JD. Necrotising enterocolitis in the newborn infant. *Clin Perinatol* 1978; **5**: 29-43.
5. Wayne ER, Burrington JD, Hutter J. Neonatal necrotising enterocolitis — evolution of new principles in management. *Arch Surg* 1975; **110**: 476-480.
6. Janik JS, Ein SH, Mancer K. Intestinal stricture after necrotising enterocolitis. *J Pediatr Surg* 1981; **16**: 438-443.
7. Rabinowitz JG, Siegle RL. Changing clinical and roentgenographic patterns of necrotising enterocolitis. *Am J Roentgenol* 1976; **126**: 560-566.
8. Polin RA, Pollack A, Barlow B, et al. Necrotising enterocolitis in term infants. *J Pediatr* 1976; **89**: 460-462.

The Ulster Medical Journal, Volume 54, No. 1, pp. 65-67, April 1985.

Management of childhood urinary tract infection

J McAloon, J G Jenkins, J H K Lim

Accepted 15th Febuary 1985.

SUMMARY

Experience with childhood urinary tract infection is reviewed in conjunction with recent information on the management of the problem by family practitioners in the same health board. The need for bacteriological confirmation of the diagnosis in every case is confirmed and the importance of radiological investigation of the urinary tract after a first infection irrespective of age or sex is emphasised, as 17% of intravenous pyelograms and 31% of micturating cystogram examinations showed significant abnormality.

INTRODUCTION

In a recent survey on the management of childhood urinary tract infection in family practice in this area,[1] it was found that a minority of affected children have their infection documented with culture of midstream urine (MSU) and that a minority of practitioners arrange further investigations. A retrospective study of the experience of the Waveney, Massereene and Mid-Ulster Hospitals with childhood urinary tract infection has consequently been carried out to assess the possible implications of the survey findings.

PATIENTS AND METHODS

A review was made of the case sheets of 82 consecutive new referrals to the paediatric departments at the Waveney, Massereene and Mid-Ulster Hospitals in whom urinary infection was suspected or proven either initially or subsequently by culture of MSU. The review period was from January 1982 to July 1983. No children with spina bifida were included.

Twenty-nine children (21 boys, 8 girls) were aged up to 23 months (group A); 26 (5 boys, 21 girls) were between 2 and 6 years (group B); 27 (6 boys, 21 girls) were older (group C). Forty were initially referred to the out-patient department and 42 were admitted to hospital with an acute illness. Initial diagnoses are shown in the Table.

Waveney Hospital, Ballymena.

J McAloon DCH, MRCP, Registrar.
J G Jenkins MD, MRCP, Consultant Paediatrician.
J H K Lim DCH, FRCP, Consultant Paediatrician.

Correspondence to: Dr J McAloon, Division of Developmental Paediatrics, Queen's University, Kingston, Ontario, Canada K7M, 1B6.

TABLE

Initial diagnoses

Diagnosis	*Number of patients*	*Age group(s)*
History of urinary tract infection		
Proven	30	ABC
Suspected	10	BC
Acute episode of urinary tract infection	14	ABC
Recurrent abdominal pain	2	C
Fever and vomiting		
(i) Otitis media	10	AB
(ii) Tonsillitis	7	AB
(iii) Gastroenteritis	6	A
Jaundice for investigation	1	A
Congenital pyloric stenosis	2	A

RESULTS

All children in groups A and B had both an intravenous pyelogram and a micturating cystogram. Children in group C had initially a pyelogram alone, but a cystogram was performed if the pyelogram proved abnormal.

Abnormalities were found in 17% of the pyelogram examinations, as follows: chronic pyelonephritis (9 cases); renal calculus (1 case); renal calculus plus hydronephrosis (1 case); megaureter plus hydronephrosis (3 cases). Five of the 14 children were in group A (all boys), 4 were in group B (2 boys, 2 girls) and 5 were in group C (all girls).

The micturating cystogram was considered abnormal if there was reflux above the pelvic brim or if there was a lesser degree of reflux but with proven recurrent infection. Thirty-one per cent of the examinations were abnormal (5 boys, 14 girls). Eleven children were in groups A and B, 8 were in group C.

All of the children were commenced on a programme of long-term follow-up with repeat urine cultures to detect any recurrence. Additionally, all of the children in groups A and B with ureteric reflux above the pelvic brim and children in group C with reflux and recurrent infection had a period of antibiotic prophylaxis. Seven children have had surgery performed on their urinary tracts.

DISCUSSION

Establishing the diagnosis of urinary infection in children, especially in young children, can be difficult. This was illustrated well by two infants with vomiting and surgically proven congenital pyloric stenosis who had a coincidental urinary tract infection proven by culture of urine obtained by bladder aspiration. Difficulty was also experienced with older children. The oldest child in the series was a 13-year-old girl referred for investigation of recurrent abdominal pains. This girl admitted to no symptoms specifically related to the urinary tract, but subsequently urine cultures documented urinary infection and investigations revealed bilateral renal scarring and bilateral ureteric reflux to renal level. There were other children in whom the diagnosis could have been missed, had treatment of the illness been

commenced without first obtaining a midstream urine specimen for culture. In contrast, 10 of the children were investigated without having had an infection documented. These children were referred to an outpatient clinic because of suspected urinary tract infection and, after further elaboration of the history, it was felt that on balance it was in the children's best interest to presume that there had been previous infection(s). Six children (groups A and B) had both a pyelogram and a cystogram while the other 4 (group C) had pyelography alone. The only abnormality discovered was ureteric reflux in a girl in group B. This observation further emphasises the value of obtaining a midstream urine specimen for culture in all cases before commencing antibiotic treatment.

The majority of abnormal findings occurred in groups A and B. This may reflect the tendency for ureteric reflux to improve with age. However, it may be that groups A and B are over-represented in this study in terms of the community as a whole, because younger children tend to be iller when they develop a urinary infection, and consequently more young children are referred to the paediatric department. This in turn raises concern that there may be a higher number of older children in the community with unrecognised risk factors. Such concern is consistent with our finding that only a minority of family physicians elect to refer their patients for further investigation.[1] It is interesting that sex was not a good index of underlying abnormalities, and this supports our practice of not discriminating on the grounds of sex when considering investigations.

The role of radiological investigation of the renal tract following urinary tract infection in childhood has recently been questioned,[2] and it has been suggested that the high incidence of abnormalities found in hospital-based studies may be due to their intrinsic selection of younger and more seriously ill patients. However, the results of the present study tally with those of Williams,[3] Dighe,[4] and Brooks,[5] who are all in agreement with the generally accepted guidelines that all children, irrespective of age or sex, should have radiological investigation of their urinary tracts after a first infection proven on urine culture, as the incidence of significant abnormalities found in any population group is high enough to make investigation essential.

We would like to thank the radiologists who performed the investigations in these children.

REFERENCES

1. McAloon J, Jenkins, Lim J. Management of childhood urinary tract infection in family practice. *Br Med J* 1984; **288**: 1729-30.
2. Houston HLA. Urinary tract infection in children. *Br Med J* 1984; **289**: 766.
3. Williams CM. Urinary tract infection in children. *Aust Fam Physician* 1976; **5**: 340-44.
4. Dighe AM, Grace JF. General practice management of childhood urinary tract infections. *J R Coll Gen Pract* 1984; **34**: 324-27.
5. Brooks D, Houston IB. Symptomatic urinary infection in childhood. *J R Coll Gen Pract* 1977; **22**: 678-83.

The Ulster Medical Journal, Volume 54, No. 1, pp. 68-71, April 1985.

Case report

Placental sulphatase deficiency

Paul P Fogarty

Jubilee Maternity Hospital, Belfast City Hospital.

Accepted 1st January 1985.

INTRODUCTION

Placental sulphatase deficiency/congenital ichthyosis is an X-linked inborn error of metabolism which was first described in 1969 by France and Liggins.[1] It is an enzymatic defect affecting steroid metabolism, clinically manifested by diminished oestrogen production during fetal life and by congenital ichthyosis post-natally. This disorder has a reported incidence of between 1 : 6,000 and 1 : 10,000 males.

CASE HISTORY

A 33-year-old woman presented at the Jubilee Maternity Unit, Belfast City Hospital, at 27 weeks' gestation and this was confirmed by ultrasonic scan. Her weight was 68.8 kg, blood pressure 110/80 mm Hg, haemoglobin 14.2 g/dl, blood group O (Rhesus-negative), and no atypical red cell antibodies were detected. The patient claimed to smoke 15 cigarettes per day. She had previously had a 12-week spontaneous abortion followed by a successful pregnancy. Throughout that pregnancy she was recorded as losing weight; labour was induced at 41 weeks and five days, and after an eight-hour labour she had a forceps delivery of a 2750 g female infant.

In her present pregnancy she was reviewed at 32 weeks' gestation and then two weeks later when, because of a weight loss of 2 kg and proteinuria, she was admitted for fetal assessment. Ultrasonic scan, cardiotochography, maternal kick-chart, haemoglobin and serum human placental lactogen were all satisfactory. A midstream sample of urine showed a significant urinary tract infection which required treatment. The urinary oestriol/creatinine ratio was reported as 0.9 nmol/μmol (normal >5); this was repeated and was consistently low (Fig. 1).

In view of the markedly low urinary oestriol results, the diagnosis of placental sulphatase deficiency was raised. Further enquiry revealed a family history of 'dry skin' affecting only male members (Fig. 2). Deficiency of the sulphatase enzyme was confirmed by the intravenous administration of 50 mg dehydroepiandrosterone sulphate (DHEAS), an oestrogen precursor. Serum oestradiol was measured at 5, 10, 15, 20, 30, 60 and 120 minutes following this and revealed no change from the pre-injection level of 5.0 nmol/l. Twenty-four hours later, 50 mg of dehydroepiandrosterone (DHEA) was administered intravenously. This compound does not require a sulphatase enzyme and serum oestradiol rose from the base line of 5.0 nmol/l to a maximum 175 nmol/l after only 10 minutes. Following this, serum oestradiol declined slowly to 50 nmol/l after 2 hours. This significant rise in serum oestradiol following the injection of DHEA but not that of DHEAS was evidence that there was placental sulphatase deficiency.

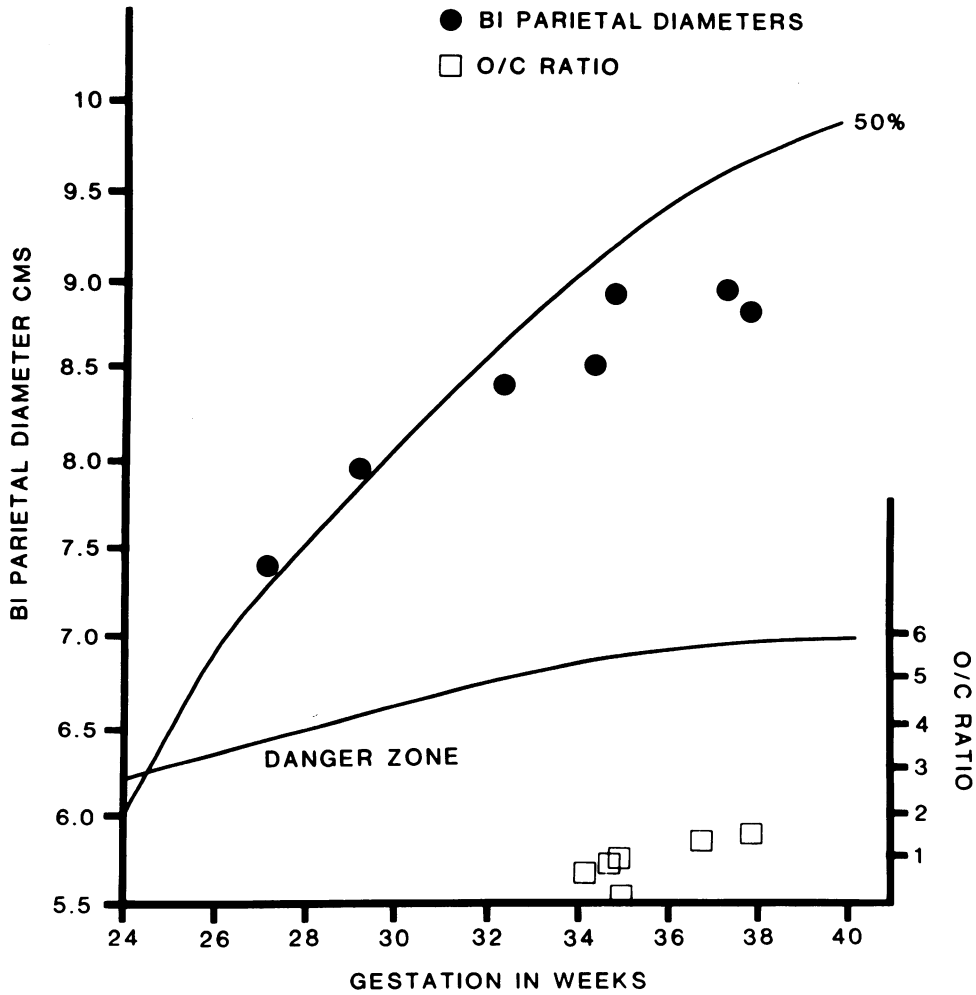


FIGURE 1. Urinary oestriol/creatinine (o/c) ratio and fetal bi-parietal diameter from week 24 to 38.

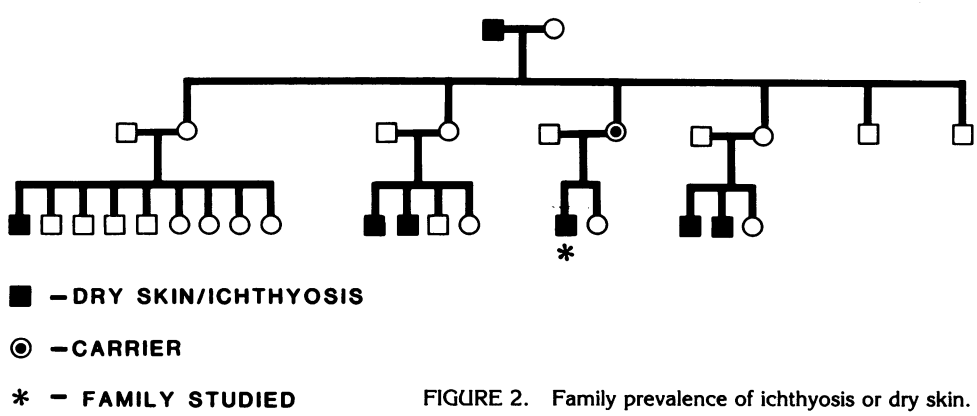


FIGURE 2. Family prevalence of ichthyosis or dry skin.

Ultrasonic scan was unsuccessful in determining the sex of the fetus. The patient was allowed to go home and was reviewed weekly. She was re-admitted at 37½ weeks' gestation, having lost a further 2 kg. Her blood pressure had risen to 140/90 mm Hg and proteinuria persisted. Serial ultrasonic scans demonstrated intrauterine growth retardation (IUGR) with an estimated fetal weight of 2 kg. Repeated urinary oestriol/creatinine ratios (O/C) remained low. It was decided to terminate the pregnancy by forewater amniotomy and intravenous syntocinon infusion. She laboured slowly at first, with a long latent phase of five hours, and required a lumbar epidural anaesthetic. Following this the cervix soon reached full dilatation. Because of fetal distress, she had a forceps delivery of a healthy male infant weighing 2125 grams. He showed no skin abnormality at birth but by the age of six weeks he was already developing severe ichthyosis.

The placenta was retained and removed manually. A specimen of one cotyledon was frozen and sent for enzyme analysis. The results confirmed a sulphatase deficiency with normal microsomal and lysosomal enzymes.

DISCUSSION

The human placenta in isolation is incapable of oestrogen synthesis without assistance from the fetal liver and the adrenal cortex. DHEAS is the major oestrogen precursor produced by the fetal and maternal adrenal glands. It is converted by the enzyme 3 ß-hydroxysteroid sulphatase which removes the sulphate group, leaving the neutral C-19 steroid nucleus which is then aromatised to oestrogen.

Prenatally, steroid sulphatase deficiency leads to reduced oestrogen production in spite of which pregnancy continues and is often prolonged. No antenatal danger has yet been shown and the low oestrogens themselves are not an indication for early interference. However, in this case, concomitant intrauterine growth retardation pointed to the need for induction. The low level of oestrogen, as in the anencephalic fetus, often delays the onset of labour, especially in the primigravida.[2] The cervix is slow to dilate, with an increase in cervical dystocia, which leads to a higher rate of Caesarean section.

Postnatally the enzyme deficiency caused an accumulation of cholesterol sulphate in the blood, cornea and skin leading to the development within the first year of ichthyosis and corneal opacities.

The genetic locus for this condition is found on the short arm of the X-chromosome at the Xg blood group locus and is one of the few genes that is not subject to inactivation. This family's pedigree is consistent with X-linkage. The prenatal diagnosis of sulphatase deficiency must be considered when urinary oestrogens are found to be low. There are many causes, such as drug therapy (using corticosteroids, antibiotics, meprobamate, mondelamine) acute urinary tract infections, anencephally, congenital adrenocortical hypoplasia and incorrect 'dates' which can lead to a reduction in urinary oestrogens. If these are excluded, sulphatase deficiency can be isolated as in this case by loading the patient with DHEAS and DHEA and following the subsequent oestradiol responses.[5] After delivery, the placenta can be analysed for deficient 3 ß-hydroxysteroid sulphase.[3] Affected individuals with X-linked ichthyosis will have a marked (15 to 30 times) elevation of serum cholesterol sulphate.[4] Therapeutically some relief is obtained from topical urea preparations. Genetic counselling and reassurance remain important in the management of patients with this disorder.

I wish to thank Dr E B Bond (Consultant Obstetrician, Belfast City Hospital) for his help in this report; my thanks are also due to Professor N C Nevin (Department of Medical Genetics, Queen's University), Mr B Sheridan (Senior Biochemist, Royal Victoria Hospital), Mrs V King (Senior Pharmacist, Belfast City Hospital), Dr F A Rose (Biochemist, Cardiff University) and Marion Crawford and Anne Petticrew (typists).

REFERENCES

1. France JT, Liggins GC. Placenta sulphatase deficiency. *Clin Endocrinol* 1969; **29**: 138-41.
2. Stanbury JB et al. The metabolic basis of disease. New York, London: McGraw-Hill, 1978; 1027-39.
3. Harkness RA, Taylor NF, Crawford MA et al. Recognising placental steroid sulphatase deficiencies. *Br Med J* 1983; **287**: 2-3.
4. Shapiro LJ, Weiss R, Webster D et al. X-linked ichthyosis due to steroid-sulphatase deficiencies. *Lancet* 1978; **1**: 70-72.
5. Oakey RE. Antenatal detection of placental steroid sulphatase deficiency. *Br J Obstet Gynaecol* 1984; **91**: 337-41.

The Ulster Medical Journal, Volume 54, No. 1, pp. 72-74, April 1985.

Case report

Spontaneous haemopneumothorax

Philippa Whitford, C F J Russell

Accepted 21st January 1985.

Spontaneous haemopneumothorax, although well documented, is a condition rarely encountered by the general surgeon. As a result of hypovolaemia and loss of respiratory function, patients with this condition are seriously ill and thus require early diagnosis and urgent treatment. We report a further patient with this interesting and potentially dangerous clinical entity.

A 32-year-old woman underwent surgical excision of a lump in her left breast in a general surgical unit of the Royal Victoria Hospital. Clinical diagnosis of fibro-adenosis was confirmed by frozen section histology. Anaesthesia was uneventful, the patient breathing spontaneously throughout the operation via a face mask. She was well post-operatively and was discharged on the evening of the surgery.

On the third post-operative day she experienced sudden, severe pain in the left shoulder and scapular region which was pleuritic in nature and accompanied by acute shortness of breath. Over the next few hours the pain became more severe, the dyspnoea more marked and she was re-admitted to hospital. On examination the patient looked ill. She was cold, clammy and grossly dyspnoeic. However, the pulse rate was 95 per minute and of good volume and the blood pressure 130/50 mmHg. Examination of the chest revealed a shift of the trachea to the right with hyper-resonance in the left apical region and stony dullness at the left base on percussion. On auscultation complete lack of air entry to the left hemithorax was noted. Chest radiograph confirmed an extensive left-sided pneumothorax with a large effusion at the left base (Fig.). A distinct air/fluid level was present and a diagnosis of haemopneumothorax was made. The haemoglobin was 8.6 g/dl.

Pleural drains, one apical and one basal, were inserted under local anaesthesia. Air bubbled from the apical drain and 1,200 ml of altered blood rapidly discharged from the inferior drain. Repeat chest x-ray showed good re-expansion of the left lung. The anaemia was corrected by transfusion of four units of blood.

Subsequently the patient made an uneventful recovery. The chest drains were removed on the fourth day and she was discharged home ten days after admission. At review six weeks later, she remained symptomatically and clinically well with a normal chest x-ray.

Royal Victoria Hospital, Belfast BT12 6BA.
Philippa Whitford MB, Senior House Officer in Surgery.
C F J Russell FRCS, Consultant Surgeon.
Correspondence to: Mr C F J Russell.

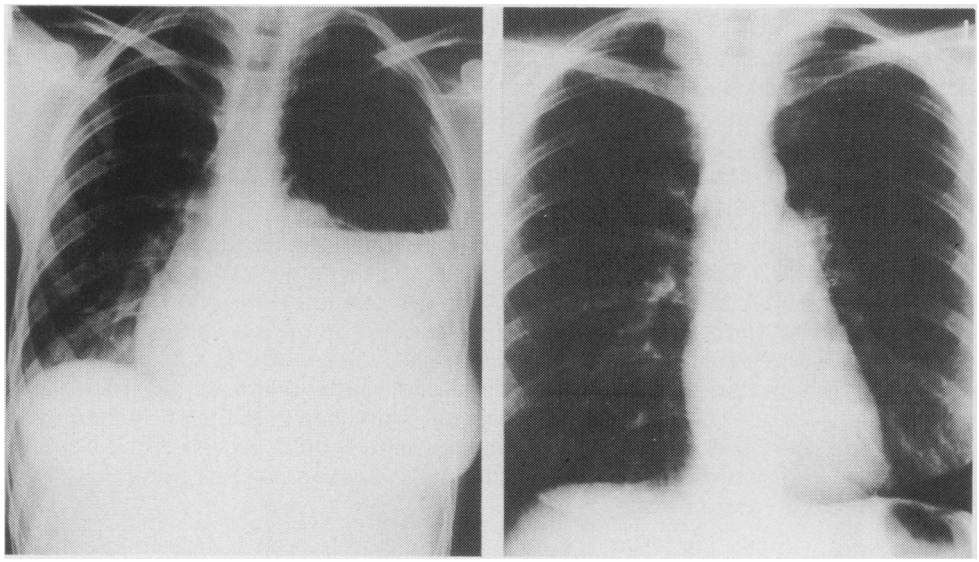


FIGURE.

Chest radiographs showing (left) large haemopneumothorax left side and (right) complete resolution and full lung re-expansion 6 weeks later.

COMMENT

Spontaneous haemopneumothorax, though relatively rare, occurs most frequently when a congenital bulla or bleb coincides with the presence of vascularized adhesions between the visceral and parietal pleura.[1] The lung lesion is the underlying cause of the pneumothorax, the haemothorax developing when the two pleural layers are pulled apart as the lung collapses. Due to the relative lack of constrictive muscle in the arterioles in these adhesions,[2] vasoconstriction does not occur and large quantities of blood may be lost into the pleural cavity. Haemopneumothorax is also seen as a secondary phenomenon in association with primary lung pathology such as carcinoma or tuberculosis. Clearly, these conditions must be excluded in the investigation of the patient with haemopneumothorax.

When no underlying pathological process can be readily identified, a diagnosis of spontaneous haemopneumothorax is justified. This condition is seen in 2·5% of patients with spontaneous pneumothorax.[3] Generally, it appears to affect a specific population group, namely young white males, occurring only occasionally in coloured patients or females. The age group involved is usually 15·45 years with a documented preponderance of males in a ratio of 15:1.[4] Our patient had no previous history of lung disease and subsequent investigation has revealed no evidence of underlying pulmonary or pleural pathology. We cannot relate the development of haemopneumothorax to her earlier minor surgical procedure or anaesthetic and feel therefore that a diagnosis of spontaneous haemopneumothorax is appropriate. The fact that the patient is female is noteworthy.

Treatment of this condition must be prompt and aggressive, combining adequate intravenous transfusion with removal of blood from the chest cavity and re-expansion of the lung. If early diagnosis is made, the insertion of two chest drains,

one basal and one apical, is usually all that is required, provided active bleeding ceases. If haemorrhage persists, thoracotomy may be necessary in order to control the source; stapling of bullae may be performed at this time. Formal chest exploration is also indicated, if diagnosis is delayed, so that complete removal of blood clots can be accomplished. Residual haematoma within the pleural cavity may result in subsequent fibrosis and lung constriction. If scar tissue does form, decortication may be required.

Some 30 years ago the reported mortality figure for spontaneous haemopneumothorax was 27%.[5] Treatment then consisted of simple aspiration of the pleural cavity. The introduction of more aggressive management in the form of wide bore chest drains and thoracotomy as required resulted in a rapid fall in mortality to 15%.[6] Today, the vast majority of patients might be expected to survive provided an awareness of the condition is maintained, early diagnosis is made and immediate treatment is instituted. The possible significance of the combination of gross blood loss and respiratory embarrassment should be remembered and spontaneous haemopneumothorax should still be recognised as potentially life-threatening in a young person.

REFERENCES

1. Faye C, Le Goaziou F, Colchen A, Personne C. Spontaneous haemopneomothorax in a young adult. *Nouv Presse Med* 1974; **3**: 1728-1729.
2. Deaton WR, Johnston FR. Spontaneous haemopneumothorax. *J Thorac Cardiovasc Surg* 1962; **43**: 413-415.
3. Hyde L, Hyde B. Benign spontaneous haemopneumothorax. *Amer Rev Tuberc* 1951; **63**: 417-426.
4. Stradling P. Spontaneous haemopneumothorax. *Proc Roy Soc Med* 1964; **57**: 329-330.
5. Cunningham JAK. Spontaneous haemopneumothorax. *NZ Med J* 1950; **49**: 708-712.
6. Calvert RJ, Smith E. Spontaneous haemopneumothorax. *Thorax* 1955; **10**: 64-72.

The Ulster Medical Journal, Volume 54, No. 1, pp. 75-77, April 1985.

An Otofuke-like virus associated with diarrhoea. Case report and electronmicroscopic study

H J O'Neill, J H Connolly, A O B Redmond, E Dermott

Accepted 21st January 1985.

SUMMARY

Virus particles similar to Otofuke virus have been found, together with rotaviruses and astroviruses, by electronmicroscopy in faeces from an infant with diarrhoea in Northern Ireland. Previously Otofuke virus has been found only in Japan whence it may have been carried to this country by a businessman.

INTRODUCTION

Several types of viruses may be seen by electronmicroscopy in faeces from childhood diarrhoea. These include rotaviruses, adenoviruses, coronaviruses and reoviruses. In addition there are small round viruses which may be classified into two groups.[1] The first, which includes enteroviruses and parvoviruses, comprises viruses with no surface structure and a smooth entire outer edge; the second, which includes astroviruses, caliciviruses and the Norwalk group of viruses comprises viruses with a structured surface and/or ragged edge.

We report the occurrence of a small round virus (34-36 nm) with a surface structure, in association with smaller particles (15-17 nm), in faeces from a child with diarrhoea. We consider this virus to be similar to the Otofuke agent which to date has been reported only in Japan.

CASE REPORT: METHODS AND RESULTS

A six-month-old boy from Downpatrick, Northern Ireland, was admitted to hospital in December 1982 for investigation of screaming after feeds associated with passage of foul smelling stools. The symptoms had started at the age of four weeks, but despite this he gained weight and was thriving. There were three other

Regional Virus Laboratory, Royal Victoria Hospital, Belfast.
H J O'Neill BA, FIMLS, Chief Medical Laboratory Scientific Officer.
J H Connolly MD, FRCPI, FRCPath, Consultant Virologist.

Belfast City Hospital.
A O B Redmond MB, FRCPI, Consultant Paediatrician.

Department of Microbiology and Immunobiology, The Queen's University of Belfast.
E Dermott BSc, PhD, Lecturer.

Correspondence to: Dr J H Connolly, Regional Virus Laboratory, Royal Victoria Hospital, Belfast BT12 6BN, Northern Ireland.

children in the family who were asymptomatic. On admission the child was put on clear fluids orally. The diarrhoea quickly subsided and he was discharged after one week as his bowel movements had returned to normal. While in hospital, urea, electrolytes, sweat test and chest x-ray were normal and bacteriology of faeces revealed no pathogens. At review, six weeks after discharge the baby remained asymptomatic.

Virus was not isolated from faeces in baboon kidney or HEp2 cells. The supernatant fluid from a 10% faecal suspension previously centifruged at 4,500 g for 1 hr was re-centrifuged at 180,000 g for 1 hr. The resultant pellet was resuspended in 25 µl distilled water, transferred to a carbon/formvar coated grid, stained with 2% phosphotungstic acid pH7 and examined in a Philips 301 electron microscope. Clusters of astroviruses measuring 28 nm and rotaviruses were seen. On the same grid an aggregate of two sizes of particles, the appearance of which suggested that they were bound by antibody, was seen (Fig. 1a). All the smaller size particles were 'empty' and measured 15-17 nm. The larger particles measured 34-36 nm, had obvious icosahedral structure and surface projections. One particle (Fig. 1b) when subjected to Markham rotation [2] showed enhancement of 10 surface projections only when n = 5 (Fig. 1c).

Enquiries revealed that the child's father had visited Japan on business, the last occasion being in May 1981. However, since then he has met Japanese businessmen every month in Northern Ireland.

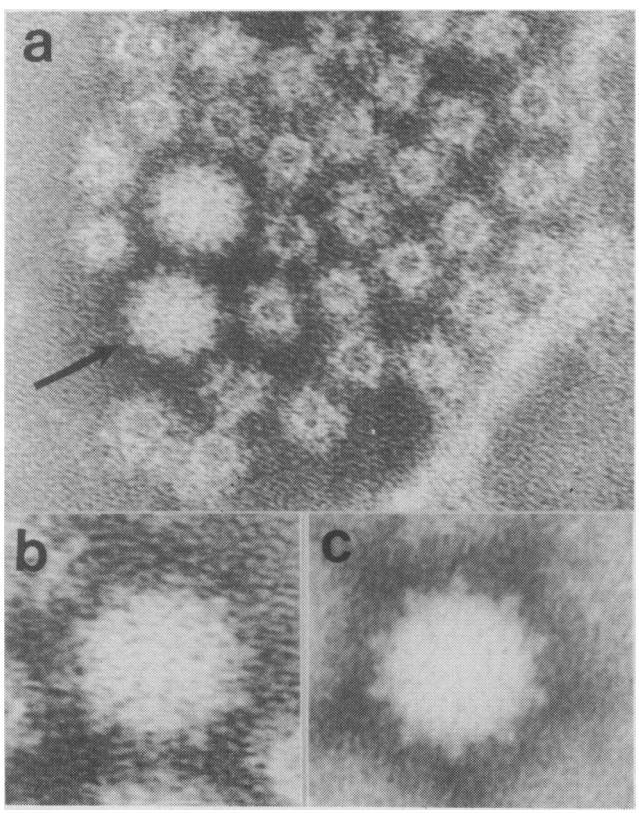


FIGURE.

(a)
Two 34-36 nm particles aggregated with smaller 15-17 nm particles × 450,000.

(b)
arrowed particle in (a) × 750,000.

(c)
same particle rotated n = 5 by Markham technique showing enhancement of 10 peripheral projections.

DISCUSSION

The size and structure of the larger particle described here is similar to the Otofuke virus[3] which also has been shown to have an antigenically related smaller particle.[4] The Sapporo virus in human diarrhoea[5] and the Newbury agent in calf diarrhoea[6] also have been shown to have smaller 'empty' particles associated with larger structured virus particles, but the published illustrations of these show a different structure from the virus described here and the Otofuke virus.

The Otofuke virus was found in faeces from children and adults with gastroenteritis in institutional outbreaks in Japan, but since rotaviruses and astroviruses are both capable of causing diarrhoea, the contribution of the Otofuke-like virus to the diarrhoea in this child is unknown. It is unlikely that the father brought the virus from Japan and infected the child, since his last visit was more than a year before the child's birth. It is possible, however, that the virus was carried by a Japanese businessman to this family. Undoubtedly similar contacts are made elsewhere and therefore it would be worthwhile looking for this virus in outbreaks of gastroenteritis in the United Kingdom.

We thank Dr E O Caul, Public Health Laboratory, Bristol, and Dr A M Field, Central Public Health Laboratory, London, for helpful comments on identification of the virus.

REFERENCES

1. Caul EO, Appleton H. The electronmicroscopical and physical characteristics of small round human fecal viruses: an interim scheme for classification. *J Med Virol* 1982; **9**: 256-265.
2. Markham RS, Frey S, Hills GJ. Methods for the enhancement of image detail and accentuation of structure in electronmicroscopy. *Virology* 1963; **20**: 88-102.
3. Taniguchi K, Urasawa S, Urasawa T. Virus-like particle, 35-40 nm, associated with an institutional outbreak of acute gastroenteritis in adults. *J Clin Microbiol* 1979; **10**: 730-736.
4. Taniguchi K, Urasawa S, Urasawa T. Further studies of 35-40 nm virus-like particles associated with outbreaks of acute gastroenteritis. *J Med Microbiol* 1981; **14**: 107-118.
5. Kogasaka R, Nakamura S, Chiba S, Sakuma Y, Terashima H, Yokoyama T, Nakao T. The 33-39 nm virus-like particles, tentatively designated as Sapporo agent, associated with an outbreak of acute gastroenteritis. *J Med Virol* 1981; **8**: 187-193.
6. Woode GN, Bridger JC. Isolation of small viruses resembling astroviruses and caliciviruses from acute enteritis of calves, *J Med Microbiol* 1978; **11**: 441-452.

The Ulster Medical Journal, Volume 54, No. 1, pp. 78-79, April 1985.

Case report

Perforated jejunal diverticulum presenting with a psoas abscess

R J Brown, R L E Thompson

Accepted 18th February 1985.

INTRODUCTION

Jejunal diverticulosis is an uncommon clinical condition. Since the decline of tuberculosis disease of the spine, psoas abscess too is rarely seen. We report a unique combination of these two conditions.

CASE REPORT

A 73 year old man presented with loss of mobility, anorexia, weight loss and pain in the left hip. Recent investigation of altered bowel habit had shown extensive diverticular disease of the sigmoid colon. On examination he was cachectic, dehydrated and pyrexic. There was redness, increased temperature, swelling and crepitus in the left groin with fixed flexion deformity and painful limitation of movement of the left hip. There were no abdominal signs. He had a leucocytosis and raised E.S.R. X-rays of his hips were normal. Coliforms, streptococcus faecalis and proteus were cultured and 1 ml of pus was aspirated from the left groin. Initial treatment was with metronidazole and cephazolin.

Computerised tomography confirmed our clinical impression of a large left psoas abscess. Shortly afterwards he became septicaemic, necessitating urgent drainage of the abscess through the left groin. Initially his condition improved with the release of pus, but after two days small bowel content appeared in the discharge and he again became septicaemic. At laparotomy he was found to have multiple diverticula on the mesenteric border of the jejunum. One of these was adherent to the posterior abdominal wall and communicated via a psoas abscess with the left groin incision. The diverticular disease of the colon was not involved. Resection of 30 cm of jejunum was required. Post-operatively he recovered normal bowel activity and both wounds healed well. Unfortunately despite prophylactic heparinisation he died two weeks after laparotomy from a pulmonary embolus.

DISCUSSION

Jejunal diverticulosis is uncommon. It affects about 1 % of the population though the true incidence may be much higher as the majority of cases are asymptomatic.

Belfast City Hospital.
R J Brown FRCS, Registrar.
R L E Thompson MB BCh, Senior House Officer.
Correspondence to: Mr R J Brown FRCS, Department of Surgery, The Queen's University of Belfast, Institute of Clinical Science, Grosvenor Road, Belfast BT12 6BJ.

When symptoms do occur, patients are usually over 70 years old and present with chronic abdominal pain or symptoms of malabsorption. Acute complications occur in less that 10% of cases and the more commonly reported of these are haemorrhage, perforation, diverticulitis without perforation and intestinal obstruction. The majority of jejunal diverticula are merely incidental findings at laparotomy, post mortem or on barium studies. These acquired protrusions into the mesenteric of the jejunum may remain unseen at laparotomy unless the surgeon distends the jejunum by manipulation to inflate the diverticula.[1]

Surgical intervention is indicated only for severe symptoms and acute complications. Certainly the incidental finding of jejunal diverticula at laparotomy does not warrant a resection. When the diverticula are extensive, it would be unwise to attempt a complete excision of the affected length of jejunum. However, with a limited resection in the presence of extensive disease, end to end anastomosis may be technically difficult.[2]

Psoas abscess, once a fairly common complication of tuberculosis of the spine, is now quite rare and more likely to be non-tuberculous. Apart from a small number of cases of primary staphylococcal psoas abscess, the suppuration can usually be traced to some adjacent septic focus. Sepsis complicating gastrointestinal perforation is the most common cause, particularly perforated appendix and perforated sigmoid diverticula. Other sources are suppurative iliac lymphadenitis, vertebral osteomyelitis and perinephric abscess.[3]

The diagnosis can be made on clinical grounds. There are usually signs of inflammation in the groin and the hip is flexed. Rotation is possible with the hip flexed but not after partial extension as this stretches the psoas muscle. A confident diagnosis should be followed by emergency drainage. However, if there is any doubt and facilities are available then the abscess can be confirmed by ultrasound or computerised axial tomography. We found the latter particularly informative. A review of the literature has failed to reveal any previous report of a perforated jejunal diverticulum presenting with a psoas abscess.

We would like to thank Mr W A Hanna for permitting us to report on this case.

REFERENCES

1. Krishnamurthy S, Kelly Mary M, Rohrmann CA, Schuffler MD. Jejunal diverticulosis. *Gastroenterology* 1983; **85**: 538-47.
2. MacGregor AB, Hamilton T. Diverticular disease of the jejunum. *J R Coll Surg Edinb* 1970; **15**: 145-50.
3. Finnerty RJ, Vordermark JS, Modarelli RO, Buck AS. Primary psoas abscess: case report and review of literature. *J Urol* 1981; **126 (1)**: 108-9.

The Ulster Medical Journal, Volume 54, No. 1, pp. 80-82, April 1985.

Case report

Clostridium difficile induced colitis occurring during cefotaxime therapy

Stephen T Green, Raymond Mackie, Hugh McMillan, James W Davie

Accepted 18th February 1985.

INTRODUCTION

While not unique to anti-microbial therapy, *Clostridium difficile* induced colitis is most frequently encountered as a complication of treatment with one or more of several antibiotics.[1-4] The condition has been described in association with some members of the cephalosporin group of drugs.[5] Only on a few occasions has cefotaxime been implicated in the past[6-11] and, to date, no cases have been reported in the United Kingdom. We describe a case in which a patient receiving intramuscular cefotaxime developed a severe persistent diarrhoea which proved to be the result of a *C. difficile* infection.

CASE REPORT

A 75-year-old man was admitted in March 1984 (day 1) with urinary retention and increasing immobility. Four years previously he had been diagnosed as having Parkinson's disease and there was a long history of prostatism complicated by recurrent bladder outlet obstruction and urinary tract infections. Apart from a degree of constipation during the months preceding admission, he had had no other symptoms referrable to the gastrointestinal tract. He had no known allergies and he had received amoxycillin and co-trimoxazole 6 months earlier without ill effect. His medication consisted of bromocriptine 2.5mg bd, levodopa 200mg tid and carbidopa 50mg tid. Examination revealed a frail man with signs of Parkinsonism and an abdomen unremarkable except for a tender distended bladder. The rectum was empty and the urine, obtained by catheterisation, was cloudy. Culture of the urine yielded *Proteus mirabilis* and *Pseudomonas aeruginosa,* both sensitive to cefotaxime. This antibiotic was commenced at a dose of 1g bd, given intramuscularly, and no other antibiotics were administered (day 6). On day 11, some faecal staining was noted on the patient's clothing. The patient's urine became sterile and cefotaxime was discontinued on day 14. In the following days the patient developed worsening faecal incontinence and the stools became foul-smelling. Faecal cultures were

Stobhill General Hospital, Glasgow.
Stephen T Green BSc, MB, ChB, Senior House Physician.
Raymond Mackie BSc, MPS, Pharmacist.
Hugh McMillan MRCP, MRCGP, Senior Registrar.
James W Davie FRCP, DRCOG, Consultant Physician.

Correspondence to: Dr Stephen T Green, Senior House Physician, Department of Geriatric Medicine, Stobhill General Hospital, Glasgow G21 3UW.

negative for shigella, salmonella and camphylobacter species while no pathogens were seen on microscopy. Codeine phosphate was commenced. On day 22, *Ps. aeruginosa,* sensitive to cefotaxime, was again cultured from the patient's urine so cefotaxime was recommenced using the same dose and route as previously. By day 29 the urinary problems had cleared and the drug was again withdrawn. The patient's diarrhoea continued to worsen, and microbiological stool analysis still yielded no definite pathogens. On day 33, the patient became toxic with a temperature of 39.2°C and he sustained several rigors. Intramuscular cefotaxime was again started after blood had been drawn for culture. These cultures grew *Ps. aeruginosa* sensitive to cefotaxime. The patient responded well to therapy, and cefotaxime was discontinued on day 46. The problem with diarrhoea had worsened but, by day 46, results obtained by applying gas-liquid chromatography to enrichment cultures of the patient's faeces suggested that the infecting agent was *C. difficile.* Oral vancomycin was commenced at a dose of 500mg tid and, after initial improvement, the dose was reduced to 125mg qid, finally being discontinued on day 57. By this time, the presence of *C. difficile* had been confirmed and *C. difficile* toxin had been detected in the stool at a titre of more than 1/800. The diarrhoea again worsened and by day 75 it became necessary to re-start oral vancomycin 500mg tid for 5 days. By day 102, the diarrhoea had recurred and was once again a serious problem. On this occasion oral metronidazole was prescribed at a dose of 200mg qid. Following this, the problem settled and by day 108 no toxin was detectable in faecal samples. On day 114, metronidazole was discontinued and the patient has since remained free from diarrhoea. Owing to the patient's physical frailty, sigmoidoscopy was not performed during his illness. He was discharged on day 121, his urinary problems having also settled.

DISCUSSION

C. difficile is an anaerobic spore-forming gram-positive organism with the ability to manufacture a potent exotoxin.[1-4] This bacterium is responsible for an acute purulent infection of the colon, and the condition can manifest itself as a pseudomembranous colitis or as diarrhoea and colitis without pseudomembrane formation.[3,4] The resulting clinical picture can be mild and self-limiting or severely debilitating and prolonged, and on occasions the condition can present as a life-threatening toxic megacolon, necessitating emergency colectomy.[3] Both organisms and free toxin are encountered in the stools of affected patients but not in the faeces of patients with other established causes for diarrhoea and, in addition, *C. difficile* is rarely carried by healthy adults.[3] Consequently, if the toxin is demonstrated in the stool, the diagnosis is considered established,[3] and it has been shown that clinical improvement is associated with the disappearance of the toxin from faecal samples.[4] Vancomycin is the established therapy of choice, and metronidazole and bacitracin are also recognised as effective drugs in this clinical setting.[3,4]

Cases of colitis (and, rarely, of pseudomembranous colitis) have been encountered in patients receiving certain cephalosporin antibiotics.[5] Cefotaxime, a third generation cephalosporin, was first isolated in 1978.[12] There have been few reports of *C. difficile* induced colitis occurring with this drug, and these cases have been encountered in West Germany,[6] France[7] and the U.S.A.[8-11] This is the first instance of the complication occurring with cefotaxime in the United Kingdom.

This case emphasises and reinforces the importance of maintaining a high index of clinical suspicion during and following cefotaxime therapy in order to avoid the patient reaching the fulminating stages of a potentially lethal but nevertheless treatable complication.

We are grateful to Dr Angus J Dougall, Consultant Bacteriologist at Stobhill General Hospital, for his advice in the preparation of this paper. We are also indebted to the technical staff of the Microbiology Laboratories at Stobhill and Ruchill Hospitals in Glasgow for performing the bacteriological investigations.

REFERENCES

1. Larson HE, Parry JV, Price AB, Davies DR, Dolby J, Tyrrell DAJ. Undescribed toxin in pseudomembranous colitis. *Br Med J* 1977; **1**: 1246-1248.
2. Larson HE, Price AB. Pseudomembranous colitis: presence of clostridial toxin. *Lancet* 1977; **2**: 1312-1314.
3. Larson HE. Botulism, gas gangrene and clostridial gastrointestinal infections. In: Weatherall DJ, Ledingham JGG, Warrell DA, eds. Oxford textbook of medicine. Oxford: Oxford University Press, 1983; **5**: 230-237.
4. Smith JWG, Smith G. Gas gangrene and other clostridial infections of man and animals. In: Wilson G, Miles A, Parker MT, Smith GR, eds. Topley and Wilson's Principles of bacteriology, virology and immunity, 7th ed, vol. 3. London: Arnold, 1984: 327-344.
5. ABPI data sheet compendium 1984-5. London: Datapharm Publications, 1984: 385-386, 393-394, 739-740, 743-745, 885-888, 1226-1227.
6. Freitag V, Friedrich O, Kopf P-O, Wittman DH. Bacteriological testing of cefotaxime, a new semi-synthetic cephalosporin. *Infection* 1980; **8** (suppl. 4): S397-S400.
7. Gineston J-L, Henry X. Recto-colite pseudomembraneuse après traitement par le céfotaxime. *Nouv Presse Med* 1982; **11**: 1951-1952.
8. Cone LA, Woodard D, Helm NA. Clinical experience in the diagnosis and treatment of infections in the compromised host. *Clin Ther* 1981; **4** (suppl. A): 45-84.
9. Yakabow AL, Wood PD. Clinical experience with cefotaxime: a review. In: Neu HC, ed. New beta-lactam antibiotics: a review from chemistry to clinical efficacy of the new cephalosporins. Philadelphia: College of Physicians of Philadelphia, 1982: 287-328.
10. Karakuis PH, Feczko JM, Goodman LJ, Hanlon DM, Harris AA, Levin S, Trenholme GM. Clinical efficacy of cefotaxime in serious infections. *Antimicrob Agents Chemother* 1982; **21**: 119-124.
11. Mader JT, LeFrock JL, Hyams KC, Molavi A, Reinherz JA. Cefotaxime therapy for patients with osteomyelitis and septic arthritis. *Rev Infect Dis* 1982; **4** (suppl. September): S472-S480.
12. Chabbert YA, Lutz AJ. HR 756, the syn-isomer of a new methoxyiminocephalosporin with unusual antibacterial activity. *Antimicrob Agents Chemother* 1978; **14**: 749-754.

The Ulster Medical Journal, Volume 54, No. 1, pp. 83-88, April 1985.

Book reviews

Multiple choice questions in human physiology. 3rd ed. By I C Roddie and W F M Wallace. (pp 423. £11.50). London: Lloyd-Luke, 1984

This is the third edition of a book of multiple choice questions (MCQs) which was first published in 1971. At that time, MCQs were something of a novelty. They were regarded as light relief from the tedium of long essay questions by the student but often with suspicion by examiners. Now examiners have come to realise the advantages of MCQs in covering a wide range of subjects, and in allowing easy and objective marking. It is the student who is suspicious of the nuances of expression that can tilt the balance between true and false. This suspicion has arisen not because MCQs are a bad method of testing knowledge and understanding, but because of bad MCQs. Sadly, many of those examiners who in the past set MCQs had never had the opportunity themselves to do them in the examination situation.

I do not know whether Professors Roddie and Wallace ever had to sit an MCQ examination. Their thoughtful approach, however, has produced a book of very high quality. It gives comprehensive coverage of pure and applied physiology. By providing explanations of most answers, the book becomes more than just a test of knowledge. However, it is for examination practice that the book will be used most. Here, with its avoidance of ambiguity, the student can learn that a straightforward approach to a clearly written question results in a correct answer. He can decondition himself from the all too prevalent paranoia which results from trying to answer badly set questions.

This book will remain essential reading for those taking undergraduate physiology examinations, and for postgraduates faced with basic science sections in higher examinations. All those who inflict MCQs on others should also refer to the questions in this book, and aim for a similar high standard.

Oxford Textbook of clinical pharmacology and drug therapy. By D G Grahame-Smith and J K Aronson. (pp 843. Cloth £25.00, paper £12.50). London: Oxford University Press, 1984.

This is an entirely new book written by two leading authorities, and so I read it with great interest. It is beautifully presented, very clear and readable, and has no typographical errors that I could see. The book is aimed at senior students, although postgraduates could also read it with benefit. It occupies a place between the short books (such as that by Turner and Richens) and the massive reference tomes. It is very reasonably priced for the large amount of information it contains.

The layout of the book is unusual. Instead of the usual presentation by systems or groups of drugs. the book is divided into four sections. The same drug may therefore appear in different sections, and so careful use of the Index is required. Section I covers the basic science aspect, but frequently refers to specific diseases and drugs. To get the most from this section, the reader should be well acquainted with the conditions discussed — this means that junior students may lose the benefit of the section. The basic logic of drug use is well described, and there is also brief discussion of drug development, trial design and ethics — I thought these were very interesting chapters that could be usefully expanded in future editions. Section II is a short note on practical aspects of prescribing. Section III is the largest, and discusses the treatment of many common disorders, including drug abuse and self-poisoning. This is the core of the book, and it is very good indeed, clearly describing current therapeutic practice. The wide clinical experience of the authors is evident, and this section should effectively silence critics who say that clinical pharmacologists spend their time devising new multicompartmental kinetic models instead of treating human beings! Section IV is a Pharmacopoeia of the common drugs with the key facts about them. Its use is limited by the sometimes illogical grouping together of drugs (e.g. adrenoceptor antagonists, non-steroidal anti-inflammatory drugs) so that recourse to the Index is again necessary.

If I have a criticism of the book, it is that the potential market is limited. It is too advanced for junior students, who will not have heard of the diseases under discussion, and not advanced enough for postgraduate work. The latter would be difficult to achieve without the book growing very large and acquiring many references, but the book could be made to fulfil a useful role as a thorough basic text. The main reason why at the moment it fails in this respect is that basic molecular mechanisms are too little discussed. Receptor mechanisms play such a vital role in all aspects of pharmacology that a whole chapter should be devoted to them. At present, junior students would have important gaps in their knowledge if they relied on this book alone.

If the book could be modified in the next edition, its use would expand to include all students. As it is, I can unhesitatingly recommend it to senior students as the best account that they are likely to read of what you do to ill people.

DPN

The Ulster Medical Journal, Volume 54, No. 1, pp. 83-88, April 1985.

Book reviews

Multiple choice questions in human physiology. 3rd ed. By I C Roddie and W F M Wallace. (pp 423. £11.50). London: Lloyd-Luke, 1984

This is the third edition of a book of multiple choice questions (MCQs) which was first published in 1971. At that time, MCQs were something of a novelty. They were regarded as light relief from the tedium of long essay questions by the student but often with suspicion by examiners. Now examiners have come to realise the advantages of MCQs in covering a wide range of subjects, and in allowing easy and objective marking. It is the student who is suspicious of the nuances of expression that can tilt the balance between true and false. This suspicion has arisen not because MCQs are a bad method of testing knowledge and understanding, but because of bad MCQs. Sadly, many of those examiners who in the past set MCQs had never had the opportunity themselves to do them in the examination situation.

I do not know whether Professors Roddie and Wallace ever had to sit an MCQ examination. Their thoughtful approach, however, has produced a book of very high quality. It gives comprehensive coverage of pure and applied physiology. By providing explanations of most answers, the book becomes more than just a test of knowledge. However, it is for examination practice that the book will be used most. Here, with its avoidance of ambiguity, the student can learn that a straightforward approach to a clearly written question results in a correct answer. He can decondition himself from the all too prevalent paranoia which results from trying to answer badly set questions.

This book will remain essential reading for those taking undergraduate physiology examinations, and for postgraduates faced with basic science sections in higher examinations. All those who inflict MCQs on others should also refer to the questions in this book, and aim for a similar high standard.

Oxford Textbook of clinical pharmacology and drug therapy. By D G Grahame-Smith and J K Aronson. (pp 843. Cloth £25.00, paper £12.50). London: Oxford University Press, 1984.

This is an entirely new book written by two leading authorities, and so I read it with great interest. It is beautifully presented, very clear and readable, and has no typographical errors that I could see. The book is aimed at senior students, although postgraduates could also read it with benefit. It occupies a place between the short books (such as that by Turner and Richens) and the massive reference tomes. It is very reasonably priced for the large amount of information it contains.

The layout of the book is unusual. Instead of the usual presentation by systems or groups of drugs. the book is divided into four sections. The same drug may therefore appear in different sections, and so careful use of the Index is required. Section I covers the basic science aspect, but frequently refers to specific diseases and drugs. To get the most from this section, the reader should be well acquainted with the conditions discussed — this means that junior students may lose the benefit of the section. The basic logic of drug use is well described, and there is also brief discussion of drug development, trial design and ethics — I thought these were very interesting chapters that could be usefully expanded in future editions. Section II is a short note on practical aspects of prescribing. Section III is the largest, and discusses the treatment of many common disorders, including drug abuse and self-poisoning. This is the core of the book, and it is very good indeed, clearly describing current therapeutic practice. The wide clinical experience of the authors is evident, and this section should effectively silence critics who say that clinical pharmacologists spend their time devising new multicompartmental kinetic models instead of treating human beings! Section IV is a Pharmacopoeia of the common drugs with the key facts about them. Its use is limited by the sometimes illogical grouping together of drugs (e.g. adrenoceptor antagonists, non-steroidal anti-inflammatory drugs) so that recourse to the Index is again necessary.

If I have a criticism of the book, it is that the potential market is limited. It is too advanced for junior students, who will not have heard of the diseases under discussion, and not advanced enough for postgraduate work. The latter would be difficult to achieve without the book growing very large and acquiring many references, but the book could be made to fulfil a useful role as a thorough basic text. The main reason why at the moment it fails in this respect is that basic molecular mechanisms are too little discussed. Receptor mechanisms play such a vital role in all aspects of pharmacology that a whole chapter should be devoted to them. At present, junior students would have important gaps in their knowledge if they relied on this book alone.

If the book could be modified in the next edition, its use would expand to include all students. As it is, I can unhesitatingly recommend it to senior students as the best account that they are likely to read of what you do to ill people.

DPN

Annual report: The Medico-Social Research Board, Dublin. (1984, pp 101). Geoffrey Dean, Director, 73 Lower Baggot Street, Dublin 2.

The Republic of Ireland's Medico-Social Research Board spent over half of its £800,000 budget on setting up a hospital inpatient enquiry scheme because of a need "to study the cost effectiveness of health care". It emerged that in Ireland the number of hospital beds used daily per hundred thousand population was ten per cent higher than in Scotland mainly because Irish patients stayed in hospital longer although the number of admissions per hundred thousand population was also slightly higher. Presumably the next step is to determine whether this difference is due to worse disease or less effective hospital care, but the Board has carefully avoided a definition of "cost effectiveness in health care" which is likely to become a major pre-occupation of hospital doctors throughout the British Isles in the next ten years.

Standardised Mortality Ratios for ischaemic heart disease and common tumours in all the EEC countries showed that the greatest deviation from the mean was for ischaemic heart disease in Irish women, 2.3 times the average EEC rate, although Irish men and English men and women all had a similar rate of 1.6 times the EEC average. The Board suggested that the increased cigarette consumption in Ireland might contribute to this finding but felt that Ireland's "strict ethical code" probably explained why Irish women had the lowest incidence of carcinoma of cervix and uterus in Europe. Whilst carcinoma of rectum amongst workers in a brewery in Dublin seems to be most common in those who drink the most beer, the overall increase in carcinoma of colon and rectum in Ireland was thought to be due to low dietary fibre.

Despite the stage image, Ireland still has one of the lowest per capita alcohol consumptions in Europe (equal to that in the UK, but half of that in France and West Germany). The Board were nevertheless reluctant to believe that deaths from alcoholic cirrhosis were as uncommon as Irish doctors had indicated on death certificates (only half the UK rate and only a fifteenth of the rate in Italy and France). It is hoped to obtain more accurate figures for cirrhosis deaths from hospital postmortem ststistics but the possibilities remain that Irishmen may be genetically protected against cirrhosis, or that fewer of the drinkers may consume enough to cause cirrhosis.

MEC

A practice of anaesthesia. 5th ed. Edited by H C Churchill-Davidson. (pp 1261. £50.00). London: Lloyd-Luke, 1984.

It is now 25 years since Wylie and Churchill-Davidson's *Practice of anaesthesia* first appeared. The number of contributing authors has increased steadily and of course Dr Dereck Wylie has dropped out. However, the volume has remained constant to its original aim, which is to explain anaesthetic principles for different conditions on a basis of anatomy, physiology and pharmacology. This has inevitably necessitated expansion and the book now runs to over 1,200 pages of double columns but it is still well printed on high quality paper and the editors have maintained the easy flowing style which has always evidenced itself, to this reviewer at least.

The number of changes since the fourth edition of 1978 is clearly great, with references of the 1980s in every chapter. The newer agents such as isoflurane, midazolam, disoprofol, vecuronium and atracurium are covered better than one could expect, knowing the usual delays in preparing books for publication.

Two new chapters have been added, in intensive therapy and the pain clinic. The former is comprehensive and treats in detail problems ranging from DIC to tetanus and physiotherapy to parenteral nutrition — as indeed one would expect from the prominent role St Thomas's Hospital has had in the development of the ITU. The management of chronic pain is also given detailed treatment covering a wide range of blocks as well as management with analgesics and tranquillisers.

In spite of its size it clearly has deficiencies. For instance, the chapter on the endocrine glands, although very up-to-date, gets only 11 pages. Surely diabetes is at least as important to the anaesthetist as myocardial infarction, and endocrine physiology should receive more detailed treatment if one is to understand nutrition and the metabolic response to trauma. However, it is easy to suggest expansion and hard to find material to throw out. Even the 22 pages on hyperbaric physiology and medicine may be of use to readers occasionally and certainly stimulates those whose physics is rusting to think again of the principles.

All in all it is a book to be recommended heartily. It should be bought during one's first months of anaesthesia and read and re-read throughout one's training. There is material for all parts of the FFARCS but perhaps even more for the anaesthetist broadening his mind during his higher professional training and beyond. At £50 and in view of the quality of its production, it is excellent value.

RSJC

Annual report: The Medico-Social Research Board, Dublin. (1984, pp 101). Geoffrey Dean, Director, 73 Lower Baggot Street, Dublin 2.

The Republic of Ireland's Medico-Social Research Board spent over half of its £800,000 budget on setting up a hospital inpatient enquiry scheme because of a need "to study the cost effectiveness of health care". It emerged that in Ireland the number of hospital beds used daily per hundred thousand population was ten per cent higher than in Scotland mainly because Irish patients stayed in hospital longer although the number of admissions per hundred thousand population was also slightly higher. Presumably the next step is to determine whether this difference is due to worse disease or less effective hospital care, but the Board has carefully avoided a definition of "cost effectiveness in health care" which is likely to become a major pre-occupation of hospital doctors throughout the British Isles in the next ten years.

Standardised Mortality Ratios for ischaemic heart disease and common tumours in all the EEC countries showed that the greatest deviation from the mean was for ischaemic heart disease in Irish women, 2.3 times the average EEC rate, although Irish men and English men and women all had a similar rate of 1.6 times the EEC average. The Board suggested that the increased cigarette consumption in Ireland might contribute to this finding but felt that Ireland's "strict ethical code" probably explained why Irish women had the lowest incidence of carcinoma of cervix and uterus in Europe. Whilst carcinoma of rectum amongst workers in a brewery in Dublin seems to be most common in those who drink the most beer, the overall increase in carcinoma of colon and rectum in Ireland was thought to be due to low dietary fibre.

Despite the stage image, Ireland still has one of the lowest per capita alcohol consumptions in Europe (equal to that in the UK, but half of that in France and West Germany). The Board were nevertheless reluctant to believe that deaths from alcoholic cirrhosis were as uncommon as Irish doctors had indicated on death certificates (only half the UK rate and only a fifteenth of the rate in Italy and France). It is hoped to obtain more accurate figures for cirrhosis deaths from hospital postmortem ststistics but the possibilities remain that Irishmen may be genetically protected against cirrhosis, or that fewer of the drinkers may consume enough to cause cirrhosis.

MEC

A practice of anaesthesia. 5th ed. Edited by H C Churchill-Davidson. (pp 1261. £50.00). London: Lloyd-Luke, 1984.

It is now 25 years since Wylie and Churchill-Davidson's *Practice of anaesthesia* first appeared. The number of contributing authors has increased steadily and of course Dr Dereck Wylie has dropped out. However, the volume has remained constant to its original aim, which is to explain anaesthetic principles for different conditions on a basis of anatomy, physiology and pharmacology. This has inevitably necessitated expansion and the book now runs to over 1,200 pages of double columns but it is still well printed on high quality paper and the editors have maintained the easy flowing style which has always evidenced itself, to this reviewer at least.

The number of changes since the fourth edition of 1978 is clearly great, with references of the 1980s in every chapter. The newer agents such as isoflurane, midazolam, disoprofol, vecuronium and atracurium are covered better than one could expect, knowing the usual delays in preparing books for publication.

Two new chapters have been added, in intensive therapy and the pain clinic. The former is comprehensive and treats in detail problems ranging from DIC to tetanus and physiotherapy to parenteral nutrition — as indeed one would expect from the prominent role St Thomas's Hospital has had in the development of the ITU. The management of chronic pain is also given detailed treatment covering a wide range of blocks as well as management with analgesics and tranquillisers.

In spite of its size it clearly has deficiencies. For instance, the chapter on the endocrine glands, although very up-to-date, gets only 11 pages. Surely diabetes is at least as important to the anaesthetist as myocardial infarction, and endocrine physiology should receive more detailed treatment if one is to understand nutrition and the metabolic response to trauma. However, it is easy to suggest expansion and hard to find material to throw out. Even the 22 pages on hyperbaric physiology and medicine may be of use to readers occasionally and certainly stimulates those whose physics is rusting to think again of the principles.

All in all it is a book to be recommended heartily. It should be bought during one's first months of anaesthesia and read and re-read throughout one's training. There is material for all parts of the FFARCS but perhaps even more for the anaesthetist broadening his mind during his higher professional training and beyond. At £50 and in view of the quality of its production, it is excellent value.

RSJC

Malabsorption and nutritional support. Edited by Marvin H Sleisenger. (pp 223-613. Illustrated). London: Saunders, 1983. (Clinics in gastroenterology; vol 12, no 2).

This book comprises a widespread and useful series of reviews on many aspects of malabsorption. The chapter authors are largely from the USA with a sprinkling of gastroenterologists from other parts of the world. The individual chapters are well-referenced. Many individual topics are discussed in detail, including intestinal immunity and malabsorption, and the relationship of alcohol, nutrition and malabsorption. The chapter dealing with breath tests will be useful as a reference to this increasingly helpful investigation of malabsorption. The production of the book is well up to the usual standard expected of this series.

DRH

Chronic pain: management principles. Edited by Steven F Brena and Stanley L Chapman. (pp 240. £12.50). London: Saunders, 1985. (Clinics in anaesthesiology; vol 3, no 1).

The medical profession, and indeed those engaged in related disciplines, have become increasingly aware in recent years that persistent pain is, in itself, a significant clinical problem — a problem, moreover, which justifies, and occasionally rewards, a serious approach to its symptomatic management. One product of this current interest in chronic pain has been a gradual realisation of its complexity, in turn promoting extensive research and an impressive proliferation of the literature. The present publication incorporates the views of fifteen authors, and, although Great Britain and Australia have distinguished representatives, depends heavily on North American experience. Dr John Bonica, to many the initiator of the pain clinic movement, reviews its history and evolution.

It is, perhaps, salutary to note that Paracelsus (AD 1490-1540) advocated opium, electrotherapy, massage and exercise. Somewhat updated, such methods form a significant part of our armamentarium today! The following chapter, by Dr Duggan, gives a concise yet very adequate summary of physiological principles. The remainder of the book, with the exception of a sensible review of basic nerve blocks by Dr Parris is confined to what might be termed non-invasive aspects such as psychological, social and organisational considerations, drug therapy and hyperstimulation analgesia.

This book deals well with a limited number of topics, but could certainly not be considered a comprehensive review of the subject. Those already involved in chronic pain work may find that reading it serves to broaden their perspectives, and those contemplating the establishment of a pain clinic should find some sections particularly thought-provoking. It may have less to offer the general reader, although it could be a worthwhile addition to a departmental library, in that pain is universal and this quite readable little book might stimulate interest in its more adequate management. At £12.50 it is not, by modern standards, expensive.

WBL

Radiological diagnosis of fractures. By D B L Finlay and M J Allen. (pp 256. Illustrated. £15.00). London: Saunders, 1984.

This rather unusual book uses line drawings of radiographs to illustrate fractures. It deals with the upper limb, lower limb, chest, spine and skull. Some sections are better than others. Whilst I feel that it would be a useful reference book in an accident and emergency department I would not recommend it as an essential book for each and every medical student or house officer.

JT

Wound healing for surgeons. Edited by Timothy Bucknall and Harold Ellis. (pp 344. £19.75) London: Saunders, 1984.

This is a fascinating book, covering many aspects of wound healing, and written in the easy-to-read, commonsense style we have come to expect of Professor Harold Ellis. The title might not tempt many a general surgeon, but, as the authors point out, this is a book 'by practising surgeons, for practising surgeons'.

All aspects of healing are touched on, not just wound healing. From basic principles, the authors move systematically through the abdominal wall, the gastrointestinal system, the hepatobiliary apparatus and the urinary tract. Later chapters deal with blood vessels, nerves, bone, joints and tendons.

The book is well edited, the chapters consistent and the references up-to-date and relevant. Not all the views stated are universally accepted, but they are backed with sound scientific argument. For example, the authors make a strong case for avoiding an intestinal anastomosis in the chest, because of the mortality associated with leakage.

At almost £20 this is not a cheap addition to your personal library, but surgeons in training will find it a worthwhile investment. In particular, it would make excellent background reading for anyone about to embark on clinical or laboratory research in this field.

STI

Malabsorption and nutritional support. Edited by Marvin H Sleisenger. (pp 223-613. Illustrated). London: Saunders, 1983. (Clinics in gastroenterology; vol 12, no 2).

This book comprises a widespread and useful series of reviews on many aspects of malabsorption. The chapter authors are largely from the USA with a sprinkling of gastroenterologists from other parts of the world. The individual chapters are well-referenced. Many individual topics are discussed in detail, including intestinal immunity and malabsorption, and the relationship of alcohol, nutrition and malabsorption. The chapter dealing with breath tests will be useful as a reference to this increasingly helpful investigation of malabsorption. The production of the book is well up to the usual standard expected of this series.

DRH

Chronic pain: management principles. Edited by Steven F Brena and Stanley L Chapman. (pp 240. £12.50). London: Saunders, 1985. (Clinics in anaesthesiology; vol 3, no 1).

The medical profession, and indeed those engaged in related disciplines, have become increasingly aware in recent years that persistent pain is, in itself, a significant clinical problem — a problem, moreover, which justifies, and occasionally rewards, a serious approach to its symptomatic management. One product of this current interest in chronic pain has been a gradual realisation of its complexity, in turn promoting extensive research and an impressive proliferation of the literature. The present publication incorporates the views of fifteen authors, and, although Great Britain and Australia have distinguished representatives, depends heavily on North American experience. Dr John Bonica, to many the initiator of the pain clinic movement, reviews its history and evolution.

It is, perhaps, salutary to note that Paracelsus (AD 1490-1540) advocated opium, electrotherapy, massage and exercise. Somewhat updated, such methods form a significant part of our armamentarium today! The following chapter, by Dr Duggan, gives a concise yet very adequate summary of physiological principles. The remainder of the book, with the exception of a sensible review of basic nerve blocks by Dr Parris is confined to what might be termed non-invasive aspects such as psychological, social and organisational considerations, drug therapy and hyperstimulation analgesia.

This book deals well with a limited number of topics, but could certainly not be considered a comprehensive review of the subject. Those already involved in chronic pain work may find that reading it serves to broaden their perspectives, and those contemplating the establishment of a pain clinic should find some sections particularly thought-provoking. It may have less to offer the general reader, although it could be a worthwhile addition to a departmental library, in that pain is universal and this quite readable little book might stimulate interest in its more adequate management. At £12.50 it is not, by modern standards, expensive.

WBL

Radiological diagnosis of fractures. By D B L Finlay and M J Allen. (pp 256. Illustrated. £15.00). London: Saunders, 1984.

This rather unusual book uses line drawings of radiographs to illustrate fractures. It deals with the upper limb, lower limb, chest, spine and skull. Some sections are better than others. Whilst I feel that it would be a useful reference book in an accident and emergency department I would not recommend it as an essential book for each and every medical student or house officer.

JT

Wound healing for surgeons. Edited by Timothy Bucknall and Harold Ellis. (pp 344. £19.75) London: Saunders, 1984.

This is a fascinating book, covering many aspects of wound healing, and written in the easy-to-read, commonsense style we have come to expect of Professor Harold Ellis. The title might not tempt many a general surgeon, but, as the authors point out, this is a book 'by practising surgeons, for practising surgeons'.

All aspects of healing are touched on, not just wound healing. From basic principles, the authors move systematically through the abdominal wall, the gastrointestinal system, the hepatobiliary apparatus and the urinary tract. Later chapters deal with blood vessels, nerves, bone, joints and tendons.

The book is well edited, the chapters consistent and the references up-to-date and relevant. Not all the views stated are universally accepted, but they are backed with sound scientific argument. For example, the authors make a strong case for avoiding an intestinal anastomosis in the chest, because of the mortality associated with leakage.

At almost £20 this is not a cheap addition to your personal library, but surgeons in training will find it a worthwhile investment. In particular, it would make excellent background reading for anyone about to embark on clinical or laboratory research in this field.

STI

Malabsorption and nutritional support. Edited by Marvin H Sleisenger. (pp 223-613. Illustrated). London: Saunders, 1983. (Clinics in gastroenterology; vol 12, no 2).

This book comprises a widespread and useful series of reviews on many aspects of malabsorption. The chapter authors are largely from the USA with a sprinkling of gastroenterologists from other parts of the world. The individual chapters are well-referenced. Many individual topics are discussed in detail, including intestinal immunity and malabsorption, and the relationship of alcohol, nutrition and malabsorption. The chapter dealing with breath tests will be useful as a reference to this increasingly helpful investigation of malabsorption. The production of the book is well up to the usual standard expected of this series.

DRH

Chronic pain: management principles. Edited by Steven F Brena and Stanley L Chapman. (pp 240. £12.50). London: Saunders, 1985. (Clinics in anaesthesiology; vol 3, no 1).

The medical profession, and indeed those engaged in related disciplines, have become increasingly aware in recent years that persistent pain is, in itself, a significant clinical problem — a problem, moreover, which justifies, and occasionally rewards, a serious approach to its symptomatic management. One product of this current interest in chronic pain has been a gradual realisation of its complexity, in turn promoting extensive research and an impressive proliferation of the literature. The present publication incorporates the views of fifteen authors, and, although Great Britain and Australia have distinguished representatives, depends heavily on North American experience. Dr John Bonica, to many the initiator of the pain clinic movement, reviews its history and evolution.

It is, perhaps, salutary to note that Paracelsus (AD 1490-1540) advocated opium, electrotherapy, massage and exercise. Somewhat updated, such methods form a significant part of our armamentarium today! The following chapter, by Dr Duggan, gives a concise yet very adequate summary of physiological principles. The remainder of the book, with the exception of a sensible review of basic nerve blocks by Dr Parris is confined to what might be termed non-invasive aspects such as psychological, social and organisational considerations, drug therapy and hyperstimulation analgesia.

This book deals well with a limited number of topics, but could certainly not be considered a comprehensive review of the subject. Those already involved in chronic pain work may find that reading it serves to broaden their perspectives, and those contemplating the establishment of a pain clinic should find some sections particularly thought-provoking. It may have less to offer the general reader, although it could be a worthwhile addition to a departmental library, in that pain is universal and this quite readable little book might stimulate interest in its more adequate management. At £12.50 it is not, by modern standards, expensive.

WBL

Radiological diagnosis of fractures. By D B L Finlay and M J Allen. (pp 256. Illustrated. £15.00). London: Saunders, 1984.

This rather unusual book uses line drawings of radiographs to illustrate fractures. It deals with the upper limb, lower limb, chest, spine and skull. Some sections are better than others. Whilst I feel that it would be a useful reference book in an accident and emergency department I would not recommend it as an essential book for each and every medical student or house officer.

JT

Wound healing for surgeons. Edited by Timothy Bucknall and Harold Ellis. (pp 344. £19.75) London: Saunders, 1984.

This is a fascinating book, covering many aspects of wound healing, and written in the easy-to-read, commonsense style we have come to expect of Professor Harold Ellis. The title might not tempt many a general surgeon, but, as the authors point out, this is a book 'by practising surgeons, for practising surgeons'.

All aspects of healing are touched on, not just wound healing. From basic principles, the authors move systematically through the abdominal wall, the gastrointestinal system, the hepatobiliary apparatus and the urinary tract. Later chapters deal with blood vessels, nerves, bone, joints and tendons.

The book is well edited, the chapters consistent and the references up-to-date and relevant. Not all the views stated are universally accepted, but they are backed with sound scientific argument. For example, the authors make a strong case for avoiding an intestinal anastomosis in the chest, because of the mortality associated with leakage.

At almost £20 this is not a cheap addition to your personal library, but surgeons in training will find it a worthwhile investment. In particular, it would make excellent background reading for anyone about to embark on clinical or laboratory research in this field.

STI

Malabsorption and nutritional support. Edited by Marvin H Sleisenger. (pp 223-613. Illustrated). London: Saunders, 1983. (Clinics in gastroenterology; vol 12, no 2).

This book comprises a widespread and useful series of reviews on many aspects of malabsorption. The chapter authors are largely from the USA with a sprinkling of gastroenterologists from other parts of the world. The individual chapters are well-referenced. Many individual topics are discussed in detail, including intestinal immunity and malabsorption, and the relationship of alcohol, nutrition and malabsorption. The chapter dealing with breath tests will be useful as a reference to this increasingly helpful investigation of malabsorption. The production of the book is well up to the usual standard expected of this series.

DRH

Chronic pain: management principles. Edited by Steven F Brena and Stanley L Chapman. (pp 240. £12.50). London: Saunders, 1985. (Clinics in anaesthesiology; vol 3, no 1).

The medical profession, and indeed those engaged in related disciplines, have become increasingly aware in recent years that persistent pain is, in itself, a significant clinical problem — a problem, moreover, which justifies, and occasionally rewards, a serious approach to its symptomatic management. One product of this current interest in chronic pain has been a gradual realisation of its complexity, in turn promoting extensive research and an impressive proliferation of the literature. The present publication incorporates the views of fifteen authors, and, although Great Britain and Australia have distinguished representatives, depends heavily on North American experience. Dr John Bonica, to many the initiator of the pain clinic movement, reviews its history and evolution.

It is, perhaps, salutary to note that Paracelsus (AD 1490-1540) advocated opium, electrotherapy, massage and exercise. Somewhat updated, such methods form a significant part of our armamentarium today! The following chapter, by Dr Duggan, gives a concise yet very adequate summary of physiological principles. The remainder of the book, with the exception of a sensible review of basic nerve blocks by Dr Parris is confined to what might be termed non-invasive aspects such as psychological, social and organisational considerations, drug therapy and hyperstimulation analgesia.

This book deals well with a limited number of topics, but could certainly not be considered a comprehensive review of the subject. Those already involved in chronic pain work may find that reading it serves to broaden their perspectives, and those contemplating the establishment of a pain clinic should find some sections particularly thought-provoking. It may have less to offer the general reader, although it could be a worthwhile addition to a departmental library, in that pain is universal and this quite readable little book might stimulate interest in its more adequate management. At £12.50 it is not, by modern standards, expensive.

WBL

Radiological diagnosis of fractures. By D B L Finlay and M J Allen. (pp 256. Illustrated. £15.00). London: Saunders, 1984.

This rather unusual book uses line drawings of radiographs to illustrate fractures. It deals with the upper limb, lower limb, chest, spine and skull. Some sections are better than others. Whilst I feel that it would be a useful reference book in an accident and emergency department I would not recommend it as an essential book for each and every medical student or house officer.

JT

Wound healing for surgeons. Edited by Timothy Bucknall and Harold Ellis. (pp 344. £19.75) London: Saunders, 1984.

This is a fascinating book, covering many aspects of wound healing, and written in the easy-to-read, commonsense style we have come to expect of Professor Harold Ellis. The title might not tempt many a general surgeon, but, as the authors point out, this is a book 'by practising surgeons, for practising surgeons'.

All aspects of healing are touched on, not just wound healing. From basic principles, the authors move systematically through the abdominal wall, the gastrointestinal system, the hepatobiliary apparatus and the urinary tract. Later chapters deal with blood vessels, nerves, bone, joints and tendons.

The book is well edited, the chapters consistent and the references up-to-date and relevant. Not all the views stated are universally accepted, but they are backed with sound scientific argument. For example, the authors make a strong case for avoiding an intestinal anastomosis in the chest, because of the mortality associated with leakage.

At almost £20 this is not a cheap addition to your personal library, but surgeons in training will find it a worthwhile investment. In particular, it would make excellent background reading for anyone about to embark on clinical or laboratory research in this field.

STI

Will Pickles of Wensleydale: the life of a country doctor. By John Pemberton. 2nd ed. (pp 224. £10.50). London: Royal College of General Practitioners, 1972, reprinted 1984.

This book by Professor John Pemberton, which has been out of print for several years, has recently been reprinted by the Royal College of General Practitioners. Moreover, publication has been arranged to coincide with the similar re-publication of Pickles' *Epidemiology in country practice,* so that two classic books will become available again together.

This biography should be compulsory reading for all aspiring general practitioners. It describes Will Pickles' childhood, his student days, the difficulties he had getting into and becoming established in practice in Wensleydale in Yorkshire. We are told how he and his partner went up dale and down dale on alternate days by horse and later by motorcycle and car visiting his patients, how he developed a great love for his patients and wouldn't accept any criticism of them, yet employed a permanent part-time debt collector who was also the local part-time sanitary inspector. The eventual good life of the country doctor — of shooting parties on the moor, golf at Leyburn and Hawes and fishing in the Ure is recalled and how he would arrange a shoot when he invited a consultant down from Leeds on a domiciliary visit. Above all, the book describes Will's observational research into epidemic illnesses and demonstrates how research can be carried out in general practice. It recounts the international acclaim he received on his lecture tours of the five continents. We are told how Will Pickles, unlike the majority of his colleagues, welcomed the National Health Service, advocated health centres and attended refresher courses for one month each year.

Professor John Pemberton, during the period he was lecturer in Social Medicine at Sheffield University before coming to Belfast, did many locums in Asygarth practice when Will's partners were away on holidays. He developed a great friendship and admiration for Will, a man twenty-seven years his senior. The book is written in a pleasing easy-to-read style and contains many of the author's views on general practice. No one better than John Pemberton could have been chosen to write this biography of one of the best known and lovable general practitioners of all time. JOW

Essential endocrinology. 2nd ed. Edited by John Laycock and Peter Wise. (pp 371. £9.50) London: Oxford University Press, 1983.

The first edition of this book appeared in 1978 and was described as a book for both pre-clinical and clinical medical students. It was written by two clinical physiologists at Charing Cross Hospital Medical School. Following Professor Lee's unfortunate early death, Dr Peter Wise, Consultant Clinical Endocrinologist at Charing Cross Hospital, has taken on joint editorship of the book for its second edition.

The book is thicker (371 compared with 224 pages) and more expensive (£9.50 as against £4.75), but there are benefits. The size of print is enlarged and the layout is much improved. The opportunity has been taken to put the picture demonstrating galactorrhoea the right way up — this is mentioned merely to point out the generally high quality of the clinical illustrations.

Specialists in endocrinology will not find this book sufficiently detailed to answer their clinical questions. However, it is not written for specialists or even for specialists in training but for clinical and pre-clinical medical students. To that end it must be judged successful. There may be some debate as to who should write such a book on a clinical topic for clinical students and I am sure that the teaching experience in a physiology department will lead to a useful ability to simplify complicated and necessarily incomplete concepts of endocrine disorders, and to encourage the interested student to progress deeper into the medical literature. Some students will prefer to buy a large textbook at the beginning of their career and work their way through it, and such large textbooks now exist summarising present concepts of British medicine. Other students prefer to progress with a series of small books on separate topics and those students will find this an excellent guide throughout their clinical career to the science and practice of endocrinology.

However, if the second edition of this book to be taken as an exercise in the progression of the art of teaching, one would prefer to see it becoming smaller, cheaper and published more frequently. Notwithstanding that criticism, the authors are to be congratulated in producing a useful, up-to-date, well-illustrated and fully documented summary of endocrinology. DRH

Will Pickles of Wensleydale: the life of a country doctor. By John Pemberton. 2nd ed. (pp 224. £10.50). London: Royal College of General Practitioners, 1972, reprinted 1984.

This book by Professor John Pemberton, which has been out of print for several years, has recently been reprinted by the Royal College of General Practitioners. Moreover, publication has been arranged to coincide with the similar re-publication of Pickles' *Epidemiology in country practice,* so that two classic books will become available again together.

This biography should be compulsory reading for all aspiring general practitioners. It describes Will Pickles' childhood, his student days, the difficulties he had getting into and becoming established in practice in Wensleydale in Yorkshire. We are told how he and his partner went up dale and down dale on alternate days by horse and later by motorcycle and car visiting his patients, how he developed a great love for his patients and wouldn't accept any criticism of them, yet employed a permanent part-time debt collector who was also the local part-time sanitary inspector. The eventual good life of the country doctor — of shooting parties on the moor, golf at Leyburn and Hawes and fishing in the Ure is recalled and how he would arrange a shoot when he invited a consultant down from Leeds on a domiciliary visit. Above all, the book describes Will's observational research into epidemic illnesses and demonstrates how research can be carried out in general practice. It recounts the international acclaim he received on his lecture tours of the five continents. We are told how Will Pickles, unlike the majority of his colleagues, welcomed the National Health Service, advocated health centres and attended refresher courses for one month each year.

Professor John Pemberton, during the period he was lecturer in Social Medicine at Sheffield University before coming to Belfast, did many locums in Asygarth practice when Will's partners were away on holidays. He developed a great friendship and admiration for Will, a man twenty-seven years his senior. The book is written in a pleasing easy-to-read style and contains many of the author's views on general practice. No one better than John Pemberton could have been chosen to write this biography of one of the best known and lovable general practitioners of all time. JOW

Essential endocrinology. 2nd ed. Edited by John Laycock and Peter Wise. (pp 371. £9.50) London: Oxford University Press, 1983.

The first edition of this book appeared in 1978 and was described as a book for both pre-clinical and clinical medical students. It was written by two clinical physiologists at Charing Cross Hospital Medical School. Following Professor Lee's unfortunate early death, Dr Peter Wise, Consultant Clinical Endocrinologist at Charing Cross Hospital, has taken on joint editorship of the book for its second edition.

The book is thicker (371 compared with 224 pages) and more expensive (£9.50 as against £4.75), but there are benefits. The size of print is enlarged and the layout is much improved. The opportunity has been taken to put the picture demonstrating galactorrhoea the right way up — this is mentioned merely to point out the generally high quality of the clinical illustrations.

Specialists in endocrinology will not find this book sufficiently detailed to answer their clinical questions. However, it is not written for specialists or even for specialists in training but for clinical and pre-clinical medical students. To that end it must be judged successful. There may be some debate as to who should write such a book on a clinical topic for clinical students and I am sure that the teaching experience in a physiology department will lead to a useful ability to simplify complicated and necessarily incomplete concepts of endocrine disorders, and to encourage the interested student to progress deeper into the medical literature. Some students will prefer to buy a large textbook at the beginning of their career and work their way through it, and such large textbooks now exist summarising present concepts of British medicine. Other students prefer to progress with a series of small books on separate topics and those students will find this an excellent guide throughout their clinical career to the science and practice of endocrinology.

However, if the second edition of this book to be taken as an exercise in the progression of the art of teaching, one would prefer to see it becoming smaller, cheaper and published more frequently. Notwithstanding that criticism, the authors are to be congratulated in producing a useful, up-to-date, well-illustrated and fully documented summary of endocrinology. DRH

Pathological basis of renal disease. 2nd ed. By M S Dunnill. (pp 568. Illustrated. £29.50). London: Baillière Tindall, 1984.

This is the second edition of a useful book originally published in 1976. It has grown by four chapters and almost a hundred pages though much of the text is unchanged from the previous edition. Its contents range widely but, as might be anticipated, several chapters are concerned with the detailed pathology of glomerulonephritis. The main clinical features of glomerular disease are presented, and the recently introduced WHO histological classification is given with some valuable accompanying explanation.

A chapter is devoted to renal involvement in liver disease, a subject of increasing interest often omitted in other texts. There are chapters dealing with topics such as the kidney in pregnancy, amyloid disease, tropical renal disease and renal complications in narcotic addicts. A section is now included on renal tumours — a defect in the previous edition. Separate chapters have been added on stone disease, tubular disorders and the various types of cystic lesion found in the kidney. It is a pity that the section on renal transplantation was not modernised to include some of the interesting work which has followed the development of monoclonal antibodies as well as the controversial discussion on the value of aspiration cytology.

The good quality illustrations are of light and immunohistological preparations with many electron micrographs — all integral parts of modern renal pathological analysis.

This readable book is an asset to any renal pathologist and to a pathology department library where it would be valuable to general pathologists and those in training. It should also be consulted by clinicians who are interested in the pathological aspects of the renal diseases they are seeking to investigate.

CMH

Inflammatory disorders of muscle. Edited by Barbara M Ansell. (pp 216. Illustrated. £12.50). London: Saunders, 1984. (Clinics in rheumatic diseases, vol 10, no 1).

This volume of this now well-established review series in rheumatology is a notable contribution to the better understanding of non-suppurative muscle disease. As usual, in this series, the contributors are well-known international authorities who approach the clinical, immunological, virological, genetic and diagnostic features in a critical and stimulating fashion, thus contributing towards a better understanding of pathogenesis. The review of treatment and management is practical and critical. The illustrations are, without exception, beautifully reproduced.

This is an outstanding review of the subject and will have a wide appeal, including as it does an up-to-date bibliography of major references. It is a book to be studied rather than read. The critical editorship of Dr Ansell is evident in the presentation and style which, for a multi-author publication, is remarkably uniform throughout. I can fully recommend this number of the *Clinics* as excellent value at the price.

MWJB

Ovulation and its disorders. Edited by W Thompson, R F Harrison and J Bonnar. (pp 185. £29.95). Lancaster: MTP, 1984. (Studies in fertility and sterility).

This book contains 33 abstracts of papers presented at the XIth World Congress of Fertility and Sterility in Dublin in 1983.

There are sections devoted to monitoring of ovulation and evaluating the function of the corpus luteum with emphasis on ultrasonography to study follicle growth and the use of serum and salivary progesterone to assess the luteal phase.

The sections on prolactin secretion and treatment of ovulation dysfunction contain little of value to the undergraduate or postgraduate student. Indeed, this book will be of interest only to those involved in active research into infertility and gynaecological endocrinology.

Its major drawback is that the papers are presented in abstract form only and thus such details that may normally be included in 'Patients and materials' are lacking. Also, as with most large conferences today because of financial constraints the only way to attract a sufficient number of participants is to accept — without editorial review — all submitted abstracts, the quality of work reported in this expensive book is inevitably variable.

AT

Pathological basis of renal disease. 2nd ed. By M S Dunnill. (pp 568. Illustrated. £29.50). London: Baillière Tindall, 1984.

This is the second edition of a useful book originally published in 1976. It has grown by four chapters and almost a hundred pages though much of the text is unchanged from the previous edition. Its contents range widely but, as might be anticipated, several chapters are concerned with the detailed pathology of glomerulonephritis. The main clinical features of glomerular disease are presented, and the recently introduced WHO histological classification is given with some valuable accompanying explanation.

A chapter is devoted to renal involvement in liver disease, a subject of increasing interest often omitted in other texts. There are chapters dealing with topics such as the kidney in pregnancy, amyloid disease, tropical renal disease and renal complications in narcotic addicts. A section is now included on renal tumours — a defect in the previous edition. Separate chapters have been added on stone disease, tubular disorders and the various types of cystic lesion found in the kidney. It is a pity that the section on renal transplantation was not modernised to include some of the interesting work which has followed the development of monoclonal antibodies as well as the controversial discussion on the value of aspiration cytology.

The good quality illustrations are of light and immunohistological preparations with many electron micrographs — all integral parts of modern renal pathological analysis.

This readable book is an asset to any renal pathologist and to a pathology department library where it would be valuable to general pathologists and those in training. It should also be consulted by clinicians who are interested in the pathological aspects of the renal diseases they are seeking to investigate.

CMH

Inflammatory disorders of muscle. Edited by Barbara M Ansell. (pp 216. Illustrated. £12.50). London: Saunders, 1984. (Clinics in rheumatic diseases, vol 10, no 1).

This volume of this now well-established review series in rheumatology is a notable contribution to the better understanding of non-suppurative muscle disease. As usual, in this series, the contributors are well-known international authorities who approach the clinical, immunological, virological, genetic and diagnostic features in a critical and stimulating fashion, thus contributing towards a better understanding of pathogenesis. The review of treatment and management is practical and critical. The illustrations are, without exception, beautifully reproduced.

This is an outstanding review of the subject and will have a wide appeal, including as it does an up-to-date bibliography of major references. It is a book to be studied rather than read. The critical editorship of Dr Ansell is evident in the presentation and style which, for a multi-author publication, is remarkably uniform throughout. I can fully recommend this number of the *Clinics* as excellent value at the price.

MWJB

Ovulation and its disorders. Edited by W Thompson, R F Harrison and J Bonnar. (pp 185. £29.95). Lancaster: MTP, 1984. (Studies in fertility and sterility).

This book contains 33 abstracts of papers presented at the XIth World Congress of Fertility and Sterility in Dublin in 1983.

There are sections devoted to monitoring of ovulation and evaluating the function of the corpus luteum with emphasis on ultrasonography to study follicle growth and the use of serum and salivary progesterone to assess the luteal phase.

The sections on prolactin secretion and treatment of ovulation dysfunction contain little of value to the undergraduate or postgraduate student. Indeed, this book will be of interest only to those involved in active research into infertility and gynaecological endocrinology.

Its major drawback is that the papers are presented in abstract form only and thus such details that may normally be included in 'Patients and materials' are lacking. Also, as with most large conferences today because of financial constraints the only way to attract a sufficient number of participants is to accept — without editorial review — all submitted abstracts, the quality of work reported in this expensive book is inevitably variable.

AT

Pathological basis of renal disease. 2nd ed. By M S Dunnill. (pp 568. Illustrated. £29.50). London: Baillière Tindall, 1984.

This is the second edition of a useful book originally published in 1976. It has grown by four chapters and almost a hundred pages though much of the text is unchanged from the previous edition. Its contents range widely but, as might be anticipated, several chapters are concerned with the detailed pathology of glomerulonephritis. The main clinical features of glomerular disease are presented, and the recently introduced WHO histological classification is given with some valuable accompanying explanation.

A chapter is devoted to renal involvement in liver disease, a subject of increasing interest often omitted in other texts. There are chapters dealing with topics such as the kidney in pregnancy, amyloid disease, tropical renal disease and renal complications in narcotic addicts. A section is now included on renal tumours — a defect in the previous edition. Separate chapters have been added on stone disease, tubular disorders and the various types of cystic lesion found in the kidney. It is a pity that the section on renal transplantation was not modernised to include some of the interesting work which has followed the development of monoclonal antibodies as well as the controversial discussion on the value of aspiration cytology.

The good quality illustrations are of light and immunohistological preparations with many electron micrographs — all integral parts of modern renal pathological analysis.

This readable book is an asset to any renal pathologist and to a pathology department library where it would be valuable to general pathologists and those in training. It should also be consulted by clinicians who are interested in the pathological aspects of the renal diseases they are seeking to investigate.

CMH

Inflammatory disorders of muscle. Edited by Barbara M Ansell. (pp 216. Illustrated. £12.50). London: Saunders, 1984. (Clinics in rheumatic diseases, vol 10, no 1).

This volume of this now well-established review series in rheumatology is a notable contribution to the better understanding of non-suppurative muscle disease. As usual, in this series, the contributors are well-known international authorities who approach the clinical, immunological, virological, genetic and diagnostic features in a critical and stimulating fashion, thus contributing towards a better understanding of pathogenesis. The review of treatment and management is practical and critical. The illustrations are, without exception, beautifully reproduced.

This is an outstanding review of the subject and will have a wide appeal, including as it does an up-to-date bibliography of major references. It is a book to be studied rather than read. The critical editorship of Dr Ansell is evident in the presentation and style which, for a multi-author publication, is remarkably uniform throughout. I can fully recommend this number of the *Clinics* as excellent value at the price.

MWJB

Ovulation and its disorders. Edited by W Thompson, R F Harrison and J Bonnar. (pp 185. £29.95). Lancaster: MTP, 1984. (Studies in fertility and sterility).

This book contains 33 abstracts of papers presented at the XIth World Congress of Fertility and Sterility in Dublin in 1983.

There are sections devoted to monitoring of ovulation and evaluating the function of the corpus luteum with emphasis on ultrasonography to study follicle growth and the use of serum and salivary progesterone to assess the luteal phase.

The sections on prolactin secretion and treatment of ovulation dysfunction contain little of value to the undergraduate or postgraduate student. Indeed, this book will be of interest only to those involved in active research into infertility and gynaecological endocrinology.

Its major drawback is that the papers are presented in abstract form only and thus such details that may normally be included in 'Patients and materials' are lacking. Also, as with most large conferences today because of financial constraints the only way to attract a sufficient number of participants is to accept — without editorial review — all submitted abstracts, the quality of work reported in this expensive book is inevitably variable.

AT

Clinical chemistry in diagnosis and treatment. By Joan F Zilva and P R Pannall. 4th ed. (pp 539. £9.00). London: Lloyd-Luke, 1984.

This is the fourth edition of what has become the standard chemical pathology text for medical students and junior hospital staff. In addition, the text has in the past proved invaluable to students studying for the Institute of Medical Laboratory Technology's special examination in chemical pathology.

The current edition has been extensively revised due to advances in knowledge and practice since the previous edition in 1979. Some may feel that the text requires expansion — in the chapters on endocrinology, for example — but the authors claim, and I feel rightly so, that, if the size and scope of the book were increased, its original purpose as a text for medical students would be defeated. This problem is partly overcome by direction to recent specialist articles at the end of each chapter.

Many of the common problems met by junior clinicians in practice, such as those of electrolyte and acid-base balance, have been discussed in detail. Many chapters contain appendices which give details of treatments for specific clinical problems. Several chapters have been included which stress the integrative roles of the chemical pathologist in the laboratory and the clinician on the ward, and a chapter on the rapidly expanding field of drug monitoring has been included. KDB

Mnemonics and tactics in surgery and medicine. By John J Shipman. 2nd ed. (pp 339. £6.50). London: Lloyd-Luke, 1984.

I have always found that mnemonics are of little use in learning medicine and I have never used them in teaching. It seems to me that it is more difficult to remember the mnemonic than to remember the facts that it points to. The use of mnemonics also supposes an approach to learning medicine by memorising 'lists' of causes, complications etc. I think that this approach is incorrect, and that students should be encouraged to understand the general pathological condition and the disturbed physiology. The reasons for the causes and complications will then become apparent and learning lists by rote will not be necessary.

It was with these thoughts in mind that I approached this book. I noted firstly that it is a second edition, so presumably there are those who find useful the approach that I abhor. Then I noted that the author says in his preface that physiology and pathology are invaluable. He explains that 'the easiest way .... is to understand and to work out a logical sequence' and he then says that mnemonics might help in this understanding process. Maybe he is right, and if you are one of those who find them useful, here is a veritable feast.

There are 414 mnemonics recorded here. Some of them are as difficult to remember as the original list; for instance the features of the Stevens-Johnston Syndrome are memorised by remembering DAMASCUS! There are also some mnemonics which are very similar. The causes of the retention of urine (p 143) is PASSING BIG DUTCH CAPS, and the second list of causes of diarrhoea (p 80) is PASS BIG DUTCH CAPS. Similarly, the features of hereditary spheracytosis (p 204) is BUGGARS, whereas the causes of a swollen joint (p 152) is IT'S A BUGGAR. I see a cause for confusion in some poor student!

If you like this sort of thing, then this is the most comprehensive manual on the market. There are also four excellent cartoons. WO-S

Acknowledgements

The Ulster Medical Journal acknowledges the generous contributions from the following bodies, without which it would not be possible to continue publication:-

Royal Victoria Hospital Medical Staff Committee, Royal Group of Hospitals Free Funds, Belfast City Hospital Free Funds, Ulster Hospital Dundonald Medical Staff Committee, Queen's University Belfast Grant from Senate Funds and the Northern Ireland Council for Postgraduate Medical Education.

Clinical chemistry in diagnosis and treatment. By Joan F Zilva and P R Pannall. 4th ed. (pp 539. £9.00). London: Lloyd-Luke, 1984.

This is the fourth edition of what has become the standard chemical pathology text for medical students and junior hospital staff. In addition, the text has in the past proved invaluable to students studying for the Institute of Medical Laboratory Technology's special examination in chemical pathology.

The current edition has been extensively revised due to advances in knowledge and practice since the previous edition in 1979. Some may feel that the text requires expansion — in the chapters on endocrinology, for example — but the authors claim, and I feel rightly so, that, if the size and scope of the book were increased, its original purpose as a text for medical students would be defeated. This problem is partly overcome by direction to recent specialist articles at the end of each chapter.

Many of the common problems met by junior clinicians in practice, such as those of electrolyte and acid-base balance, have been discussed in detail. Many chapters contain appendices which give details of treatments for specific clinical problems. Several chapters have been included which stress the integrative roles of the chemical pathologist in the laboratory and the clinician on the ward, and a chapter on the rapidly expanding field of drug monitoring has been included. KDB

Mnemonics and tactics in surgery and medicine. By John J Shipman. 2nd ed. (pp 339. £6.50). London: Lloyd-Luke, 1984.

I have always found that mnemonics are of little use in learning medicine and I have never used them in teaching. It seems to me that it is more difficult to remember the mnemonic than to remember the facts that it points to. The use of mnemonics also supposes an approach to learning medicine by memorising 'lists' of causes, complications etc. I think that this approach is incorrect, and that students should be encouraged to understand the general pathological condition and the disturbed physiology. The reasons for the causes and complications will then become apparent and learning lists by rote will not be necessary.

It was with these thoughts in mind that I approached this book. I noted firstly that it is a second edition, so presumably there are those who find useful the approach that I abhor. Then I noted that the author says in his preface that physiology and pathology are invaluable. He explains that 'the easiest way .... is to understand and to work out a logical sequence' and he then says that mnemonics might help in this understanding process. Maybe he is right, and if you are one of those who find them useful, here is a veritable feast.

There are 414 mnemonics recorded here. Some of them are as difficult to remember as the original list; for instance the features of the Stevens-Johnston Syndrome are memorised by remembering DAMASCUS! There are also some mnemonics which are very similar. The causes of the retention of urine (p 143) is PASSING BIG DUTCH CAPS, and the second list of causes of diarrhoea (p 80) is PASS BIG DUTCH CAPS. Similarly, the features of hereditary spheracytosis (p 204) is BUGGARS, whereas the causes of a swollen joint (p 152) is IT'S A BUGGAR. I see a cause for confusion in some poor student!

If you like this sort of thing, then this is the most comprehensive manual on the market. There are also four excellent cartoons. WO-S

Acknowledgements

The Ulster Medical Journal acknowledges the generous contributions from the following bodies, without which it would not be possible to continue publication:-

Royal Victoria Hospital Medical Staff Committee, Royal Group of Hospitals Free Funds, Belfast City Hospital Free Funds, Ulster Hospital Dundonald Medical Staff Committee, Queen's University Belfast Grant from Senate Funds and the Northern Ireland Council for Postgraduate Medical Education.