Volume 61, No 2. OCTOBER 1992
ISSN 0041-6193

THE ULSTER MEDICAL JOURNAL

[continued on back cover

Published by

THE ULSTER MEDICAL SOCIETY

CASE REPORTS

2. Plukker JTM, Joosten HJM, Rensing JBM, Van Haelst UJGM. Primary liposarcoma of the mediastinum in a child. *J Surg Oncol* 1988; **37**: 257-63.
3. Shibata K, Koga Y, Onitsuka T, *et al.* Primary liposarcoma of the mediastinum — a case report and review of the literature. *Jpn J Surg* 1986; **16**: 277-83.
4. Standerfer RJ, Armistead SH, Paneth M. Liposarcoma of the mediastinum: report of two cases and a review of the literature. *Thorax* 1981; **36**: 693-4.
5. Yang R, Elliston L, Paterson R, Sahmel R. Dysphagia and cough in a patient with a posterior mediastinal mass. *Chest* 1987; **92**: 529-30.
6. Schweitzer DL, Aguam AS. Primary liposarcoma of the mediastinum. *J Thorac Cardiovasc Surg* 1977; **74**: 83-97.
7. Enzinger FM, Weiss SW. Soft tissue tumours. St Louis: CV Mosby, 1988: 346-82.
8. Razzuk MA, Urschel HC, Race GJ, Kingsley WB, Paulson DL. Liposarcoma of the mediastinum: case report and review of the literature.*J Thorac Cardiovasc Surg* 1971; **61**: 819-26.
9. Bogoth ER, English E, Perrin RG, Tator CH. Successful surgical decompression of spinal extradural metastases of liposarcoma. *Spine* 1983; **8**: 228-35.
10. Mendez G Jr, Isikoff MB, Isikoff SK, Sinner WM. Fatty tumours of the thorax demonstrated by CT. *Am J Roentgenol* 1979; **133**: 207-12.
11. Munk PL, Müller NL. Pleural liposarcoma: CT diagnosis. *J Comput Assist Tomogr* 1988; **12**: 709-10.
12. Prohm P, Winter J, Ulatowski L. Liposarcoma of the mediastinum: case report and review of the literature. *Thorac Cardiovasc Surg* 1981; **29**: 119-21.

The Ulster Medical Journal, Volume 61, No. 2, October 1992.

The Ulster Medical Journal

The Journal of the Ulster Medical Society. First published in 1932.
Successor to the Transactions of the Ulster Medical Society (1884–1929), and the Transactions of the Belfast Clinical and Pathological Society (1854–1862)

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

THE ULSTER MEDICAL JOURNAL

NOTICE TO CONTRIBUTORS

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

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. Three copies of each manuscript should be submitted, including tables and figures.

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 19·5 × 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**: 1766-70 and Lancet 1979; **1**: 429-30). 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**: 136-41.

   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. Twenty-five reprints of each article will be forwarded free of charge to the corresponding author. Further reprints can be obtained from the printers, Messrs Dorman & Sons Ltd, 1-3 Holmes Street, Belfast BT2 7JG, who should be approached directly.

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 16mm and 35mm microfilm and 105mm microfiche from University Microfilms, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

9. For reprint information in the United States contact: International Reprint Corporation, 968 Admiral Callaghan Lane, Apt 268, PO Box 12004, Vallejo, California 94590 USA. Telephone (707) 553-9230, Fax (707) 552-9524.

The Ulster Medical Journal, Volume 61, No. 2, October 1992.

THE ULSTER MEDICAL SOCIETY

Whitla Medical Building
97 Lisburn Road
Belfast BT9 7BL

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 £30.00 per annum, payable in advance to the Honorary Treasurer.

J McKENNA, *President.*
J I LOGAN, *Hon. Secretary.*
M E CALLENDER, *Hon. Treasurer.*

MEMBERS **£10.00.** (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 — Annual subscription of Fellows **£25.00.** Husbands and wives who are both Fellows will be entitled to pay a combined subscription of **£30.00.**

All Fellows and Members of the Society who have paid subscriptions for 40 years or alternatively have 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.

SUBSCRIPTIONS: Any individual wishing to take out a direct subscription may do so. The cost is £30.00 for one volume (two numbers) of the Journal. Please apply for a banker's order to:

THE HONORARY TREASURER,
ULSTER MEDICAL SOCIETY,
WHITLA MEDICAL BUILDING,
97 LISBURN ROAD,
BELFAST BT9 7BL,
NORTHERN IRELAND.

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.

MEMBERSHIP: All Registered Medical Practitioners may apply for Fellowship or Membership of the Ulster Medical Society. Applicants should complete the form below and return it to:

THE HONORARY SECRETARY,
ULSTER MEDICAL SOCIETY,
WHITLA MEDICAL BUILDING,
LISBURN ROAD,
BELFAST BT9 7BL,
NORTHERN IRELAND.

APPLICATION FOR FELLOWSHIP OR MEMBERSHIP
OF THE ULSTER MEDICAL SOCIETY

Please enter your full name as recorded by the GMC. *This form may be photocopied.*

Forenames _____ Surname _____

Address _____

_____ Postcode _____

Degrees _____ Date of Graduation _____

Proposer (Print)_____ Seconder (Print) _____

Your proposer and seconder should be Fellows or Members of the Ulster Medical Society. Please contact the Honorary Secretary if you have difficulty in finding a proposer or seconder.

The Ulster Medical Journal, Volume 61, No. 2, pp. 127 – 133, October 1992.

Neural tube defects — prenatal diagnosis and management

R Ann Hamilton, J C Dornan

Accepted 1 August 1992.

SUMMARY

Neural tube defects rank second to congenital heart disease as a major cause of congenital malformation. Recent developments in ultrasound have improved prenatal diagnosis. Due to anomaly scans at 18 weeks gestation and the availability of a genetic clinic, prenatal diagnosis of neural tube defects at the Royal Maternity Hospital was 91·2% during 1987–1989. However, only 50% of parents accept termination of pregnancy and it is questionable if prenatal diagnosis is of benefit to those who wish to continue with the pregnancy. Parents may accept the situation better at birth, having had time to come to terms with it, helped with support from the obstetrician, clinical geneticist, paediatrician, genetic nurse and social worker. For some affected fetuses who have better muscle function and leg movement at term it appears from the literature that the outcome may be improved by caesarean section delivery. In Ireland fetuses with neural tube defects will continue to be delivered, as termination is unacceptable to many, but despite this there may be a positive benefit from prenatal diagnosis of neural tube defects. Prospective randomised controlled trials are needed to confirm benefit from delivery by caesarean section for fetuses with a good prognosis. As a result of prenatal diagnosis of a neural tube lesion the fetus should enjoy benefit in terms of physical morbidity, and the parents should benefit in terms of psychological morbidity.

INTRODUCTION

Neural tube defects are a heterogeneous group of malformations resulting from failure of neural tube closure up to the fourth week after conception. They have a multifactorial origin and range widely in severity from anencephaly to surgically correctable meningocele. They may be accompanied by neurologic, musculoskeletal, urologic and developmental abnormalities. They rank second to cardiac

Royal Maternity Hospital, Belfast BT12 6BA.

R Ann Hamilton, MB, ChB, MRCOG, Research Fellow.

Department of Obstetrics and Gynaecology, The Queen's University of Belfast, Institute of Clinical Science, Belfast BT12 6BA.

J C Dornan, MD, FRCOG, Science Lecturer, and Consultant Obstetrician and Gynaecolgist.

Correspondence to Dr Hamilton, Senior Registrar in Obstetrics and Gynaecology, Jubilee Maternity Hospital, Lisburn Road, Belfast BT9 7AB.

abnormalities as a cause of major congenital malformation, with a wide variation in prevalence worldwide and a well recognised increased prevalence in Northern Ireland of 3·3 per 1000 deliveries. Hydrocephalus is present in 80% of cases with spina bifida.

Recent ultrasound developments have resulted in better detection of these conditions prenatally. Anencephaly was the first fetal malformation detected by ultrasound [1] and in 1986 Nicolaides described ultrasonic markers of neural tube defects, scalloping of the frontal bones called 'the lemon sign' and anterior curvature of the cerebellar hemispheres called 'the banana sign'.[2] An alternative to ultrasound is biochemical screening by measurement of maternal serum alpha-fetoprotein. At present there is no alpha-fetoprotein screening in Ireland and its use in Great Britain is controversial.

Prenatal diagnosis of a major malformation allows the parents the option of termination of pregnancy, but it is questionable if it is of any benefit to them if termination is unacceptable. The aim of this paper is to report our experience with the diagnosis and management of pregnancies complicated by neural tube defects.

METHODS

Since 1987 in the Royal Maternity Hospital, all women who book early are offered an ultrasound scan performed at 18 weeks' gestation by ultrasonographers. A scan carried out at booking allows gestation to be confirmed thus ensuring accurate timing of this anomaly screening scan. Very few women refuse to have this anomaly scan booked. Any woman at risk of a child being affected with a neural tube defect either due to a past obstetric or family history, is offered referral to the genetic clinic. Recently maternal alpha-fetoprotein screening has been introduced for a trial period. These latter procedures are offered as an 'opt-in' service. If an abnormality is detected by an ultrasonographer at 18 weeks' gestation, an obstetrician is immediately informed. The obstetrician then informs the parents of the findings and an appointment is made for the next genetic clinic. At the genetic clinic the parents are seen by a clinical geneticist, prior to a further ultrasound scan and confirmatory tests performed by an obstetrician trained in perinatal medicine. Management is individualised after careful discussion involving the parents, obstetrician and clinical geneticist regarding the severity of the lesion and prognosis for the child.

If the parents decide that the pregnancy should be terminated, this is performed using gemeprost pessaries. Should termination be unacceptable, the parents receive full support throughout the remainder of the pregnancy. Recently, delivery by elective caesarean section at 37 weeks' gestation is offered to parents, where it is felt that there is a good prognosis for the child. Based on postnatal criteria established by Lorber in 1971[3] a good functional prognosis is predicted if the lesion is below the first lumbar vertebra, and is not associated with severe kyphoscoliosis, severe hydrocephalus or other gross congenital defects.

Pregnancies complicated by neural tube defects which were managed in the Royal Maternity Hospital during the three year period 1987–1989 are reviewed. The figures were obtained from delivery record books, genetic clinic records and the Infant Surgical Unit in the Royal Belfast Hospital for Sick Children.

RESULTS

During the three year period there were 34 pregnancies complicated by neural tube defects among 10,042 deliveries, a prevalence of 3·4 per 1000 deliveries. Of the 34 cases, 13 were anencephalics and 18 were spina bifida with or without hydrocephalus. Three cases were isolated hydrocephalus. Seventeen were female fetuses, 13 male and four unknown.

Over 90% of the cases were diagnosed prenatally (Table I). The three undiagnosed were a closed myelomeningocele, an occipital meningocele and a case with hydranencephaly. These cases were 'missed' diagnoses rather than 18 week anomaly scans not done, or refused. Of the three cases diagnosed in the third trimester one was booked at 33 weeks' gestation. 76·5% of the cases were diagnosed in the second trimester and an abnormality was missed initially in 14 women who had scans prior to 14 weeks' gestation. 41% of the abnormalities were diagnosed at the routine antenatal clinic by ultrasound scan. These were mostly anencephalic fetuses diagnosed at the booking visit, or fetuses with hydrocephalus noted later in the pregnancy. 35%, mainly spina bifida, were identified at the 18 week scan by the ultrasonographers and 15% were diagnosed at the genetic clinic in women who were high risk and had accepted genetic counselling.

TABLE I

Prenatal diagnosis of neural tube defects

Time of diagnosis	*No of cases (%)*
1st trimester	2 (5·9)
2nd trimester	26 (76·5)
3rd trimester	3 (8·8)
Undiagnosed	3 (8·8)

Sixteen women decided to have the pregnancy terminated after prenatal diagnosis. Of 13 women with anencephalic fetuses nine (69%) opted for termination and of 18 women with spina bifida fetuses seven (39%) opted for termination. The severity of the lesion did not always appear to influence the decision, as four women with anencephalic fetuses continued with the pregnancy (Table II). Marital status also appeared unimportant, as four of the women were unmarried at the time of diagnosis; one was late booked, one decided to terminate the pregnancy and two married after the diagnosis was made and before the delivery.

TABLE II

Action taken after prenatal diagnosis of neural tube defect

Action	*No of cases*
Termination of pregnancy	16 (9 anencephaly 7 spina bifida)
Continuation of pregnancy	15 (4 anencephaly 11 spina bifida)
Undiagnosed	3

Two fetuses diagnosed ultrasonically as having encephaloceles had large cystic hygromas at delivery. Of the 18 patients who continued with the pregnancy, eight had delivery by caesarean section, five electively at 37 weeks' gestation and three as emergency procedures. Ten women had vaginal delivery, four with associated destructive procedures. The perinatal outcome is shown in Table III. All the babies delivered by caesarean section had five-minute Apgar scores greater than seven. The follow up of the nine babies who survived the neonatal period is shown in Table IV.

TABLE III

Perinatal outcome of pregnancies after prenatal diagnosis of neural tube defects

Mode of delivery	*No of cases*	*Outcome*
Vaginal delivery with or without destructive procedure	10	8 stillbirths 1 early neonatal death 1 livebirth
Caesarean section	8	8 livebirths

TABLE IV

Follow up of neonates after prenatal diagnosis of neural tube defects

Mode of delivery	*Follow up*
Normal vaginal delivery 1	Alive and well
Elective caesarean section 5	4 early closure with or without shunt Alive and well
	1 unsuitable for closure Alive
Emergency caesarean section 3	2 alive and well 1 died at 15 months

DISCUSSION

The incidence of neural tube defects during 1987–1989 in the Royal Maternity Hospital is similar to the higher than normal prevalence recognised throughout Northern Ireland. Compared with other studies which report equal distribution of anencephaly and spina bifida, we found 52% spina bifida compared with 38% anencephaly.[4] We found a preponderance of abnormality among female fetuses.

Despite achieving a diagnosis in 90% of cases, it was initially missed in fourteen women who had a scan prior to 14 weeks' gestation, due to difficulty in clear visualization of the spine and cerebellum before 18 weeks. No case of open neural tube lesion was missed in those women booked for confinement in the Royal Maternity Hospital who had a routine ultrasound scan at 18 weeks' gestation (approximately 10,000 during the three year period).

Regarding prenatal diagnosis, efficacy is described as detection and termination of those cases within a given population who are known to be affected *in utero.*

A discrepancy between diagnosis and efficacy exists due to several factors. Firstly, women not being screened either because they book late or decline screening, secondly, inaccuracy of the test itself and lastly, failure to terminate the pregnancy. In the present study efficacy was 47% which is comparable with other studies,[5, 6] but in studies from England only 10% of women diagnosed as carrying a fetus with neural tube defects declined termination compared with 50% in our population.[5, 6, 7] In our clinic the parents are fully supported in whatever decision they make regarding the pregnancy, and it is therefore difficult to question why some decide not to have an affected pregnancy terminated, but it is felt that in most cases it is on religious or conscience grounds.

The recent introduction of gemeprost pessaries has improved the termination procedure and has almost eliminated the need for extra-amniotic prostaglandin. It is important that a fetus delivered after an induced abortion for an abnormality is sent for examiantion by a paediatric pathologist and by a clinical geneticist experienced in dysmorphology, as this may influence subsequent genetic counselling.

This point is highlighted in a report from the Manchester Regional Centre where of 71 cases aborted for neural tube defects, 56 were confirmed, two also had cleft palates, five had multiple abnormalities, three had autosomal recessive malformations, and one had a cystic hygroma; all of these may lead to an increased risk in subsequent pregnancies.[8] In the majority of our cases although the fetus was examined by a clinical geneticist, postmortem examination was refused by the parents. The importance of this procedure should be explained to the parents.

In our study some of the parents of an anencephalic fetus, who had declined termination, requested early delivery in the third trimester. At this stage these parents accept 'early delivery' as opposed to 'termination', and we regard this as supporting the parents in their choice rather than failure of the support system offered to them. Chervenak *et al* in 1984 feel that termination at this stage is both moral and legal if the fetus is affected with a condition incompatible with postnatal survival by more than a few weeks, characterised by total or virtual loss of cognitive function, and where highly reliable diagnostic procedures are available.[9] Anencephaly fulfils these criteria, with a study of 102 cases reporting no false positives or false negatives in prenatal diagnosis.[10] The results of our study were similar.

Major chromosomal aberrations may be associated with neural tube defects[11] as may other anatomical abnormalities and these should be excluded by a detailed ultrasound scan by an experienced sonographer and by other appropriate investigations e.g. amniocentesis or cordocentesis. The team involved in counselling should include the obstetrician, clinical geneticist, neonatologist, paediatric surgeon, genetic nurse and social worker. Where the parents elect to continue with the pregnancy or when the diagnosis is made late, they should receive full support throughout the antenatal period.

One advantage of prenatal diagnosis is that it allows choice of timing and mode of delivery to provide optimal outcome. For those who opt to continue with a pregnancy after anencephaly has been diagnosed, vaginal delivery should obviously be aimed for. However, parents should not be told that the child will die immediately, as some survive for short periods. A retrospective study of 130 cases

with meningomyelocele by Stark and Drummond in 1970,[12] reported 14% showing signs of cerebral birth injury. The authors considered that for many neonates the neurologic deficit increased during vaginal delivery. There is evidence from Luthy *et al*[13] that if a fetus with spina bifida has good muscle function and leg movement by the time the lungs are mature, paralysis is minimised by an elective caesarean section at 37 weeks' gestation prior to the onset of labour. This study is the only prospective one; though it was not randomised. It compares 47 children delivered by elective caesarean section with 113 delivered vaginally or by caesarean section after the onset of labour. Of those born by pre-labour caesarean section subsequent motor function was better, but neonatal complications and subsequent intellectual performance showed no difference between the two groups. The authors claim better outcome was due partly to a less-impaired series of cases, partly because of selection of only the least-impaired for caesarean section and less damage to the infants as a result of the method of delivery. A group from North Carolina reviewed 32 affected children delivered by caesarean section compared with 40 delivered vaginally who were followed up for one year. They showed no significant difference in mortality, hospital stay, neurologic or developmental status.[14]

Of the group delivered vaginally, three developed meningitis compared with only one in the section group but a larger study would be needed to show any significance in this finding. Our findings, although not randomised or comparable, show a poor outcome associated with vaginal delivery, only one of ten surviving.

For the family the quality of life is important. Of the nine survivors in our study one died at the age of 15 months, one required no treatment after delivery, one had a lesion too extensive for closure and the remaining six required closure of the defect. It is impossible to compare the two groups as the cases with better prognosis were chosen for operative delivery and therefore introduce bias in favour of better outcome.

What is the future regarding prenatal diagnosis of neural tube defects? It is generally anticipated that a decreasing number of children will be born with these conditions as primary prevention and prenatal diagnosis advance further. However, in Ireland, whether the defect is diagnosed by ultrasound, serum alpha-fetoprotein or amniocentesis, we will continue to deliver affected babies as termination is unacceptable to many parents. It is possible that prenatal diagnosis may not only contribute to decreasing the number of affected children but may also decrease the associated morbidity and mortality for those who wish to continue with the pregnancy. Regarding the mode of delivery, each case needs to be assessed individually. Only a prospective randomised controlled trial involving a group of centres will prove any potential benefit from elective caesarean section.

REFERENCES

1. Campbell S, Johnstone FD, Holt EM, May P. Anencephaly: early ultrasonic diagnosis and active management. *Lancet* 1972; **2**: 1226-7.
2. Nicolaides KH, Campbell S, Gabbi SG, Guidetti R. Ultrasound screening for spina bifida: cranial and cerebellar signs. *Lancet* 1986; **2**: 72-4.
3. Lorber J. Results of treatment of myelomeningocele: an analysis of 524 unselected cases, with special reference to possible selection for treatment. *Dev Med Child Neurol* 1971; **13**: 279-303.

4. Windham GC, Edmonds LD. Current trends in the incidence of neural tube defects. *Pediatrics* 1982; **70**: 333-7.
5. Roberts CJ, Hibbard BM, Elder GH *et al.* The efficacy of a serum screening service for neural tube defects: the South Wales experience. *Lancet* 1983; **1**: 1315-8.
6. Lorber J, Ward AM. Spina bifida — a vanishing nightmare? *Arch Dis Child* 1985; **60**: 1086-91.
7. Second report of UK collaborative study on alpha-fetoprotein in relation to neural tube defects. Amniotic fluid alpha-fetoprotein measurement in antenatal diagnosis of anencephaly and open spina bifida in early pregnancy. *Lancet* 1979; **2**: 651-61.
8. Clayton-Smith J, Farndon PA, McKeown C, Donnai D. Examination of fetuses after induced abortion for fetal abnormality. *Br Med J* 1990; **300**: 295-7.
9. Chervenak FA, Farley MA, Walters L, Hobbins JC, Mahoney MJ. When is termination of pregnancy during the third trimester morally justifiable? *NEJM* 1984; **310**: 501-4.
10. Murken JD, Stengel-Rutkowski S, Swinger E. Prenatal diagnosis of genetic disorders. Stuttgart: Ferdinand Enke, 1979: 94-192.
11. Chervenak FA, Goldberg JD, Chiu T, Gilbert F, Berkowitz RL. The importance of karyotype determination in a fetus with ventriculomegaly and spina bifida discovered during the third trimester. *J Ultrasound Med* 1986; **5**: 405-6.
12. Stark G, Drummond M. Spina bifida as an obstetric problem. *Dev Med Child Neurol* 1970; **12** Suppl 22: 157-60.
13. Luthy DA, Wardinsky T, Shurtleff DB *et al.* Cesarean section before the onset of labour and subsequent motor function in infants with meningomyelocele diagnosed antenatally. *NEJM* 1991; **324**: 662-6.
14. Bensen JT, Dillard RG, Burton BK. Open spina bifida: does caesarean section delivery improve prognosis? *Obstet Gynecol* 1988; **71**: 532-4.

The Ulster Medical Journal, Volume 61, No. 2, pp. 134 - 138, October 1992.

Coeliac disease: clinical presentations, correlations of dietary compliance, symptomatic response and repeat biopsy findings

N G McElvaney, R Duignan, J F Fielding

Accepted 9 August 1992.

SUMMARY

Gastrointestinal symptoms were present at the time of diagnosis in 81 (76%) of 107 patients with coeliac disease: 56% had diarrhoea / steatorrhoea, 32·7% abdominal pain and 15% constipation. Gastrointestinal symptoms were commonest in young adults (20–39 years) and less frequent in children (0–19 years).

Anaemia, low serum levels of folic acid, albumin and calcium, and raised serum alkaline phosphatase may be of help in raising the index of diagnostic suspicion, but in over half of our patients with clinically and histologically active disease these values were within normal limits.

In patients adhering to a gluten free diet remission of symptoms correlated well with histological response; the continuation of symptoms indicated a higher incidence of histological abnormality. No patient not complying to the diet had normal histology on repeat biopsy. Five patients died over the ten year period, one from a small bowel lymphoma.

INTRODUCTION

Classical coeliac disease presenting with diarrhoea / steatorrhoea, abdominal pain, weight loss and abdominal distention presents a relatively easy diagnostic problem which is definitively solved on jejunal biopsy. Once started on a gluten free diet most patients improve clinically and the jejunal villous pattern should return to normal. Not all patients present in this classical fashion and symptomatic response does not necessarily correlate with return to a normal villous pattern.

We studied 107 patients and reviewed their clinical presentations in conjunction with the haematological and biochemical tests. We correlated disease activity on biopsy to symptoms, and to dietary compliance. We also studied prognosis

Department of Medicine and Gastroenterology, The Charitable Infirmary, Dublin 1.

N G McElvaney, MB, MRCPI, Tutor Registrar.

R Duignan, MB, Senior House Officer.

J F Fielding, BSc, MD, FRCPI, FRCP, FACG, Professor of Medicine.

Correspondence to Prof J F Fielding, Department of Medicine and Gastroenterology, Beaumont Hospital, Dublin 9, Ireland.

particularly in comparison to a similar study carried out in the Royal Victoria Hospital, Belfast.

MATERIALS AND METHODS

We reviewed the records of all patients with subtotal villous atrophy on jejunal biopsy in the gastroenterology department of Jervis St. Hospital, Dublin between October 1975 and April 1986. A diagnosis of coeliac disease was accepted if, in the appropriate clinical setting, the histology revealed subtotal villous atrophy.[1] The age at diagnosis, age at onset of symptoms, duration of follow up and relevant family history were identified. The reasons for performing the biopsy were identified and symptoms present at time of the biopsy diagnosis noted.

Patients were classified into those who claimed to be adhering to a gluten free diet and those who said they were not keeping to the diet: no dietetic assessment of these claims was made. Differences between groups were tested for significance using Chi squared analysis, with Yates correction.

RESULTS

The diagnosis of coeliac disease was made in 107 patients, 73 female (68·2%) and 34 male (31·8%). A positive family history was noted in 19 patients, and 15 of the patients in this study were related to at least one other study member. Forty two were diagnosed below age 20 years, forty between 20 and 40, and only five over the age of 60, two of these being over 70 years. Twelve of the 107 patients had been lost to follow up. The average duration of follow up of the remaining 95 patients was 9·2 years.

Gastrointestinal symptoms were the commonest indication for biopsy (81 patients). Symptoms at presentation were diarrhoea/steatorrhoea (60 patients), abdominal pain (35), constipation (16), lethargy (16), and weight loss (9): fainting, paraesthesia, bone pain, anorexia and abdominal swelling occurred only in a few patients. Gastrointestinal complaints as an indication for biopsy between the different age groups are summarised in Table I.

TABLE I

Symptoms related to patient age

	Age group (years)			
	0 – 19	*20 – 39*	*40 – 59*	*60 +*
Total	*42*	*40*	*20*	*5*
Gastrointestinal symptoms	25 (60%)	38 (95%)*	14 (70%)	4 (80%)
Diarrhoea/steatorrhoea	24 (57%)	22 (55%)	11 (55%)	3 (60%)
Abdominal pain	14 (33%)	17 (42%)	4 (20%)	0
Constipation	5 (12%)	3 (8%)	4 (20%)	4 (80%)
Weight loss	5 (12%)	2 (5%)	2 (10%)	0
Anorexia	0	0	3 (15%)	0
Abdominal swelling	2 (5%)	0	0	0
Vomiting	0	0	0	0

*Significantly greater than the 0 – 19 ($p < 0 \cdot 001$) and 40 – 59 ($p < 0 \cdot 01$) age groups.

Forty six (45%) of 21 patients were anaemic at presentation (Hb < 12·0 g/dl): 20 (21%) were macrocytic and 15 (14%) microcytic. Serum folic acid was less than 2·0 mg/dl in 42 (39%). Hypoalbuminaemia (< 40 g/l) was found in 45 (42%), hypocalcaemia (< 2·1 mmol/l) in 28 (26%) and an elevated serum alkaline phosphatase (> 250 μ/l) in 27 (25%).

Information concerning dietary compliance or lack of it and the results of further biopsies are shown in Table II.

TABLE II

Claimed adherence to gluten free diet in 95 patients

		Result of repeat biopsy		
	No	*Normal*	*Abnormal+*	*Not done*
Complying with diet (75)				
Remission of symptoms	48	31	9	8
Improvement in symptoms	24	5	9	10
No change	3	1	1	1
Not complying with diet (20)				
Remission of symptoms*	2	0	2	0
Improvement in symptoms	11	0	8	3
No change	7	0	4	3

+Abnormal; either subtotal or partial villous atrophy.[1]

*The two patients who claimed remission of symptoms despite non adherence to a gluten free diet had both initially adhered to their diets and had normal jejunal biopsies, but reverted to subtotal villous atrophy on stopping their diet.

Five patients died over this ten year period, two from malignancy, and one each from pulmonary embolus, cerebrovascular accident and myocardial infarction. Of those dying from malignancy one had a small bowel lymphoma and the other a cerebral glioma. The patient with lymphoma had initially presented at age 61 with diarrhoea, flatulence, anergia and weight loss: a jejunal biopsy revealed subtotal villous atrophy and she was commenced on a gluten free diet with good symptomatic effect. Repeat biopsy after 14 months still showed subtotal villous atrophy, and around that time her condition deteriorated with recurrence of diarrhoea, weight loss and development of ascites. Abdominal CT scan revealed lymphadenopathy which was confirmed on laparotomy. She died following acute occlusion of a leg artery.

DISCUSSION

Gastrointestinal symptoms predominate in the initial presentation of coeliac disease. Diarrhoea/steatorrhoea was the commonest presenting complaint noted by Barry, Baker and Read[2] but the incidence in our population (56%) was considerably lower. Other authors have found between 74% and 97% of their patients to have diarrhoea or steatorrhoea.[3,4,5] Barry, Baker and Read commented on the fact that diarrhoea is less common as a presenting complaint in the adult

population but we found it consistently in our various age groups. Gastrointestinal symptoms accounted for 95% of presenting complaints in the 20–39 year age group, but only 60% of the 0–19 year age group. The other age groups were intermediate.

In our population 15% gave constipation as a presenting complaint, leaving 29% without any symptoms of altered bowel habit. Constipation has been reported as a presenting complaint in childhood coeliac disease, in 12 out of 112 children, nine of whom had faecal impaction.[6] In our study constipation occurred in all age groups.

Abdominal pain was the second most frequent complaint, especially in the 20–39 group, which also contrasts with one previous report,[2] although others found up to 42% of patients with this symptom.[5]

Jejunal biopsy is a safe diagnostic investigation. The indications for biopsy depend mainly on clinical, haematological and biochemical abnormalities. The correlation between these parameters and disease activity is of practical importance, but in our study an abnormally low haemoglobin was detected in only 45%, an abnormal serum folate in 39% and a low albumin in only 42% of patients. The Belfast study also indicated the need for caution in allowing a normal blood test to allay a clinical suspicion of malabsorption.[5]

Serum albumin concentration was below 35 g/dl in only 14% of patients. In a western population on a normal diet, values of less than 40 g/dl would be a more reasonable index of hypoalbuminaemia,[6] and 42% of our patients were below this level. Benson and his colleagues detected hypoproteinaemia in 73% of patients.[3]

On review of those claiming adherence to diet and complete remission of symptoms, and who had repeat biopsies, 78% had normal villi; in those claiming improvement but not full remission only 36% had a normal villous pattern. All those not adhering to diet but claiming improvement had subtotal or partial villous atrophy on repeat biopsy. Return to a normal villous pattern correlated well with complete remission of symptoms in patients on diet, but not in those who only had improvement in symptoms. A normal villous pattern did not occur in patients not adhering to diet.

Nineteen (17·8%) of our patients were first degree relatives. Selection bias would account for this figure which is higher than the previously recorded risk of coeliac disease among first degree relatives: 10·3% from the West of Ireland,[8] 11·2% from England[9] and 11·5% from America.[10]

Five of our patients died; only two from malignancy, one being a lymphoma. Most previous studies show a high incidence of lymphoma and carcinoma in coeliac patients. Brandt *et al*[11] found five cases of intestinal lymphoma in 74 patients: Holmes *et al*[12] found 28 malignancies in 210 patients, 22 being either lymphoma or carcinoma of the gastrointestinal tract. The average age at diagnosis of coeliac disease in these lymphoma patients was 53 years,[12] whereas 95% of our patients were under 60 years and 76% under 40 years at the time of diagnosis. In the study by Holmes and his colleagues[12] the duration from diagnosis of coeliac disease to lymphoma averaged 26 years, and averaged 25 years from diagnosis to carcinoma. Longer follow up and advancing age in the present group of patients may well result in an increased incidence of malignancy.

REFERENCES

1. Morson BC. In: Colour atlas of gastrointestinal pathology. BC Morson (ed). Oxford: Oxford University Press, 1988; 130.
2. Barry RE, Baker PK, Read AE. The clinical presentation. In: Clinics in gastroenterology; coeliac disease. WT Cooke, P Asquith (eds). London: WB Saunders, 1974; 55-69.
3. Benson GD, Kowlessar OD, Sceiserger MM. Adult celiac disease with emphasis upon response to the gluten-free diet. *Medicine* (Baltimore) 1964; **43**: 1-40.
4. Cooke WT, Peeney ALP, Hawkins CF. Symptoms, signs and diagnostic features of idiopathic steatorrhoea. *Quart J Med* 1954; **22**: 59-78.
5. Boyd S, Collins BJ, Bell PM, Love AHG. Clinical presentation of coeliac disease in adult gastro-enterological practice. *Ulster Med J* 1985; **54**: 140-7.
6. Egan-Mitchell B, McNicholl B. Constipation in childhood coeliac disease. *Arch Dis Child* 1972; **47**: 238-40.
7. Fielding JF. Clinical assessment in the follow up of patients with regional enteritis; its correlation with haematological and biochemical parameters. *Irish Med J* 1971; **64**: 221-4.
8. Mylotte MJ, Egan-Mitchell B, Fottrell PF, McNicholl B. Familial coeliac disease. *Quart J Med* 1972; **41**: 527-8.
9. Stokes PL, Ferguson R, Holmes GKT, Cooke WT. Familial aspects of coeliac disease. *Quart J Med* 1976; **180**: 567-82.
10. MacDonald WC, Dobbins WO, Rubin CE. Studies on the familial nature of celiac sprue using biopsy of the small intestine. *New Engl J Med* 1965; **272**: 448-56.
11. Brandt L, Hagander B, Nordén Å, Stenstam M. Lymphoma of the small bowel in adult coeliac disease. *Acta Med Scand* 1978; **204**: 467-70.
12. Holmes GKT, Stokes PL, Sorahan TM, Prior P, Waterhouse JAH, Cooke WT. Coeliac disease, gluten-free diet and malignancy. *Gut* 1976; **17**: 612-9.

The Ulster Medical Journal, Volume 61, No. 2, pp. 139 – 143, October 1992.

What do medical students know about chronic pain and its management?

W I Campbell

Accepted 11 August 1992.

SUMMARY

A questionnaire on chronic pain management was completed by 114 third year, and 80 fifth (final) year medical students. The results demonstrated a significant improvement in most facets of chronic pain knowledge and management by the end of the medical course. A postal survey was undertaken of all pain clinics in Northern Ireland to identify current activity, as these figures are not collected in Korner returns.

Despite the improvement in knowledge by final year, there were still many students who did not know what type of problem to refer to a pain clinic (30%), who ran the clinics (> 40%), or the types of treatment commonly employed. More formal education is required on this subject, at both undergraduate and postgraduate level.

INTRODUCTION

The management of chronic pain is difficult, despite the frequency with which it is encountered in medical practice. Various medical specialties have something to offer in these situations. Usually this involves treating the pathological process giving rise to the pain, but occasionally one may have to resort to the empirical management of the symptom alone.

Advances in chronic pain management lag far behind other branches of medicine and the subject is considered to be largely ignored in the undergraduate curriculum.[1] This study was aimed at determining the extent of medical student knowledge on the subject of chronic pain and its management at two periods during the five year undergraduate training in Belfast — late in the third year and in the final year. Data relating to chronic pain management resources are not available through the Department of Health and Social Services, either by region or nationally (a Korner number has yet to be allocated for this purpose). A further survey was therefore carried out to gather this information for Northern Ireland, as baseline data.

METHOD

Third year and final year medical students at the Queen's University of Belfast were asked to complete a questionnaire whilst awaiting a lecture. Well-attended lectures were chosen and the students were asked to complete the questionnaire

The Ulster Hospital, Dundonald, Belfast BT16 0RH.

W I Campbell, MD, FFARCSI, Consultant Anaesthetist.

within 10 minutes without conferring. The participants were asked to respond to multiple choice questions on the definition of chronic pain, the types of treatment and services available. Open-ended questions were included — location of the nearest pain clinic, the number of clinics within Northern Ireland, who ran them, and the types of problem most frequently encountered.

A second questionnaire was sent to national health service hospitals within Northern Ireland to evaluate organised activity aimed at chronic pain management and the resources available at the time of the medical student study. Questions included the number of hours spent each week on chronic pain management, the number of patients seen and the extent of teaching. Those contacted included anaesthetists running pain clinics, neurosurgeons and clinical psychologists. The Northern Ireland Hospice figures were not obtained. It was agreed that information from this second survey would not allow the identification of any individual clinic or establishment.

Results from the medical student survey were analysed using chi squared analysis, with Yates correction and Fisher's exact test, as appropriate.

RESULTS

A total of 194 medical students completed the questionnaires and a comparison of the results between third and final year medical students is presented in Table I (pain clinic knowledge) and Table II (types of problem seen, treatments available and definition of chronic pain). Less than half of the third year students knew that pain clinics existed, but the fifth (final) year students all knew of them and most knew where the nearest one was located. A large proportion of each group expressed uncertainty regarding the number of pain clinics in Northern Ireland, but more than half of the final year students knew who ran them. Most final year students selected the correct definition for chronic pain, and the correct type of pain problem to be referred to a pain clinic.

To put these results in perspective, the survey of chronic pain management in Northern Ireland is given. Fifteen consultant anaesthetists offer a chronic pain management service, 12 of these do so by holding regular pain clinics. This service is available at 11 hospitals, offering a total of 21·5 sessions per week (72·25 hours). This time is divided between outpatient and inpatient work in the ratio of 2 : 1 respectively. A total of 5,158 outpatient attendances during the study year occurred at these clinics — 1,313 of these were new patients. All but two sites had regular nursing help and access to X-ray facilities. Four sites made regular use of theatre facilities, whilst another four did so on an occasional basis. Teaching was limited to postgraduates, most of this occurring at three sites but five other sites had the occasional postgraduate attender. Two neurosurgeons and one psychologist also dealt with specialised aspects of chronic pain management on a regular basis. Other disciplines were not included in these results as they do not offer a dedicated service or clinic for chronic pain management.

DISCUSSION

Pain is one of the body's protective mechanisms. Following injury, nociception is a warning of tissue damage activating a withdrawal from the source of danger. Acute pain is self-limiting, generally responding favourably to simple and conventional treatments, such as opioids or non-steroidal anti-inflammatory drugs.

TABLE I

Medical student awareness of pain clinics and who runs them — numbers (percentage)

		Third year medical students n = 114		*Final year medical students* n = 80	
		No	%	No	%
Know that pain clinics exist		55	(48)	80	(100) **
Aware of pain clinic existence through lectures		13	(11)	28	(35) **
Knew where the nearest pain clinic was sited		26	(23)	65	(81) **
Thought the number of pain clinics in Northern Ireland was					
	1 – 3	10	(9)	18	(23) *
	4 – 10	7	(6)	15	(19) *
	Unknown	97	(85)	46	(58) **
Thought that the pain clinic was run by	anaesthetist	5	(4)	44	(55) **
	psychologist	5	(4)	2	(3) NS
	physician	10	(9)	9	(11) NS
	others	12	(11)	13	(16) NS
	unknown	82	(72)	12	(15) **
Selected the appropriate type of problem for pain clinic referral		63	(55)	66	(83) **

NS = Not significant. *p = 0·01. **p = 0·001.

TABLE II

Common problems referred to pain clinics and types of treatment considered available there. (More than one treatment could be selected)

		Third year medical students n = 114		*Final year medical students* n = 80	
		No	%	No	%
Correct definition of chronic pain selected		40	(35)	66	(83)
Thought the most frequent type of problem referred was					
	cancer pain	15	(13)	12	(15) NS
	back pain	9	(8)	22	(28) **
	arthrosis	12	(11)	10	(12) NS
	musculoskeletal	0	(0)	4	(5) *
	neuralgias	0	(0)	4	(5) *
	other	5	(4)	4	(5) NS
	unknown	73	(64)	24	(30) **
Methods of chronic pain management available	opioids	33	(29)	56	(70) **
	non-steroidal anti-inflammatory drug	39	(34)	53	(66) **
	psychotropic drugs	48	(42)	52	(65) **
	nerve blocks	36	(32)	77	(96) **
	physiotherapy	43	(38)	63	(79) **
	hypnosis	26	(23)	39	(49) **
	acupuncture	31	(27)	50	(63) **
	unspecified	10	(9)	8	(10) NS

NS = Not significant. *p = 0·01. **p = 0·001.

Chronic pain is generally more difficult to manage, pathways being multiple and devious, often without serving any useful purpose.[2] In addition, the aetiology of chronic pain may be difficult to determine.[3] Pain is even difficult to define, being a mixed sensory and affective feeling. Perhaps the best definition of pain given to date is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage".[4] For the purposes of this survey, chronic pain was defined as pain persisting for three or more months, (Taxonomy Committee of the International Association for the Study of Pain).[5]

The results of the medical student survey demonstrated a marked increase in knowledge of both chronic pain and its management between third year and final year. Thirty-five per cent of the final year students indicated that they had heard of pain clinics through formal lectures, whilst the remainder came across these clinics whilst working in hospitals. Although most final year students knew where the nearest pain clinics were, 45% did not know who ran them, and 17% would have referred inappropriate problems (reflected by the fact that these individuals did not know the correct definition of chronic pain). Of the methods of chronic pain management available, only two-thirds of the final year students were aware of the value of non-steroidal anti-inflammatory drugs, psychotropic drugs and acupuncture, despite their common use and relative effectiveness.[6, 7] On questioning students after the survey, most of the final years' knowledge on chronic pain was gleaned piecemeal from lectures on other subjects and from clinical attachments.

The type of problems seen and their method of referral in Northern Ireland is typical of the rest of the British Isles, with approximately half of all new referrals coming from orthopaedic surgeons, the remainder coming through other hospital doctors and general practitioners. Although carcinoma was responsible for about 50% of all pain clinic referrals 30 years ago, and indeed was the reason for their inception by anaesthetists due to their knowledge of nerve blocks, this is no longer the case.[9, 10] The most frequent type of problem seen now is related to spinal arthritis and supportive tissue injury, with neurogenic and symphathetically mediated pain accounting for most of the remainder; these are appropriate problems for pain clinic referral. Apart from the 30% of final year students who didn't know the type of problem usually referred, the referral pattern shown in Table II is quite close to that seen in most pain clinics.

National statistics give some indication of the size of the problem due to chronic pain, which may cost the NHS hundreds of millions of pounds annually, and the Department of Health and Social Services a similar amount in respect of claims for back pain. The loss to industry alone was estimated to be £1,018 million for the year 1982.[11] Although pain clinic activity may not result in a notable fall in these costs, it can markedly improve the quality of life in many of those who suffer chronic pain.

With an increasing awareness of the need for better training in the management of acute pain, there is a place for simultaneous teaching in the origins and management of the massive problem of chronic pain, as knowledge in this field is changing rapidly.[13] Since this survey was carried out, at least one formal lecture on the subject is given annually to fourth year medical students, informing them

of the correct definition of chronic pain, some methods of management, and where advice can be obtained. Following the General Medical Council's initiatives for curricular reform,[14] and acknowledging the widespread concern about overloading of the undergraduate medical curriculum, The Queen's University of Belfast has recently set up a working party on curricular reform which will look into this problem.

REFERENCES

1. Mehta M. The history of The Intractable Pain Society of Great Britain and Ireland. *Pain* 1980; **8**: 121-2.
2. Melzack R, Wall P. The challenge of pain. London: Penguin Books, 1988: 34-6.
3. Mehta M, Sluijter ME. The treatment of chronic back pain. A preliminary survey of the effect of radiofrequency denervation of the posterior vertebral joints. *Anaesthesia* 1979; **34**: 768-75.
4. Merskey H, *et al.* Pain terms: a list with definitions and notes on usage. Recommended by the IASP subcommittee on taxonomy. *Pain* 1979; **6**: 249-52.
5. Merskey H. International Association for the Study of Pain (Subcommittee on Taxonomy). Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. Pain Supplement 3: S5. Amsterdam: Elsevier, 1986.
6. Budd K. Psychotropic drugs in the treatment of chronic pain. *Anaesthesia* 1978; **33**: 531-4.
7. Mehta M. Alternative methods of treating pain. *Anaesthesia* 1978; **33**: 258-63.
8. Gauci CA. The pain relief unit — a clinical audit. *Journal of the Pain Society of Great Britain and Ireland.* IPS Forum 1989; **6**: 9-11.
9. Swerdlow M, Mehta MD, Lipton S. The role of the anaesthetist in chronic pain management. *Anaesthesia* 1978; **33**: 250-7.
10. Lipton S. Relief of pain in clinical practice. Oxford: Blackwell Scientific Publications, 1979: 306-66.
11. Wells N. Back pain. London: Office of Health Economics, 1985; **78**: 9-11.
12. Commission on the provision of surgical services. Report of the working party on pain after surgery. The Royal College of Surgeons of England. The College of Anaesthetists. 1990.
13. McQuay HJ, Dickenson AH. Implications of nervous system plasticity for pain management. *Anaesthesia* 1990; **45**: 101-2.
14. Annual Report of the General Medical Council. London, 1991: 12-3.

The Ulster Medical Journal, Volume 61, No. 2, pp. 144 – 150, October 1992.

Benefits of an exercise class for elderly women following hip surgery

S A Henderson, Olwen E Finlay, Niamh Murphy, C Boreham, R A B Mollan, D H Gilmore, T R O Beringer

Accepted 20 September 1992.

SUMMARY

A prospective cohort study of a twice weekly exercise programme for six months was undertaken to determine the benefits of an exercise class for 28 elderly women following hip surgery. The effects of the exercise programme were monitored using cycle ergometry. Walking speed was measured on entry and at 3, 6 and 12 months. Twenty-six subjects completed the programme with an overall attendance rate of 88%. Measures of fitness, calculated from cycle ergometry, did not improve significantly apart from test duration. In contrast there was a significant improvement in mean walking speed, with a 50% increase between 0 and 3 months and a further 21% increase between 3 and 6 months. This improvement was maintained at 12 months.

INTRODUCTION

Elderly women with osteoporotic fractures of the hip or primary osteoarthritis requiring total hip replacement form the majority of orthopaedic patients requiring hip surgery. Their numbers are increasing as the proportion of elderly in the population increases.[1] They are considerable consumers of rehabilitation services both in hospital and in the community.

The quality of life for elderly women following hip surgery is largely dependent upon their ability to pursue a variety of physical activities. Reduction in levels of normal physical activity with age and following hip surgery leads to a deterioration in both exercise capacity and physical condition, setting up a vicious circle which

Department of Orthopaedic Surgery, Queen's University of Belfast, Musgrave Park Hospital, Belfast BT9 7JB.

S A Henderson, MD, FRCS, Senior Registrar.

R A B Mollan, MD, FRCS, FRCSI, Professor of Orthopaedic Surgery.

Department of Physical Education, Queen's University of Belfast, Belfast BT9 5EX.

Niamh Murphy, MA(Ed), Physical Education Officer.

C A G Boreham, PhD, Director of Physical Education.

Department of Geriatric Medicine, Royal Victoria Hospital, Belfast BT12 6BA.

Olwen E Finlay, MCSP, DMC, SRP, Superintendent Physiotherapist.

D H Gilmore, MRCP(UK), Consultant Physician.

T R O Beringer, MD, FRCP, FRCPI, Consultant Physician.

Correspondence to Mr Henderson.

may eventually jeopardise their capacity for independent living.[2] These functional consequences of this decline in physical activity are an important public health issue.[3]

Regular exercise in the elderly has beneficial effects on cardiovascular fitness,[4, 5] skeletal muscle strength,[6] bone mass,[7, 8] glucose tolerance, flexibility and general wellbeing.[9] Most of these studies have been carried out with elderly subjects who are in good health, but similar improvements in elderly individuals with chronic disease have also been demonstrated.[4]

The ability to produce improvements in fitness and activity levels in elderly patients following hip surgery using an exercise programme would be of major significance, especially if it resulted in prolonged independence and an improved quality of life. A study was therefore undertaken to monitor the effects of an exercise programme in elderly women following hip surgery to ascertain if beneficial improvements in fitness and functional capacity occurred. In addition, a submaximal cycle ergometer test and a self-selected walking speed test were compared as methods of assessing the fitness of this group.

METHODS

Elderly female subjects who had previously undergone surgery for osteoporotic hip fracture or hip replacement for primary osteoarthritis and who were living independently in the community were recruited from hospital orthopaedic clinics. Subjects were excluded if there was severe pre-existing illness. After informed consent subjects were enrolled in a twice-weekly exercise programme for six months.

Exercise programme

The exercise programme was developed by chartered physiotherapists skilled in the care of the elderly and took place in the physiotherapy department of the geriatric day hospital, Royal Victoria Hospital. The emphasis was on weight-bearing activities. After an initial warm-up period of supervised walking, stair climbing, flexibility and active weight-bearing activities, the intensity of exercise increased and subjects took part in aerobic type dance routines accompanied by music. This was followed by a cool-down period which involved stretching and balance exercises. Between each routine a short relaxation period was permitted, the duration of which decreased as fitness improved. The length of the exercise class increased from 25 minutes to 45 minutes during the six months of the study.

Fitness tests

A modified physical work capacity test was undertaken by all subjects at the commencement and completion of the six-month exercise training period on an electronically braked cycle ergometer (Seca, Germany). Heart rate was measured by telemetry (Polar Electro, Finland) with the heart rate of the last 10 seconds in each workload taken as the steady state. Skinfold thickness was measured at four sites using Harpenden calipers, and percentage body fat and lean body mass calculated.[10] Following an habituation period subjects rested for 10 minutes before commencing the test. To allow for the effect of the decline in maximal heart rate with increasing age[11] and to adhere to safe guidelines for submaximal

exercise, physical work capacity was calculated at 85% of the age-related maximal heart rate (PWC 85%).[12] The initial workload in watts was determined at 75% of lean body mass[13] and set to the nearest 5 watts. Subsequent workloads were set as described elsewhere[12] and estimated maximum oxygen uptake (VO_2 max) calculated.[14]

Walking speed was measured at 0, 3, 6 and 12 months on a standard flat 64 metre carpet and vinyl course. Patients were instructed to complete the distance at a comfortable walking speed and timed using a hand held stop-watch. All walking speed measurements were undertaken prior to the group exercises. Results were analysed using analysis of variance techniques and paired t-tests.

RESULTS

A total of 28 elderly female subjects were enrolled, 26 completed the 6 month exercise programme and 22 attended the 12 month assessment of walking speed. During the study period one subject withdrew because of intercurrent illness and a second because of the onset of hip pain in the unoperated side. Three subjects were unable to complete the 12 month assessment because of illness and a fourth declined. At initial attendance four subjects employed walking sticks for indoor and outdoor mobility, and at completion all subjects were able to walk without aids.

The 26 subjects had a mean age of 71·5 (range 63 – 82) years, mean height 159 cm (range 147 – 170), mean weight 64·15 kg (range 45 – 91) and mean body fat of 28·2% (range 23 – 37). No significant change in body fat or body weight occurred between enrolment and completion of the exercise programme. Thirteen of the subjects had received total hip replacement (mean 12·5 months previously) and 13 internal fixation of the femoral neck (mean 12·2 months previously). There were no significant differences in the above variables between the two groups. Compliance with the study was excellent with an overall attendance rate of 88%.

The mean walking speed on entry to the study for all 26 subjects was 0·82 m/sec, after three months 1·23 m/sec and after six months 1·49 m/sec (Figure). At 12 months the mean walking speed for the 22 subjects was 1·31 m/sec. There was thus a 50% increase in mean walking speed between entry and three months and a further 21% increase between three and six months. The increase in walking speed at three, six and 12 months in comparison to that on entry was significant ($p < 0·0001$).

The total hip replacement subjects had a higher walking speed on entry to the study than the fracture group ($p < 0·05$). Both groups had a significant increase in walking speed during the first three months and the difference between the groups was maintained. By six months a further increase in walking speed had occurred but there was no longer a significant difference between the groups. The improvement in walking speed in both groups was maintained at twelve months.

Heart rate was monitored for each subject during a typical exercise session. On average 88·7% of total exercise time was spent at heart rates equivalent to greater than 50% of maximum heart rate (220 beats/min minus age in years), and 26·4% of time at greater than 70% of maximum heart rate.

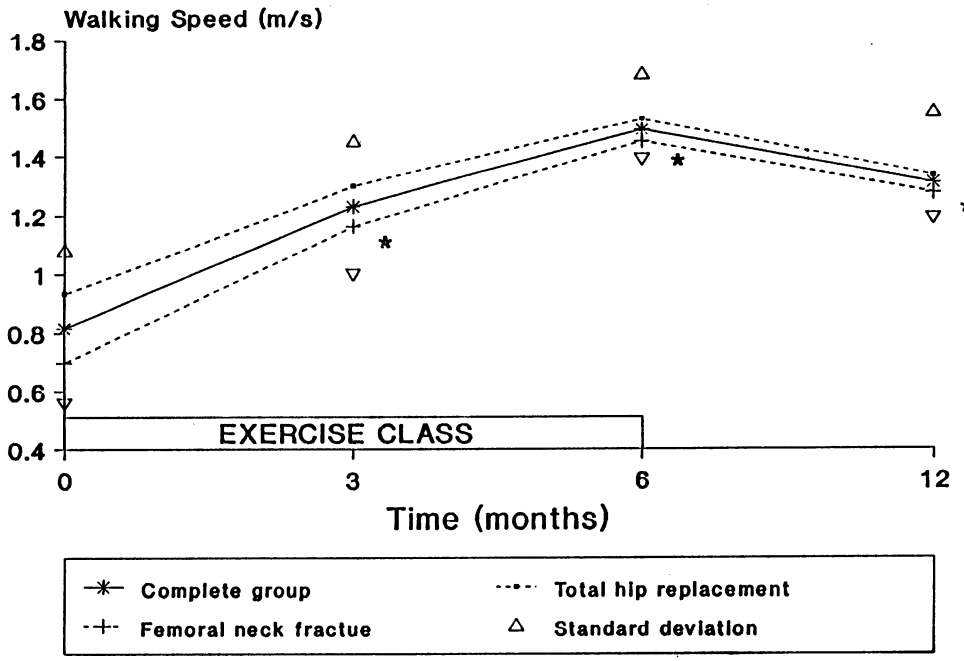


Figure. Walking speed and exercise * $p < 0 \cdot 001$ in comparison to walking speed at entry.

Three parameters of fitness were determined from the bicycle ergometer test, namely physical working capacity equivalent to 85% of age related maximum heart rate (PWC 85%), estimated VO_2 max (mls/kg/minute) and exercise duration (mins). The results of the pre and post-training tests are given (Table). Due to hip pain and abnormal heart rate responses, only half of the subjects completed the cycle ergometry workloads satisfactorily. There was no significant difference in physical working capacity or estimated maximum oxygen uptake between pre and post-training tests. There was, however, a significant increase in test duration from pre to post-training ($p = 0 \cdot 001$). A total of 46% of subjects completed three workloads at the pre-training test, which increased to 89% at the post-training test.

TABLE

Fitness changes (cycle ergometry); mean values (± SD) before and after completion of exercise programme

	n	*At entry*	*At 6 months*
85% physical work capacity (watts)	13	63·23 (±27·7)	50·00 (±17·9)
Estimated maximum oxygen uptake (ml/kg/min)	20	21·28 (±7·1)	19·18 (±5·9)
Exercise duration (mins)	26	4·8 (±2·1)	5·77 (±0·7)*

* $p < 0 \cdot 0001$ compared with duration at entry.

DISCUSSION

The exercise classes were perceived as enjoyable by the subjects and this was reflected in the excellent compliance rate of 88% attendance overall. Subjects felt fitter, were more able to carry out their normal daily activities and enjoyed the opportunity for social contact.

Objective benefit was demonstrated by the improvement in walking speed, which was sustained six months after completion of the exercise programme. At the entry to the study only three of the 26 subjects were able to achieve the minimum recommended walking speed of 1·07 metres/second required to negotiate a "pelican" road crossing,[15] while on completion of the exercise period 25 of the 26 subjects were able to exceed the requirement. Uncorrected walking speed has been shown to be a reliable clinical method of monitoring gait rehabilitation for hospitalised elderly.[16] On entry to this study the mean walking speed of 0·82 m/sec was lower than the mean speed of 1·16 m/sec of a group of elderly females living independently with a similar mean age of 72 years[17] and also of an older group of 79 year old Swedish women with a mean walking speed of 0·92 m/sec.[18] This low figure and the subsequent substantial improvement in walking speeds of this group who had received hip surgery and conventional rehabilitation over a year prior to entry into the trial demonstrates the failure of present post-operative rehabilitation to achieve maximal recovery. While a small improvement of 8% in walking speed may occur in the second year after hip arthroplasty,[19] the improvement in walking speed of 45% at the end of the six-month exercise period in this study was substantial and too great to be due to natural recovery alone. These surprisingly large improvements in walking speed were achieved with a twice weekly exercise programme with most improvement occurring after three months and sustained at 12 months. The parameters to which these improvements may be attributed are unclear and merit further investigation, as does the duration and frequency of exercise necessary to elicit health and cardiovascular fitness benefits in the elderly.[20]

It has been calculated that for the average 70 – 75 year old female, walking at 1·38 m/sec is maximum aerobic exercise,[3, 21] and it is of interest that the mean walking speed achieved at completion of the study of 1·49 m/sec exceeded this. Although the exercise involvement in this study falls short of the recommended guidelines for developing and maintaining cardio-respiratory fitness in healthy adults (of at least 20 minutes training, three days per week at 60 to 90% of maximum heart rate), our regimen may be an appropriate and achievable exercise protocol for elderly people.

In contrast to the walking speed results we were unable to demonstrate a significant improvement in physical work capacity or oxygen uptake. This reflects methodological problems including hip pain associated with the cycling action, and erratic heart rate response to exercise contributing to reduced sensitivity of cycle ergometry as an accurate measure of fitness changes in the elderly. There was, however, a significant increase in the exercise duration undertaken on the cycle ergometry testing on comparing the initial and post-training measurements. The correlation between self-selected walking pace and maximum oxygen uptake has been found to be not significant in elderly subjects.[22] It has also been stated that exercise training in the elderly will increase submaximal performance to a

greater degree than any changes in maximum oxygen uptake,[23] which may explain the improvements in cycling duration and walking speed without corresponding improvements in physical work capacity. Other studies have shown that training reduces the stress imposed by submaximal exercise.[24] Such improvements are highly relevant to everyday life for an elderly population as most activities of daily living require submaximal response. Observed improvements in submaximal function may have been due to improvements in flexibility and muscle strength which reportedly occur with exercise.[24, 25, 26, 27] Similarly, exercise programmes have been reported to result in improved muscle strength and mobility even in the very elderly and may in part be due to muscle hypertrophy as well as improved neural recruitment patterns.[6] These parameters were not assessed in this study and we were unable to identify the physiological changes contributing to improvements in walking speed and submaximal performance.

The bicycle ergometer test was found to be an insensitive measure of improved submaximal function in this group of elderly subjects. Given its substantial cost in terms of time and equipment, the simpler measurement of walking speed is preferable.

This study indicates the major benefits which a wider provision of resources for promotion of exercise in the elderly with musculo-skeletal problems could provide. These improvements were particularly noteworthy as the seventh decade is thought to be the critical age for accelerated decline in function.[23] Many elderly women are not achieving their full potential following hip surgery for osteoporotic fracture or osteoarthritis and would benefit from further rehabilitation.

We wish to acknowledge the considerable assistance of the elderly volunteers as well as the physiotherapy department and day hospital staff, and the Red Cross drivers who assisted in completion of this study. In particular we wish to thank Jacqueline Crosby and Hilary Preston for their enthusiasm and contribution to the exercise class.

REFERENCES

1. Lewis AF. Fracture of neck of the femur: changing incidence. *Br Med J* 1981; **283**: 1217-20.
2. Bassey EJ. Age, inactivity and some physiological responses to exercise. *Gerontology* 1978; **24**: 66-77.
3. Editorial. Physical activity in old age. *Lancet* 1986; ii: 1431.
4. Morey MC, Cowper PA, Feussner JR, *et al.* Evaluation of a supervised exercise program in a geriatric population. *J Am Geriatr Soc* 1989; **37**: 348-54.
5. Smith WDF. Fitness training for the elderly: Canadian experience. *Geriatr Med* 1989; **Vol 19,** (No 11): 55-61.
6. Fiatarone MA, Marks EC, Ryan ND, Meredith CN, Lipsitz LA, Evans WJ. High-intensity strength training in nonagenarians. *J Am Med Assn* 1990; **263**: 3029-34.
7. Smith EL, Reddan W. Physical activity — a modality for home accretion in the aged. *Am J Roentgenol* 1976; **126**: 1297.
8. Smith EL Jnr, Reddan W, Smith PE. Physical activity and calcium modalities for bone mineral increase in aged women. *Med Sci Sports Exerc* 1981; **13**: 60-4.
9. Lampman R. Evaluating and prescribing exercise for elderly patients. *Geriatrics* 1987; **8**: 63-5, 69-70, 73-6.
10. Durnin JV, Rahaman MM. The assessment of the amount of fat in the human body from measurements of skinfold thickness. *Br J Nutr* 1967; **21**: 681-9.

11. Astrand PO, Rodahl K. Textbook of work physiology. Physiologic bases of exercise, 2nd edition. New York: McGraw-Hill, 1977: 349-53.

12. Cooke CB. Welsh heart programme directorate. Clinical survey manual. Heart Beat Report 1986, No 3, Cardiff.

13. Council of Europe Eurofit. European test of physical fitness. Council of Europe Committee for the Development of Sport (1988) Rome.

14. Shephard RJ (ed). Endurance fitness, 2nd edition. Toronto: University of Toronto Press, 1977.

15. Department of transport, highways and traffic: A Departmental Standard. TD 28/87; 1987; 4-5.

16. Friedman PJ, Richmond DE, Baskett JJ. A prospective trial of serial gait speed as a measure of rehabilitation in the elderly. *Age Ageing* 1988; **17**: 227-35.

17. Bendall MJ, Bassey EJ, Pearson MB. Factors affecting walking speed of elderly people. *Age Ageing* 1989; **18**: 327-32.

18. Lundgren-Lindquist B, Aniansson A, Rundgren A. Functional studies in 79 year olds. III, walking performance and climbing capacity. *Scand J Rehab Med* 1983; **15**: 125-31.

19. Olsson SS, Jernbergr A, Tryggö D. Total hip replacement with a straight stem prosthesis: a 2-year follow up of 126 CAD operations. *Acta Orthop Scand* 1984; **55**: 146-51.

20. American College of Sports Medicine position stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. *Med Sci Sports Exerc* 1990; **22**: 265-74.

21. Saltin B. Fysisk Praestationsevne. Aerob Kapacitel. Manedss Krift Prakt. *Laeg* 1980; **58**: 193-216.

22. Cunningham DA, Rechnitzer PA, Pearce ME, Donner AP. Determinants of self-selected walking pace across ages 19 to 66. *J Gerontol* 1982; **37**: 560-4.

23. Cunningham DA, Patterson DH. Discussion exercise, fitness and ageing in exercise fitness and health. A consensus of current knowledge. In: Bouchard C, Shephard RJ, Stephens T, Sutton JR, McPherson BD (eds). Human Kinerics Publ, Campaign 1990.

24. Benestad AM. Trainability of old men. *Acta Med Scand* 1965; **178**: 321-7.

25. De Vries H. Exercise intensity threshold for improvement of cardiovascular-respiratory function in older men. *Geriatrics* 1971; **26**: 94-101.

26. Chapman EA, De Vries HA, Swezey R. Joint stiffness: effects of exercise on young and old men. *J Gerontol* 1972; **27**: 218-21.

27. Sidney KH, Shephard RJ, Harrison JE. Endurance training and body composition of the elderly. *Am J Clin Nutr* 1977; **30**: 326-33.

The Ulster Medical Journal, Volume 61, No. 2, pp. 151 – 156, October 1992.

The Belfast hip screener: from infancy to maturity

W G Kernohan, Bernadette P Trainor, Pauline E Haugh, Adrienne F Johnston, R A B Mollan

Accepted 21 September 1992.

SUMMARY

Hip dislocation remains the most significant childhood orthopaedic abnormality despite the efforts of neonatal screening, first described in 1910. A new method of enhancing the performance of screening, the Belfast hip screener, is a non-invasive device developed to detect and interpret the vibrations ("clicks" and "clunks") *which are emitted as the hips are physically tested. A progress report is presented covering ten years' work from early records made with tape recorders to modern methods of digital signal processing.*

INTRODUCTION

Developmental dislocation of the hip is a term covering a spectrum of childhood hip disease ranging from instability at birth to established dislocation. In Northern Ireland the disease affects two infants per thousand live births, though the incidence varies with sex, family history and environmental factors such as posture in utero, delivery, and nursing habits.[1, 2, 3] Formerly called 'congenital', this term should be dropped from common usage as it excludes the possibility of late development.[4, 5] In developmental hip dislocation the head of the femur while reducible is unstable, or it lies outside its normal position which is within the acetabulum. Early detection and immediate treatment of the condition in an abduction splint generally result in normal development without admission to hospital.[1, 6] However, almost 50% of cases present late (beyond three months of age), necessitating extensive treatment, including traction and repeated surgical intervention, which does not guarantee a successful outcome.[7]

The distinguished French orthopaedic surgeon Dr Pierre le Damany was among the first to describe a physical test to achieve early diagnosis.[8] He developed the clinical examination of the neonatal hip by two manoeuvres. The first was simply

Department of Orthopaedic Surgery, The Queen's University of Belfast, Musgrave Park Hospital, Belfast BT9 7JB.

W G Kernohan, PhD, Lecturer.

R A B Mollan, MD, FRCS, FRCSI, Professor of Orthopaedic Surgery.

Musgrave Park Hospital, Belfast BT9 7JB.

Bernadette P Trainor, SRN, Research Staff Nurse.

Pauline E Haugh, SRN, Research Staff Nurse.

Adrienne F Johnston, RGN, Research Staff Nurse.

Correspondence to Dr Kernohan.

a modification of the 'telescoping' manipulation in which the femur is moved along its length without rotation. The second described the exact placing of the examining hand and the stabilisation of the pelvis, as well as the control of the greater trochanter by the middle finger, while the femoral head was gently lifted in an out of the acetabulum (Figs 1, 2). Notice his emphasis on positioning and the description of a *"ressaut"* (spring-back) when the hip reduces. In 1935 Ortolani[9] devised a similar test to detect dislocated hips noting audible and palpable vibration events during testing. Barlow,[10] in 1962 amended this manoeuvre so that those hips that dislocate can be detected, though, in fact, he added little to the tests described by le Damany.

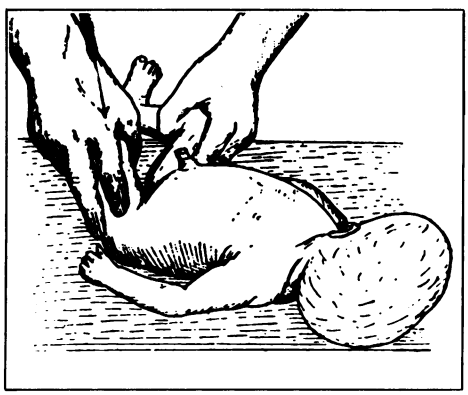


Fig 1. Position des mains de l'opérateur et position de l'enfant dans la recherche de la hanche subluxable (le Damany, 1910).

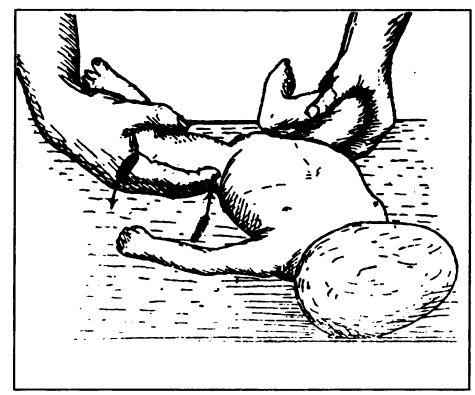


Fig 2. En portant la cuisse dans la position de flexion-abduction, la subluxation se reduit avec un ressaut (le Damany, 1910).

PILOT STUDIES

The use of miniature accelerometers to detect neonatal hip vibration during screening for developmental dislocation of the hip has been carried out in Belfast since 1980.[11] The original vibration technique based on analogue amplifiers and tape recording allowed these tests to be analysed in terms of detection and interpretation.[12] The first trial of the system was carried out on children referred to a research clinic.[13, 14] The study involved selected groups totalling 306 selected neonates, referred for vibration arthrometry. The largest subset (N = 217), was already diagnosed with 'clicky' hips. On testing, a total of over 600 vibration episodes were captured for analysis. To be certain of the clinical outcome the children underwent a long follow-up with several attendances over a four year period. Abnormal vibrations indicating instability had a high vibration level with low frequency. Spurious movements of the hip produced vibration with a low level and low frequency whereas a safe click had a low level of vibration and a higher frequency. Several infants went on to require treatment. When the records were examined retrospectively it was clear that the unstable hips were signalled at birth by low frequency, high amplitude vibration emissions. The study included a group of normal, clinically silent, hips, which also produced some vibration during testing, though on analysis these were entirely different vibrations. Using vibration arthrometry it was possible to predict or classify vibration detected from new hips

with unknown pathology. The result of testing this classification system suggested a sensitivity of 75%, a specificity of 99%, a positive predictive value of 86% and a negative predictive value of 99% ($p < 0{\cdot}005$). The technique of vibration arthrometry was proposed not only to facilitate hip screening, but also to assist with careful monitoring of clicky hips. The results of the trial permitted the Belfast hip screener to be updated, giving a preliminary diagnosis of hip disorders at the time of testing.

The analogue system of recording vibrations in developmental dislocation of the hip with its separate recording and later lab-based analysis stage, was a useful one for basic work and provided flexibility. However, it became clear that a more convenient and portable system would have advantages, particularly for less computer-literate users. One of the first attempts to reduce the system complexity involved a modified Apple microcomputer, which was programmed to record hip vibrations digitally during testing.[15, 16] The computer then acted both as a vibration datalogger and as an analyser. There were three phases of the software: capture, display and analysis. This system has come to be known as the Belfast hip screener and has allowed larger trials to be performed.

POPULATION STUDIES

A large-scale trial of vibration arthrometry for hip screening was carried out on a group of 3000 cases during December 1984 — December 1985 with final follow-up from 1988 to 1990. The aim was to evaluate the potential of vibration arthrometry in early detection of developmental dislocation of the hip on a representative group of neonates chosen at random from five maternity hospitals serving counties Antrim, Down and Tyrone. Since significant hardware development took place during the trial, not all vibrations were recorded in the same fashion. The initial 500 cases were recorded on analogue reel-to-reel tape.[17] The method proved valuable as it was possible to elicit vibration events from approximately one quarter of all the hips tested; vibrations were recorded from some normal hips, from all "clicky" hips, and from all unstable hips. The vibrations were measured and the differences between normal and "clicky" hips were statistically significant. The remainder have been categorised and detailed analysis is underway.

After further development of the computerised system, a clinical study of 300 neonates focused on the ability to detect hip vibration manually in a comparative study of vibration arthrometry with existing maternity screening practice. Over half (185) produced hip vibration during testing, including several cases who later received treatment. This random study of babies showed the value of vibration arthrometry in early detection of developmental dislocation of the hip, as very few hip vibrations (7%) were found by routine examination by a doctor. An experienced examiner, the research nurse, recorded 86% of the vibrations using the method of vibration arthrometry. It was concluded that objective screening by vibration arthrometry would detect a higher proportion of vibration events in neonates, during the early stages of developmental dislocation of the hip.

DISCUSSION

More widespread implementation of vibration arthrometry using this hip screener would warrant an economic appraisal. Twelve patients, who had recently received

hip replacement surgery as a result of late diagnosis of developmental dislocation were reviewed, and all their costs were identified, although no estimates were included for the pain and suffering endured by the patients.[18] We estimate that screening with the Belfast hip screener would be successful if it were able to raise the detection of developmental dislocation of the hip at birth from 50%, as at present, to 63%, with additional screening using ten teams of nurses examining every neonate in Northern Ireland.

Changes and improvements in treatment over the years, particularly antibiotic therapy, have reduced the hospital stay needed for corrective procedures. Therefore a study of the cost of treatment in a more recent cohort of cases of developmental dislocation of the hip was undertaken.[19] The potential cost-saving of screening, by examining the costs in 36 late cases born in 1980, was found to be £6,674 per case. This included all the costs of treatment up to 1991. While treatment costs have fallen, significant savings are still possible is cases are detected early because the cost of treatment in a nappy splint amounts to just £150. Of course, there are less quantifiable costs for the individuals diagnosed late; principally the lifetime pain and suffering resulting from failure to diagnose, but also loss of earnings.

The use of vibration arthrometry in practice depends on the method of discrimination, the ability to distinguish normal from abnormal. Decision analysis has been used to provide a discriminant method based on prior studies and this has been incorporated into the computer programme.[20] Furthermore the technique

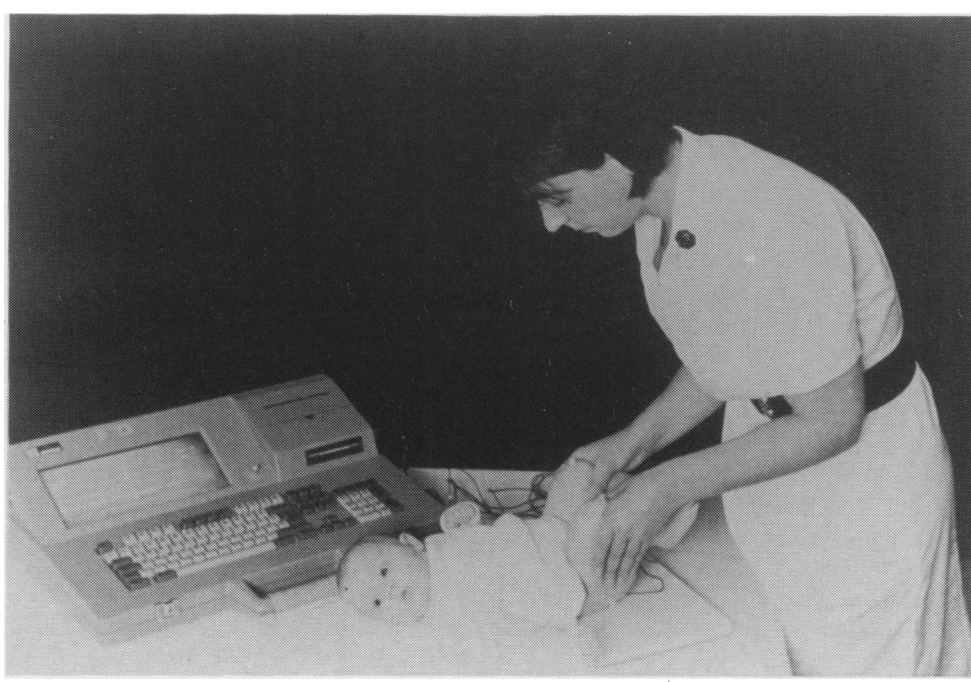


Fig 3. Vibration arthrometry is used in early detection of dislocated and dislocatable hips by recording and analysing vibrations produced during testing of the hip. The nurse is seen operating the Belfast hip screener.

depends on careful, reproducible expert manipulation of the neonatal hip, and current work has introduced additional sensors to measure applied force, angles, and displacement of the hip during testing. In this way the execution of the hip test can be monitored and standardised. Increased reproducibility amongst our population of responsible examiners will follow.

In order that more extensive clinical trials may be performed, the Belfast hip screener was redesigned (Fig 3) to meet British Standards (BS5724) and this was possible only with commercial support from the local electronics industry (Systems Solutions Ltd., 75 Belfast Road, Carrickfergus, N. Ireland). Eventually the benefits of this form of screening may be made widely available, and a marketing strategy devised in an attempt to achieve an early diagnosis of neonatal hip dislocation beyond the confines of Musgrave Park Hospital. Developmental dislocation of the hip is one of the biggest problems in paediatric orthopaedics. Without doubt, vibration arthrometry is not the complete solution, but there is evidence that important new information has become available using the technique. This has gained international recognition, and economic analysis has further shown that it may be implemented at little or no cost to the national health service.

REFERENCES

1. Bennett JT, MacEwen GD. Congenital dislocation of the hip. Recent advances and current problems. *Clin Orthop* 1989; **247**: 15-21.
2. Burke SW, Macey TI, Roberts JM, Johnston III C. Congenital dislocation of the hip in the American Black. *Clin Orthop* 1985; **192**: 120-3.
3. Crossan JF, Wynne-Davies R. Research for genetic and environmental factors in orthopaedic diseases. *Clin Orthop* 1986; **210**: 97-105.
4. Ilfeld FW, Westin GW, Makin M. Missed or developmental dislocation of the hip. *Clin Orthop* 1986; **203**: 276-81.
5. Klisic PJ. Congenital dislocation of the hip — a misleading term: brief report. *J Bone Joint Surg* 1989; **71B**: 136.
6. Morrissy RT, Cowie GH. Congenital dislocation of the hip. *Clin Orthop* 1987; **222**: 79-84.
7. Catford JC, Bennet GC, Wilkinson JA. Congenital hip dislocation: an increasing and still uncontrolled disability? *Br Med J* 1982; **285**: 1527-30.
8. le Damany P. La luxation congénitale de la hanche. Paris: Félix Alcan, 1912.
9. Ortolani M. Un segno poco noto e sua importanza per la diagnosi precoce di prelussazione congenita dell'anca. *Pediatria* 1937; **45**: 129-36.
10. Barlow TG. Early diagnosis and treatment of congenital dislocation of the hip. *J Bone Joint Surg* 1962; **44B**: 292-301.
11. Cowie GH, Mollan RAB, Kernohan WG, Bogues BA. Vibration emission in detecting congenital dislocation of the hip. *Orthop Rev* 1984; **13**: 30-5.
12. Cowie GH, Bogues BA, Kernohan WG, Mollan RAB. A new aid in the diagnosis of congenital dislocation of the hip. *J Bone Joint Surg* 1983; **65B**: 656.
13. Kernohan WG, Cowie GH, Patterson CC, Mollan RAB. Discriminating between innocent clicks and clunks due to dislocation of the neonatal hip. In: Societe Internationale de Recherche Orthopedique et de Traumatologie, Munich, 1987.
14. Kernohan WG, Cowie GH, Mollan RAB. Vibration arthrometry in congenital dislocation of the hip. *Clin Orthop* 1991; **272**: 167-74.

15. Shaw SN, Kernohan WG, Bogues BA, Mollan RAB. An Apple-based screening aid for congenital dislocation of the hip. *Proc Med Microcomput Appl.* Liverpool: University Press, 1985: 23-4.

16. Shaw SN, Kernohan WG, Mollan RAB. The development of a screening aid for congenital dislocation of the hip. In: Technology in health care. Byford GH (ed). Biological Engineering Society, London, 1985: 107-8.

17. Kernohan WG, Trainor BP, Nugent GEM, Walker PE, Timoney M, Mollan RAB. Low frequency vibration emitted from unstable hip in human neonate. *Clin Orthop* (In press).

18. Kernohan WG, Trainor BP, Mollan RAB, Normand CEM. Cost-benefit appraisal of screening for congenital dislocation of the hip. *J Management in Med* 1990; **4**: 230-5.

19. Kernohan WG, Trainor BP, Mollan RAB, Normand CEM. Cost of treatment of congenital dislocation of the hip. *Int J Health Plan Man* 1991; **6**: 229-33.

20. Kernohan WG, Patterson CC, Mollan RAB. Discriminant analysis applied to orthopaedics. In: *Applications of Statistics in Medicine,* Institute of Mathematics and its Applications, Cardiff 1988.

The Ulster Medical Journal, Volume 61, No. 2, pp. 157 – 162, October 1992.

Continuing medical education for general practitioners — a Northern Ireland plan

Agnes McKnight, Karen Mills

Accepted 7 July 1992.

SUMMARY

Following the introduction of the 1990 contract for general practitioners and the new postgraduate education allowance, the Northern Ireland Continuing Medical Education Group, comprising the clinical tutors from four Belfast hospitals and members of the University department of general practice, was established to provide high quality co-ordinated continuing medical education for general practitioners.

A questionnaire was sent to all general practitioners in Northern Ireland to find out their needs and preferences with regard to continuing education. The responses indicate the value of small group discussion as well as the traditional lecture and the important role of local hospital consultants in providing education. Therapeutics, recent advances in medicine and learning new skills are all desirable topics. The group intends to use this information in the provision of courses for general practitioners in the province.

INTRODUCTION

With the introduction in April 1990 of the new general practice contract and the new postgraduate education allowance for general practitioners, the provision of continuing medical education for general practitioners became an open market. Before the introduction of the new contract the regional adviser for general practice to the Northern Ireland Council for Postgraduate Medical Education was responsible for arranging most courses. Now the role of the regional adviser is mainly that of accrediting and monitoring educational courses and also facilitating and promoting high quality education. Anyone can now run a course for general practitioners and charge a fee, but the providers of courses have to apply to the regional adviser for accreditation. General practitioners can qualify for the postgraduate education allowance which is £2,025 each year, provided they attend 25 days of approved and balanced courses in a five year period.

In June 1990 discussions began between members of the department of general practice at Queen's University, the regional adviser in general practice and the clinical tutors from the postgraduate centres under the Northern Ireland Council for Postgraduate Medical Education with the aim of establishing a liaison group

Department of General Practice, The Queen's University of Belfast, 1 Dunluce Avenue, Belfast BT9 7HR.

Agnes McKnight, MD, FRCGP, Senior Lecturer in General Practice.
Karen Mills, BSc, Research Fellow.

Correspondence to Dr A McKnight.

for the provision of high quality education for general practitioners. The clinical tutors were already organising courses for general practitioners in their local hospitals and postgraduate centres. However, the "open market", possible competition between postgraduate centres, and the necessity to include courses covering service management and health promotion in general practice, with which clinical tutors were unfamiliar, made them feel that it was important to liaise more with general practitioners and with each other. Members of the university department of general practice wished to see continuing medical education develop, with the possibility of not only an annual co-ordinated programme but even a five-year programme which might eventually lead to the award of a diploma or degree. The group felt it was a priority to find out what general practitioners wanted with regard to their continuing education, and also to ascertain the degree of support there would be for the annual programme it planned to provide.

METHOD

A questionnaire was devised to ask about the general practitioner's training and experience, the value of various methods of postgraduate education, who should be involved in teaching in a postgraduate centre, and the range of topics to cover. Questions were included on the preferred weekdays, the length of courses, and whether or not they should be residential. Further questions included interest in either an annual or a five-year planned programme, and how much practitioners would be prepared to pay for these. The questionnaire was sent to all 950 general practitioners in Northern Ireland with an accompanying letter explaining the group's aims, and a reply-paid envelope.

RESULTS

Four hundred and ninety four completed questionnaires were received out of the 950 sent (52% response rate). Details of respondents' training and experience in general practice are shown in Table I together with comparable figures for all principals in Northern Ireland. Three hundred and eighteen respondents (64%) had undergone a course of vocational training for general practice.

Of those who replied, 438 (89%) said they would be interested in an annual subscription programme which would provide them with at least five days of

TABLE I

Experience and training

Number of years as a principal in general practice	*This study: Number of respondents (%)*	*Northern Ireland 1991*
> = 30	38 (8)	152 (16)
> = 20 and < 30	61 (12)	141 (15)
> = 10 and < 20	156 (32)	277 (29)
< 10	234 (47)	380 (40)
Not answered	5 (1)	—
Total	494	950

balanced education chosen from a Northern Ireland accredited course calendar, and 404 (82%) said they would be interested in a five year planned programme of continuing education covering important areas of general practice, delivered in modules providing at least five days of education each year. The response to the question on the value of the different forms of education is shown in Table II. Seminars and symposia were judged to be the most valuable, and tape/slide or video programmes the least valuable. Distance learning, small group work and workshops were mentioned by a few respondents. The response to the question on the importance of various people as teachers is shown in Table III. Local consultants were judged to have a very important role. Consultants and general practitioners with a national reputation, and local general practitioners were also judged to be important. The respondents thought that non-medical academics, social workers, nurses or drug firm representatives were less effective as teachers of general practice.

TABLE II

Responses to the question on the value of different forms of education

	Very valuable	*Moderately valuable*	*Little or no value*	*Uncertain*
Seminar	152 (30%)	250 (50%)	70 (14%)	22 (4%)
Symposium	147 (29%)	319 (64%)	21 (4%)	7 (1%)
Clinical attachment	119 (24%)	232 (46%)	41 (8%)	102 (20%)
Lecture	117 (23%)	329 (66%)	46 (9%)	2 (0%)
Case conference	87 (17%)	289 (58%)	89 (18%)	29 (5%)
Ward round	53 (10%)	200 (40%)	137 (27%)	104 (21%)
Tape/slide programme	51 (10%)	322 (65%)	102 (20%)	19 (3%)
Video programme	15 (3%)	298 (60%)	163 (33%)	18 (3%)

TABLE III

The importance of various people as teachers at a postgraduate centre for general practice

	Very important *No. (%)*	*Moderately important* *No. (%)*	*Little or no importance* *No. (%)*
Local consultant	307 (62%)	179 (36%)	8 (1%)
Local general practitioner	208 (42%)	240 (48%)	46 (9%)
National consultant	207 (41%)	246 (49%)	41 (8%)
National general practitioner	174 (35%)	244 (49%)	76 (15%)
Nurse	41 (8%)	309 (62%)	144 (29%)
Non-medical academic	31 (6%)	304 (61%)	159 (32%)
Social worker	18 (3%)	257 (52%)	219 (44%)
Pharmaceutical representative	15 (3%)	200 (40%)	279 (56%)

The response to the question on the importance of different topics in general practice education is shown in Table IV. Therapeutics, clinical topics, learning new skills and practice management were considered to be very important by the majority.

TABLE IV

The importance of different topics in postgraduate education for general practice

	Very important *No. (%)*	*Moderately important* *No. (%)*	*Little or no importance* *No. (%)*
Therapeutics	299 (60%)	192 (38%)	3 (0%)
Clinical topics	276 (55%)	213 (43%)	5 (1%)
Learning new skills	260 (52%)	229 (46%)	5 (1%)
Practice organisation/management	258 (52%)	217 (43%)	19 (3%)
Recent advances/new technology	241 (48%)	238 (48%)	15 (3%)
Case presentations	153 (31%)	311 (62%)	30 (6%)
Health promotion	150 (30%)	298 (60%)	46 (9%)
Medical/social problems	118 (23%)	336 (68%)	40 (8%)
Medical ethics	102 (20%)	312 (63%)	80 (16%)
The team	79 (16%)	296 (59%)	120 (24%)

As 234 (47%) of replies came from doctors who had been principals in general practice for less than 10 years we compared their responses to certain questions with those of their colleagues who had been principals for 10 years or more. Those who had been principals for less than 10 years showed more interest in participating in both an annual programme (X^2 $p = 0{\cdot}001$) and a five year planned programme (X^2 $p = 0{\cdot}01$). They were also significantly less likely to regard lectures ($p = 0{\cdot}05$) or ward rounds ($p = 0{\cdot}05$) as of value, or to include national consultants as teachers in a postgraduate centre. A significantly greater number ($p = 0{\cdot}01$) considered it to be advantageous to include local general practitioners as teachers. They also considered education on practice organisation or management to be a very important topic.

Table V shows days on which doctors preferred to attend meetings. The least popular time for a postgraduate meeting was a Monday morning. Wednesday and Thursday afternoons were the most convenient. Single weekday meetings were very popular with all respondents. Half of the general practitioners liked longer meetings lasting between two and five days and just over one third liked residential or non-residential weekend meetings. Only 75 respondents (15%) liked to attend residential five day courses and less than half were interested in evening working sessions at a residential course. If residential meetings were chosen the vast majority of respondents preferred to stay at a hotel.

Two hundred and seventy respondents (55%) stated they would be prepared to pay between £200 and £300 for a programme of 10 half-days of postgraduate education either as an annual fee, or in annual instalments for a five year plan. There was no significant difference between doctors who had been principals for less than 10 years or for 10 years or more.

TABLE V

Days of the week on which general practitioners would find it convenient to attend postgraduate meetings

	No.	*%*
Monday am	48	(9%)
Monday pm	87	(17%)
Tuesday am	102	(20%)
Tuesday pm	217	(43%)
Wednesday am	145	(29%)
Wednesday pm	280	(56%)
Thursday am	119	(24%)
Thursday pm	264	(53%)
Friday am	87	(17%)
Friday pm	128	(25%)
Saturday am	120	(24%)
Saturday pm	80	(16%)
Sunday am	80	(16%)
Sunday pm	92	(18%)

DISCUSSION

Providing continuing education for general practitioners is not an easy task. They are a disparate group ranging in age from the late twenties to seventy years old, from being vocationally trained to having started the hard way directly after registration. Some are interested in continuing medical education, some see it mainly in financial terms. To suit all participants in one type of course would be an impossibility. We plan to provide a variety of courses lasting between one and five days using different educational techniques in several different centres throughout the province. We shall offer choices within courses and be prepared to run programmes for a small number of participants. General practitioners' own experiences are a rich resource for learning, and they may increase their knowledge more effectively through experimental techniques such as discussion or problem solving.

We will use all the educational methods mentioned but at the same time try to develop the small group discussion, the clinical attachment, video case analysis and learning of new practical skills. It is reassuring for hospital doctors to see how much they are valued as teachers by general practitioners. We would endeavour to integrate continuing medical education in hospitals and general practice. We envisage that hospital doctors and general practitioners can learn together on topics relevant to both groups. It is reassuring to see that general practitioners, especially the younger ones, feel they have a lot to offer each other in educational terms, but disappointing that nurses and social workers are felt to be of little or no importance. We shall try to address this by incorporating all the members of

the primary care team into our courses, including physiotherapists, speech and occupational therapists, clinical psychologists, dietitians and chiropodists. They are all very valuable people in the primary care setting with a wealth of knowledge to share with their colleagues if given the appropriate opportunity.

Disease management usually figures very highly on a list of topics. This is only to be expected as general practitioners are very aware of what they need to learn, which is generated by the real tasks and problems in everyday clinical practice. They wish to apply newly acquired skills or knowledge to their immediate circumstances, and as educators we must recognise this. If we are to succeed in our objectives we must keep our courses relevant to the needs of the participants. Service management is also felt to be very important by a large number of the general practitioners, especially the younger group who can accept change more easily. The introduction of the new contract has obviously influenced this response. Health promotion, although a requirement for a balanced programme, is still not felt to be important by many practitioners. It will be interesting to see, if health promotion develops as a part of the general practitioner's work, how this will affect its perceived importance in the educational scene.

Analysis of the response to this questionnaire encouraged the group to pursue its aim of setting up an office to provide continuing medical education for general practitioners. The Queen's University of Belfast was persuaded to fund the project for a period of three years by which time the organisation should be able to support itself from subscriptions paid by the practitioners. An office has been established within the Department of General Practice, staffed by a course manager and secretary. Since September 1991 we have run three five-day courses, (in the Belfast City, Royal Victoria and Ulster Hospitals), one three-day course in the Department of General Practice and several one-day symposia. These have been successful judging by the comments on the evaluation forms issued to all participants. We have sent out an annual subscription programme to continue from April 1992 until the end of March 1993 which will give general practitioners a wide choice of topics, dates and venues so that they can plan the time for their education well in advance. We also intend to produce a computerized database which will hold a record of the courses attended by each subscriber. In the longer term the group's aim will be to provide a five-year curriculum of continuing medical education for interested general practitioners, giving them an opportunity for further development and achievement. A further advantage of a five-year curriculum might become evident in the event of the introduction of "re-accreditation" of general practitioners in the United Kingdom.

We would like to thank Professor Robin Shanks who stimulated the establishment of the group, Professor Roy McClelland, Dr Ben Moran and Dr Brew Atkinson who were involved in the initial discussions, and Professor Philip Reilly, Mr Terry Irwin, Dr Ken Wilson, Dr Paul Weir and Dr Patrick Bell who provide programmes of education for general practitioners with continuing enthusiasm. Finally we would like to thank Arlene Stockman, Course Manager and Margaret McAnulty, Course Secretary for their dedication, hard work and enthusiasm for the project.

The Ulster Medical Journal, Volume 61, No. 2, pp. 163 – 165, October 1992.

Five cases of *Alcaligenes* pseudobacteraemia

J R Kerr, C H Webb

Accepted 7 July 1992.

SUMMARY

A cluster of five cases of pseudobacteraemia due to the organism Alcaligenes denitrificans *occurred in three hospital medical wards over a four week period. The same organism was isolated from four of twelve commercially prepared bottles for erythrocyte sedimentation tests. The most likely explanation for the outbreak is that the ESR bottles were filled prior to inoculation of blood culture bottles. The outbreak was brought to an end by advising on correct procedure.*

INTRODUCTION

Pseudobacteraemia means false – positive bacteraemia, and it implies contamination of the blood culture medium, usually from an environmental source. As opportunistic pathogens become more common it is increasingly difficult to distinguish pseudobacteraemia from true pathogens in blood culture. We describe a cluster of five cases of pseudobacteraemia due to the organism *Alcaligenes denitrificans* over a four week period, occurring in three adjacent hospital medical wards. A case cluster can suggest an outbreak of infection, but since this organism is known to exist in the environment an investigation to identify a possible source was carried out.

METHODS

Inoculated blood culture bottles are incubated at 37°C and sampled for bacterial growth at intervals using a semi-automated blood culture system. If growth is detected, a sample of the blood culture is aseptically withdrawn from the bottle for gram staining and subculture on solid media. Routinely, these plates are also incubated at 37°C, but once an organism suspected of being an *Alcaligenes* species was isolated, culture plates were incubated at 30°C, which improved recovery.

Informal discussions with junior medical staff failed to reveal any breach of blood sampling protocols. In view of previous reports of pseudobacteraemia traced to contamination of the bottles used for full blood picture and erythrocyte sedimentation rate tests, we tested four of each of these bottles from each of the three medical wards involved. The haematology bottles tested are commercially manufactured by Sherwood Medical, UK. They are made of glass and each contains 0·5 ml sodium citrate. They are designed to take 4·5 ml blood and are

Department of Bacteriology, Royal Group of Hospitals, Belfast BT12 6BA.
J R Kerr, BSc, MB, Registrar in Bacteriology.
C H Webb, MB, BDS, FRCPath, Consultant Bacteriologist.
Correspondence to Dr Kerr.

gamma-irradiated at the end of production to ensure sterility. The ESR bottles tested are commercially manufactured by Sterilin, UK. They are plastic and each contains 0·4 ml of sodium citrate. They are designed to take 2 ml blood and are prepared under aseptic conditions although not irradiated. From both the haematology and the ESR bottles, 20 µl of contained sodium citrate was plated on to blood and MacConkey agars. A gentamicin disk was placed on each plate to screen for any gentamicin-resistant, gram-negative organisms, since the *A. denitrificans* stain had consistently exhibited this characteristic.

Antibiotic sensitivity testing was performed using the Stokes method with *Pseudomonas aeruginosa* National Collection of Type Cultures 10662 (Central Public Health Laboratory, Colindale Avenue, London) as a control organism. Isolates were incubated on blood agar at 30°C for 48 hours. Identification was performed using the API 20NE system (BioMerieux, France) for non-enteric gram-negative bacilli. This is a commercially prepared, semi-automated method using a plastic strip containing twenty capules; the capules contain the necessary reagents for twenty different biochemical tests. A suspension of the test organism is inoculated into each of the capules and the results read after forty-eight hours.

RESULTS

All five strains isolated from blood cultures had the same biochemical profile on testing with the API 20NE system, and were identified as *Alcaligenes denitrificans*. All had a similar antiobiotic sensitivity profile, being resistant to gentamicin and amikacin, and sensitive to netilmicin, ciprofloxacin, aziocillin and ceftazidime.

Twelve haematology bottles were tested and no bacteria were isolated. Twelve ESR bottles were tested, four from each of the three wards, and of these four grew a gram-negative, cytochrome oxidase-positive bacillus which was resistant to gentamicin on the primary culture plates. Of the four positive ESR bottles, two came from one ward and one each from the other wards. The full biochemical and antibiotic sensitivity profiles of these isolates were similar to that of the blood culture strains (Table).

TABLE

Isolates from ESR bottles

Alcaligenes denitrificans		
Resistant	Sensitive	Variable
gentamicin	ceftazidime	netilmicin
amikacin	ciprofloxacin	aziocillin

DISCUSSION

An organism similar to that which caused five episodes of pseudobacteraemia was isolated from the ESR bottles available in each of the three wards. Gram-negative organisms have frequently been isolated from haematology and ESR bottles, and these contaminated bottles have been shown to be the cause of pseudobacteraemia.[1, 2, 3, 4] In these cases, the most likely route of contamination

has been that after drawing blood from the patient the syringe needle was removed and the ESR bottle filled before the blood culture medium was inoculated. Even if a new sterile needle was placed on the syringe before the blood culture bottles were inoculated, the syringe nozzle could already have been contaminated during filling of the ESR bottle. This contamination might occur either by direct contact with the inside of the ESR bottle or by aerosol.

Successful sampling of blood for culture requires a careful technique with attention to detail. The operator's hands should be washed and gloves may be worn. Various recommendations have been made for skin disinfection of the chosen venepuncture site. Gillies[5] advocated a soap and water wash followed by alcohol and povidone iodine, whereas Tyrrel *et al*[6] advise two applications of 70% alcohol or an alcohol-based disinfectant. It is essential that alcohol is allowed to evaporate to dryness and iodine, if used, is allowed two to three minutes to act. After disinfection the operator should not touch the venepuncture site. Following sampling the needle should be changed prior to inoculation of the blood culture bottles. It is helpful if an assistant is available to rip off the protective cap from the blood culture bottles and disinfect the diaphragm with an alcohol wipe. If additional blood samples for other tests are also required, the extra bottles should be filled after blood culture inoculation to avoid possible contaminating organisms from them being carried over to the blood culture bottles. While the doctors who inoculated the blood culture bottles did not feel that contamination of this type had occurred, the outbreak was brought to an end by advising on the correct procedure.

Pseudobacteraemia is an important phenomenon in terms of patient well-being, clinical and laboratory workload, and the cost of unnecessary treatment. Fortunately, none of our cases were treated unnecessarily with antibiotics because monitoring in the bacteriology department raised the suspicion of pseudobacteraemia at an early stage.

REFERENCES

1. Whale K. Pseudobacteraemia: a bedside fault. *Lancet* 1983; I: 830.
2. Cookson BD, Mehtar S, Sadler G. Serratia pseudobacteraemia. *Lancet* 1982; II: 1276-7.
3. Willson PA, Petts DN, Baker SL. An outbreak of pseudobacteraemia. *Br Med J* 1981; **283**: 866.
4. Ispahani P, Lewis HJ, Greaves PW. Pseudobacteraemia, again. *Lancet* 1985; II: 383-4.
5. Gillies RR. Blood culture. In: Gillies RR, Dodds TC. Bacteriology illustrated. Edinburgh: Churchill Livingstone, 1984: 129-31.
6. Tyrrel DAJ, Phillips I, Goodwin CS, Blowers R. The use of the laboratory in diagnosis, therapy and control. London: Edward Arnold, 1979: 312-3.

The Ulster Medical Journal, Volume 61, No. 2, pp. 166 - 174, October 1992.

Historical Review

The evacuation of Burma: fifty years ago

J H Gorman, J S Logan, D A D Montgomery

1. Memories of the evacuation of Burma in 1942 in face of the Japanese invasion — J H Gorman

When asked to write a few notes about my spell in Burma, April and May 1942, I had to read again Field Marshal Sir William Slim's book, especially the first part of "Defeat into Victory", in order to refresh my memory with the dates and place names in Burma with which I was concerned. It is hard to believe that it took place 50 years ago and one gets no younger in body and mind.

I had joined the Indian Medical Service as a regular in 1929 as a Lieutenant: in 1942 I had just been promoted to Major. I was then serving in the Peshawar-Kohat District, Northern Command, as Deputy Assistant Director of Hygiene under Colonel A McCrea IMS, who was my Assistant Director of Medical Services (with whom I had previously served in Quetta at Western Command), and was enjoying the comparative freedom and calm of the Khyber Pass. After six months there I received out of the blue, on 12th April 1942, an express secret order from Army HQ to proceed immediately in uniform to Burma and act as Director of Public Health, Burma, for the evacuation of that country. I had to leave within forty-eight hours for Delhi, to see Colonel Ed Cotter IMS, Public Health Commissioner with the Government of India at the Director General Indian Medical Service's office, to be briefed on the evacuation situation at that time. I received the necessary travel warrant and papers authorising my journey from Peshawar District HQ to the Director General's office in Delhi, was duly briefed on the Burma situation, and directed to leave forthwith for Calcutta Airport (Dum Dum), to proceed by Chinese National Airways plane to Burma.

The travel ticket issued to me was for the Chinese National Civil Airways route from Calcutta to Chungking in western China (Chinese Army HQ at that time) via Lashio, a Burmese town in northeast Burma near the Chinese frontier. In Lashio I met the Civil Surgeon, an IMS Officer in Charge, on 16th April and I explained my identity and my purpose. He told me that a train would leave next morning, 17th April, for Maymyo (just northeast of Mandalay). He said that he would be leaving Lashio to cross into China by the Burma Road probably in a day or two because of the fast approaching Japanese invasion proceeding up the eastern border roads parallel to the Salween river valley. (The Japanese actually arrived there on 27/28th April after defeating a Chinese army in the area).

I proceeded by rail on 17th April 1942 to Maymyo, approximately 100 miles distant, and reported on the same day to Army Headquarters, Burma and to his

J H Gorman, MB, BCh, BAO, DPH, Lt Col Indian Medical Service (Retired), 1 Rathmore Road, Bangor, Co Down, Northern Ireland.

Excellency the Governor, Sir Reginald Dorman-Smith at Government House, as indicated in my orders. The Governor personally had initiated the express secret communication to the Government of India, concerning his requirement immediately of a capable officer to take over the duties of Director of Public Health, Burma, for the evacuation to India. He welcomed me personally, thanked me for my speedy arrival, and asked me what I needed to get on with the work. I asked for a reliable car and a good driver who knew the country. I got the Prime Minister's car (as he was leaving for India next day) and driver, within the hour, and he wished me good luck. I set off to meet the Surgeon-General IMS, Burma. He was leaving for India next morning.

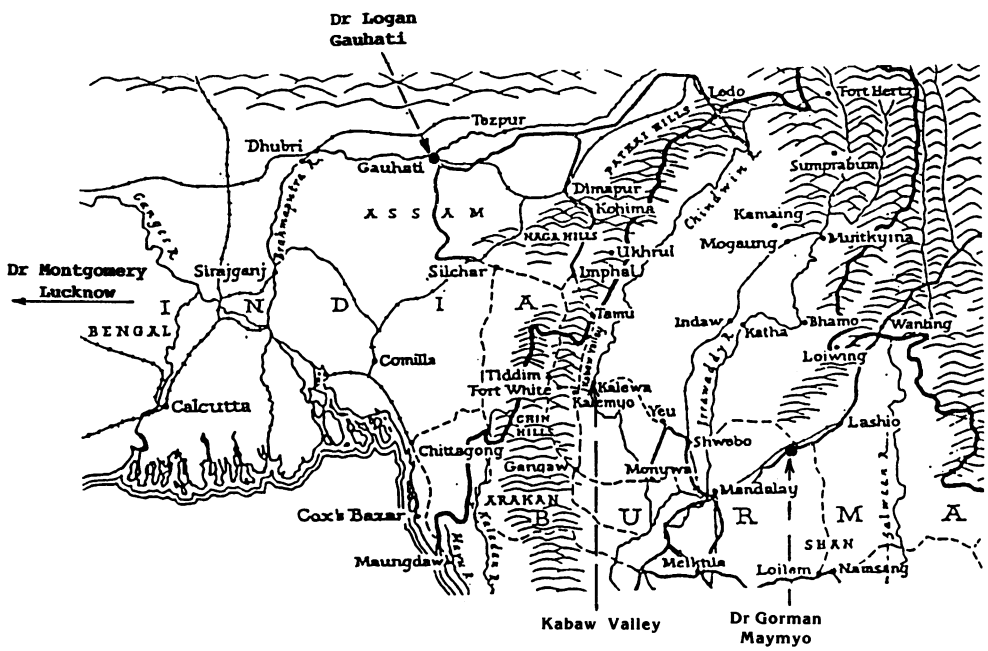


Fig 1. The Indian-Burmese border showing the places referred to in the text.
From Slim WJ, Defeat into Victory, 1956.

I then called on the Officer Commanding the British Military Hospital, a Lieutenant-Colonel RAMC, whose bungalow was nearby, and stayed the night with him. His wife had just left with other close friends the previous day to *walk out* to India by the Assam border. Next morning we were walking in the garden after breakfast when a Japanese air raid developed; bombers came overhead and we dropped into a shallow trench which had been dug in the garden for this purpose. No damage was done to the bungalow. I thanked the Colonel for his hospitality, expressed my sympathy for his unhappy situation, and left for Mandalay with car and driver to meet the volunteer evacuation staff and assistants in Mandalay HQ. I got the strong impression that they had not been informed of my coming, or of my arrival in Burma. I was given an overall account of the local situation, the distribution of local refugee camps, and of the general organisation. This seemed

satisfactory and was said to have been in operation since March. At first there had been just a trickle of fleeing refugees, then a deluge, especially after Rangoon fell to the Japanese on 9th March 1942. I settled in with the volunteers of the evacuation staff, comprising a young doctor employed in civil life by an industrial company in Burma, a police superintendent, a representative of the Irrawaddy Flotilla Company, and a trading company assistant. These people spent a lot of time attending the riverside docks and local camps, ensuring the correct passage of refugees by ships of the Irrawaddy Flotilla Company, a means of transport up the river Chindwin to Kalewa, their next destination and a roadhead to the India/Burma Frontier.

I visited the local and area refugee camps, checking the various measures for ensuring health, and supplies of adequate drinking water and food, and for keeping watch on infectious diseases, especially cholera, plague, dysentery and smallpox. Refugees were mainly walking out to India on the side paths and jungle tracks where possible, in order to leave the roads clear for troop movements. Refugee camps or halts were usually sited at suitable ten mile distances, especially on the jungle routes north of Kalewa to Tamu and Imphal and Kohima. The camps were mainly using basha type structures and charpoys.* Camp volunteers supervised the administration and health measures necessary at each large camp en route to India.

The most dangerous health risk of all was the notorious Kabaw valley track, ninety miles long, malaria infested and leading through dense jungle to Tamu. The monsoon was expected around 20th May, but heavy rain fell before that. The Burma Army HQ were now working hard to improve the track to Tamu and Imphal and to stock it with supplies and water where necessary, General Headquarters India were apparently doing the same for their side of the frontier.

The evacuation staff left Mandalay and Ye-u on about the 28th April on orders of the Military Authority, and proceeded to the Chindwin river, across country due west of Ye-u. Our cars at the river were damaged as far as possible and left behind. A country boat took us to the west bank of the Chindwin, and the next day we were picked up at a small riverside station, as refugees, by a steamer of the Irrawaddy Flotilla Company and conveyed to Kalewa, where we disembarked and separated.

Our job as a unit in central Burma at Mandalay, Sagaing, Shwebo, Kyaukmyaung and Ye-u areas was finished and the troops were moving via Ye-u to Shwegyin on the Chindwin for a crossing to Kalewa, to disembark, and sink any "available steamers" to prevent them falling into Japanese hands.

At Kalewa I was myself a refugee without staff, but still Director of Public Health, Burma so I was able to contact the local District Commissioner (who stayed on until the very last evacuee passed through) to find out the local refugee situation. No special arrangements were available at Kalewa as refugees made straight off from the steamers or land approaches on the West bank of the Chindwin by the shortest land route through local jungle paths to Kalemyo and to Tamu.

---

* basha — a bamboo structure covered with a tarpaulin or thatch.
charpoy — string bedstead.

I set off up the road from Kalewa via the highly malarious Kabaw Valley, for Tamu and after two hours on the road I was overtaken by a jeep driven by an IMS officer, Captain George Ledgard, who was acting as Liaison Officer between the Indian and Burmese Governments in respect of refugee problems from Kalewa to Tamu (some 200 miles distant). At Tamu, the last small village in Burma, I reported to the Senior IMS Liaison Officer, Lieutenant-Colonel Taylor, under whose command I automatically came, and was assigned to take charge of a large evacuee camp. This I did for a week, when I was given orders to return to New Delhi, India, and report to the DGIMS and Colonel Cotter IMS, Public Health Commissioner to the Government of India.

I got a lift on some lorries carrying wounded and sick casualties from the Burma army to Imphal and Kohima, and the next day by lorry to the railhead at Dimapur, approximately fifty miles from Kohima. After Tamu I was available for general military duties as an MO. I arrived at Dimapur railhead on 17th May 1942, reported to the Military Commandant and was posted to a rest camp at Dimapur. On 19th May, I was ordered to take charge of some fifty troops and junior officers and proceed by rail to a reinforcement camp at Gauhati. On arrival there on 20th May I became seriously ill with dysentery and malignant tertian malaria (probably from the Kabaw Valley) and was admitted to 16 Indian Casualty Clearing Station there, under the command of Lieutenant-Colonel A N T Meneces, RAMC. From here I was evacuated immediately by steamer down the Brahmaputra river, and by rail to The British Military Hospital, Calcutta. I was discharged from the hospital on 2nd June, on a month's leave on medical certificate. On completion of this leave, I was posted as Assistant Director of Hygiene, Southern Army (Rear headquarters at Poona) and then to advanced HQ at Bangalore where the 33 Corps were forming and training, later to operate in the renewed operations for the reconquest of Burma.

The story of retreat from and evacuation of Burma is well told by General Sir William Slim,[1] the new Burma Corps Commander (from 13th March to 20th May 1942) in his book "Defeat into Victory" and unless this has been read it would not be possible to appreciate the problems and extreme hardships by all directly involved.

This shows the campaign "warts and all", in its true light, as an outstanding fighting retreat over 900 miles of tough country against a well-trained and capable enemy. Having participated in the retreat in a small way gives me a sense of real pride in the achievements of General "Bill" Slim and the 1st Burma Corps.

Rangoon fell on 9th March, 1942 and the last battle took place in the "basin" at Shwegyin near Kalewa around 10th to 12th May 1942. The monsoon rain descended in torrents on 12th May making the "roads" (jungle tracks) impassable and prevented any possible follow up by the Japanese Army. This enabled the "Burma Army" to continue the retreat to the Indian Frontier, unmolested. The troops that survived might look like scarecrows, but they looked like soldiers too!

---

1. Slim, Field Marshall WJ, *Defeat into Victory*. London: Cassell & Company Ltd., 1956.

2. **Events at Gauhati** — J S Logan

16 Indian Casualty Clearing Station at Gauhati, to which Lieutenant-Colonel Gorman was admitted as he describes previously, was raised hurriedly at Meerut about the month of April 1942, part of the measures to meet the Japanese invasion of Burma and the threat to India. The officer commanding was a regular officer, Lieutenant-Colonel A N T Meneces, RAMC, As usual, drafts of officers, RAMC and IMS were posted to the Unit, drafts of other ranks of the Indian Hospital Corps, and a few British other ranks of the RAMC, but no NCO. There was no Viceroy's commissioned officer, no quartermaster, no warrant officer of the Indian Medical Department or of the RAMC. There were three motor vehicles but no drivers. Except for the CO, the RAMC officers had arrived in India only a few weeks before. None at that time spoke Urdu. None of the IMS officers was a regular. Stores were issued, ordnance and medical, but of necessity they remained unopened and unchecked. There was no time for unit organisation or training. In a few days the CCS began the 2000 mile journey by rail across India to Gauhati in Assam on the eastern bank of the Brahmaputra. The great river was crossed not by a bridge, but by ferry boats. The CCS then occupied the one-storey buildings of the Bishop Cotton College and immediately opened, not without some confusion. As Colonel Gorman says, Gauhati was one of the key points on the route of evacuation of the sick and wounded of the Burma Army and of other formations, coming from the north. Access to central India could only be by ferry across the river to the railway system of Bengal or south by river steamer. Train after train arrived on the metre gauge line from the north, and the troops tumbled, those that were able, out onto the platform. While the improvised wards were full of malaria and dysentery cases (and there were great difficulties in treating them and feeding them) the worst chaos lay in sorting and dispersing the train arrivals.

One remembers a tetanus case taken off a luggage rack, and a manic Scottish soldier who had been tied hands and feet by his comrades to control him. Two blue-jacketed Chinese tumbled out of one train and squatted down on the platform to light feverishly their primitive opium pipe. A Chinese general with trench foot detrained, accompanied by a Chinese medical officer who had been a houseman in Wigan. We received one distressed young officer, who had had the brachial plexus destroyed by a septic gunshot wound still pouring pus. The numbers all but overwhelmed the CCS. While the MOs gave a hand with all jobs and toil (including scrubbing down the verandahs each morning) they had to spend much time in the wards, and in selecting and seeing sick and wounded onto the river steamers which ran down the river to Sirajganj on the right bank in Bengal. That was why a British private soldier was made an acting unpaid lance corporal, and put in charge of directing and loading bus after bus (the rickety local buses) for the journey up to Shillong in the Khasi hills. He did nobly but it was a cruel task for him, coping with senior officers (who could not believe they had to get into these vehicles and abandon their kit, for which there was no room). He eventually was exhausted and admitted to the CCS himself — the "willing horse" syndrome. We could not get suitable food for the patients. The local supply officer, when appealed to, cycled round the countryside himself and came back

J S Logan, MD, FRCP, Honorary Archivist, Royal Victoria Hospital, Belfast BT12 6BA.

with a dozen eggs in a basket! I myself, to treat the amoebic dysenteries, had to go round the drug shops in the bazaar buying up phials of emetine tablets for solution and injection. There were not many to be had. Much of this shocking state might have been avoided or corrected earlier if the commanding officer had not been ordered to act as Senior Medical Officer of the area and was not much with the Unit.

There was no second-in-command. No surgery could be done of course; the surgeon was almost at once posted away, there was no steriliser, and no way of staffing or equipping a theatre. It can be said that the treatment of the patients in the CCS was successful. We saw every malarial syndrome except blackwater fever, but I do not remember a death from malaria; or dysentery. The evacuation by river steamer was also a success. It was the staging process, the reception at the railway station, the sorting, the temporary accommodation, the feeding, the care of kit, the dispersal which were shocking — because of lack of staff and resources. Things improved with time and the posting of some nursing sisters (including one from Ballycarry) and the arrival of some Burmese girls trained in nursing by an American missionary. And of course everyone from officers to sepoys worked very hard to keep things going. It was awkward, however, when General Sir Harold Alexander, who had been GOC Burma, called on his way down the line, and the officers in one ward complained to him they had yet to see an MO. In the middle of all this, being short of bedpans, we broke into a locked cupboard in the college and requisitioned a bedpan we saw there. Someone made representation to the Government of Assam and the unit was officially rebuked for what the Government of Assam in writing called "this typical military filibuster"!

A final memory; in sorting the mass of abandoned kit I found the secret service card of a senior Burmese Government official, a European, and the letter conveying to him the thanks of the Government of Burma for his success in interrogating a high placed Burmese traitor. I found an address for him and sent him these things. He was grateful but wrote to ask, had I seen anything of the little bag of rubies which he had had with him? I could find no rubies. But worse things happen in war: exhausted women and children had been lost by the track-side in the Kabaw valley. There was a glimpse of civilisation when the Deputy Commissioner of Kamrup District, a courteous, cultivated Indian gentleman of the Indian Civil Service, came one evening to dinner and tried to engage me in conversation about the poetry of James Stephens. I was abashed at knowing practically nothing of this Irish poet.

Colonel Meneces had a distinguished career in the Army Medical Services. He received the Distinguished Service Order in the war and ultimately, before retirement, as a Major-General was Director of Medical Services of the British Army of the Rhine.

3. Events at Lucknow — D A D Montgomery

Lucknow, the capital of the United Provinces, played an important role in the reception of the sick and wounded evacuated after the defeat of the army in Burma. In Assam into which the retreating army had entered, there were few medical facilities and certainly nothing capable of coping with a demoralised army with its malnourished troops many of whom had malaria or dysentery or both, and the wounded. The conditions at Gauhati described by Dr Logan in the preceding paper confirmed the necessity of moving the bulk of the sick and wounded into the Eastern provinces of India.

At that time I was Staff Captain (Medical) of Lucknow District Headquarters of Eastern Command. Early in April the District HQ moved to Ranikhet, a hill station 6000 ft up in the approaches to the high mountains of the Himalays. This yearly move of the British Army to the hills, in order to avoid the worst of the hot weather, was an established custom. That it took place in 1942 during the Japanese advance into Burma reveals how little appreciation there was in military circles of the danger which the invasion of Burma threatened.

Some inkling of the problems likely to arise in Lucknow following the evacuation of casualties from Burma took place towards the end of April and I was sent down to Lucknow to make what preparations I could to meet the likely demand on our medical services. Lucknow was an important railway centre where the narrow guage lines from Bengal and Bihar joins the broad guage line from Delhi to Calcutta. There were two military hospitals in Lucknow, the British Military Hospital and the Indian General Hospital and over the next few weeks I was able to staff and equip 1000 beds in various empty barracks in the cantonment. There was very little information as to what we were to expect and by the 1st of May casualties started to arrive by train and improvised ambulance trains both on the narrow gauge from Gauhati and on the broad gauge from Sirajganj and Calcutta. The seriously sick and wounded whom I felt it would be unwise to send further were taken off and admitted to our hospitals each of which expanded their bed capacity as rapidly as they could together with the support of the additional beds in the barracks. Once our beds were full to overflowing I had to send the trains on to other hospitals in Eastern and Central Commands. The movement of trains was largely worked out between the railway staff and myself and I must pay tribute to the willing co-operation of railway personnel over the next few months. The Medical Directorate at GHQ kept in regular contact with us in Lucknow regarding the number of trains received or passing through and gave practical help in selecting destinations to which trains should be sent. So great was the traffic during May to September 1942 that train loads were sent throughout Central Command, to some of the Princely States and reached places as far away from Burma as Poona and Sialkot.[1]

The reception of trains or their re-routing imposed an enormous strain on the medical services in Lucknow especially with the administrative services away in the hills. Ambulances had to be provided and food had to be prepared for both

---

D A D Montgomery, MBE(Mil), CBE, MD, FRCP, FRCOG, 59 Church Road, Newtownbreda, Belfast BT8 4AN.

British and Indian troops. For the latter the resources of the catering staff were so stretched that it was impossible to make a distinction between food for Hindus and Muslims or for the caste system and all had to accept the same food and I do not remember hungry soldiers complaining or refusing to eat the food which we supplied.

Trains continued to arrive at all hours of the day and night and I spent many days and nights in Lucknow Station where I had a camp bed in the Station Master's Office. All this happened at the height of the hot weather with shade temperature between 110° – 120°F and at night down to 95° – 100°F. One only survived by drinking copious amounts of fluid with large supplements of salt. I had a team of four officers to help me, one IMS and three RAMC and they were a tremendous support but I bore the ultimate responsibility. Gradually we evolved a reception and distribution scheme that seemed to meet the needs of the situation in Lucknow which was approved by the DDMS Eastern Command and the DMS in Delhi. The monsoon rains were late that year and it was early in July before the rain came. Certainly there was relief from the blistering heat but the hot damp weather was enervating physically and mentally in the conditions under which we had to work.

By the end of August the pressure of ambulance trains to Lucknow had eased and I was sent on leave to Ranikhet for three weeks. The change to the cooler weather was marvellous for I was quite exhausted after the busy months of work in Lucknow. On my return to Lucknow I found that I had been appointed to a new post of DADMS (Distribution) at GHQ but seconded to Lucknow District where I continued with the work that I had been doing but with a wider remit for organising the evacuation of casualties throughout Eastern Command. This was the first of a number of such posts recorded in the orders for the evacuation and distribution of casualties referred to in the postscript to these papers. Much of the experience gained in Lucknow was reported to GHQ and incorporated in this document.

Fifty years on it is difficult to recapture the sights and sounds of a busy Indian railway junction and to remember details of the seemingly endless stream of packed trains and the sick and tired soldiers. There was always the anxiety of wondering "will I be able to cope" for I was a very junior officer to be left to deal with this crisis. Somehow by improvisation and very hard work we managed. The fact that no regular or more senior officer was sent to take over suggested that my ADMS was satisfied with our efforts and allowed me to carry on.

EDITORIAL NOTE. It was for distinguished service in the crisis of the evacuation that Captain D A D Montgomery, RAMC (as he then was) was appointed to Membership of the Military Division of the Order of the British Empire.

4. Conclusion: A ticket to Burma

Junior doctors today might like to reflect on how they would have coped with the circumstances described by the authors of this trilogy. The disorder and difficulties were eventually controlled. By an immense administrative effort, of which the Government of India, the War Department and the Medical Directorate could be proud, the evacuation and distribution of sick and wounded were reinforced, supplied and organised. Air evacuation began. In late 1943 GHQ Evacuation and Distribution Staff was formed. In 1944 the Medical Directorate was able to publish instructions which seemed to cover in detail every method and route of evacuation in India Command. These are available in *Instructions regarding the Evacuation and Distribution of Invalids and Casualties in the India Command.*[2] Many lessons can still be learned from this book.

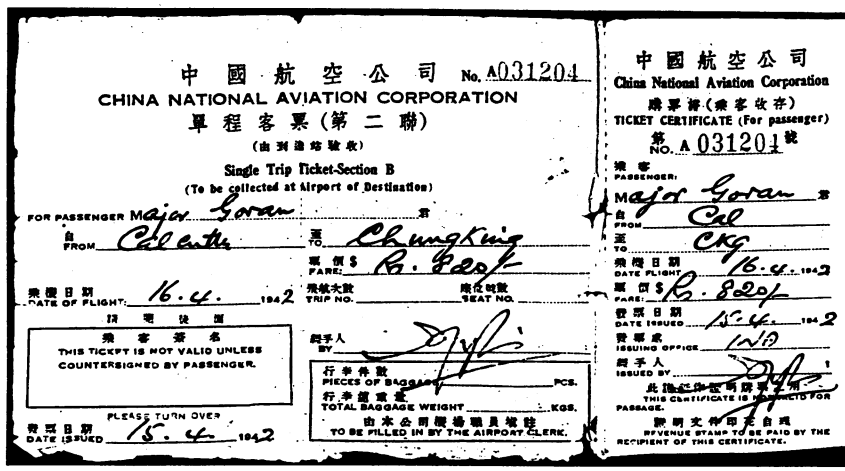

中國航空公司 No. A031204
CHINA NATIONAL AVIATION CORPORATION
單程客票(第二聯)
Single Trip Ticket-Section B
(To be collected at Airport of Destination)
FOR PASSENGER Major Gorman
FROM Calcutta TO Chungking
FARE: Rs. 820/-
DATE OF FLIGHT: 16.4. 1942
THIS TICKET IS NOT VALID UNLESS COUNTERSIGNED BY PASSENGER.
PLEASE TURN OVER
DATE ISSUED 15.4. 1942
PIECES OF BAGGAGE
TOTAL BAGGAGE WEIGHT KGS.
TO BE FILLED IN BY THE AIRPORT CLERK.
中國航空公司
China National Aviation Corporation
TICKET CERTIFICATE (For passenger)
NO. A 031201
PASSENGER: Major Gorman
FROM Cal
TO CKG
DATE FLIGHT 16.4. 1942
FARE: Rs. 820/-
DATE ISSUED 15.4. 1942
ISSUING OFFICE IND
ISSUED BY
THIS CERTIFICATE IS NOT VALID FOR PASSAGE.
REVENUE STAMP TO BE PAID BY THE RECIPIENT OF THIS CERTIFICATE.

Fig 2. The air ticket from Calcutta to Chungking issued to Major Gorman on 15 April 1942.

Major Gorman (as he then was) kept the air ticket from Calcutta to Chungking, which was the initial move in this trilogy (Fig 2). He had graduated in medicine in Belfast in 1926, and received the diploma in public health in 1936. Commissioned in 1929 into the Indian Medical Service, after military duty he was appointed in 1939 to the civil public health branch of the Government of India. He held the posts of Port Health Officer, Calcutta; Assistant Director of Hygiene (Famine Relief) in the great Bengal famine of 1943; Director of Public Health of Madras Province; and Port Health Officer, Bombay. The Public Health Commissioner with the Government of India noted that in the famine of 1943 his organisation had carried out "vaccination against smallpox and inoculation against cholera to an extent hitherto undreamt of among the civil population in India". Following this outstanding career in the Indian Medical Service Lieutenant-Colonel Gorman engaged in general practice in Belfast.

---

1. Crewe FAE. The Army Medical Services. Campaigns. Volume 5, Burma. London: HMSO, 1966.
2. Instructions regarding the Evacuation and Distribution of Invalids and Casualties in the India Command. Issued by the Adjutant-General's Branch (Medical Directorate). Printed by the Manager, Government of India Press, Simla, 1944.

The Ulster Medical Journal, Volume 61, No. 2, pp. 175 - 178, October 1992.

Case report

Anomalous origin of the left coronary artery presenting in an adult

E W Chew, M Varma, J G Murtagh

Accepted 10 March 1992.

Anomalous origin of the left coronary artery from the pulmonary artery is a rare congenital cardiovascular defect with an incidence of 1 in 300,000 live births. It is the most common anomaly of the coronary vasculature, with a frequency of 0·5% of all congenital cardiac defects. When presenting in infancy it is known as the Bland-White-Garland syndrome.

The mortality rate among infants and children without operation is between 80% and 90%.[1] Of 14 adults with the condition who survived to between 16 and 60 years, ten died suddenly and unexpectedly, associated with sudden physical exertion.

We present a 55-year-old woman whom we believe to be only the fifth patient over the age of 50 with this condition in whom the diagnosis was made during life; successful bypass grafting was achieved using the left internal mammary artery.

CASE REPORT. A 55-year-old woman was referred for evaluation of a five year history of exertional chest pain. Apart from a 15 year history of hypertension there was no other significant past medical history. She had undergone five pregnancies without complications. There was a grade 3/6 mid-systolic murmur at the cardiac apex. Electrocardiograph showed left ventricular hypertrophy with T wave inversion in the lateral leads. Chest X-ray showed cardiomegaly. She exercised for 12 minutes on the treadmill (modified Bruce protocol) without chest pain, and showed minimal ST segment changes in the lateral leads.

Selective right coronary angiography showed a tortous dilated (10–12 mm) vessel supplying collaterals to the left coronary artery (Figure). The contrast material then flowed into the pulmonary artery. The left ventricle was dilated with a hypokinetic antero-lateral wall, and mild mitral regurgitation was present. Cardiac catheterisation data was consistent with impaired left ventricular function,

Cardiac Unit, Belfast City Hospital, Belfast BT9 7AB.
Eng Wooi Chew, MD, MRCP, Registrar.
J G Murtagh, MD, MRCP, Consultant Cardiologist.

The Erne Hospital, Enniskillen BT74 6AY.
M Varma, MD, FRCP, Consultant Physician.

Correspondence to Dr Chew.

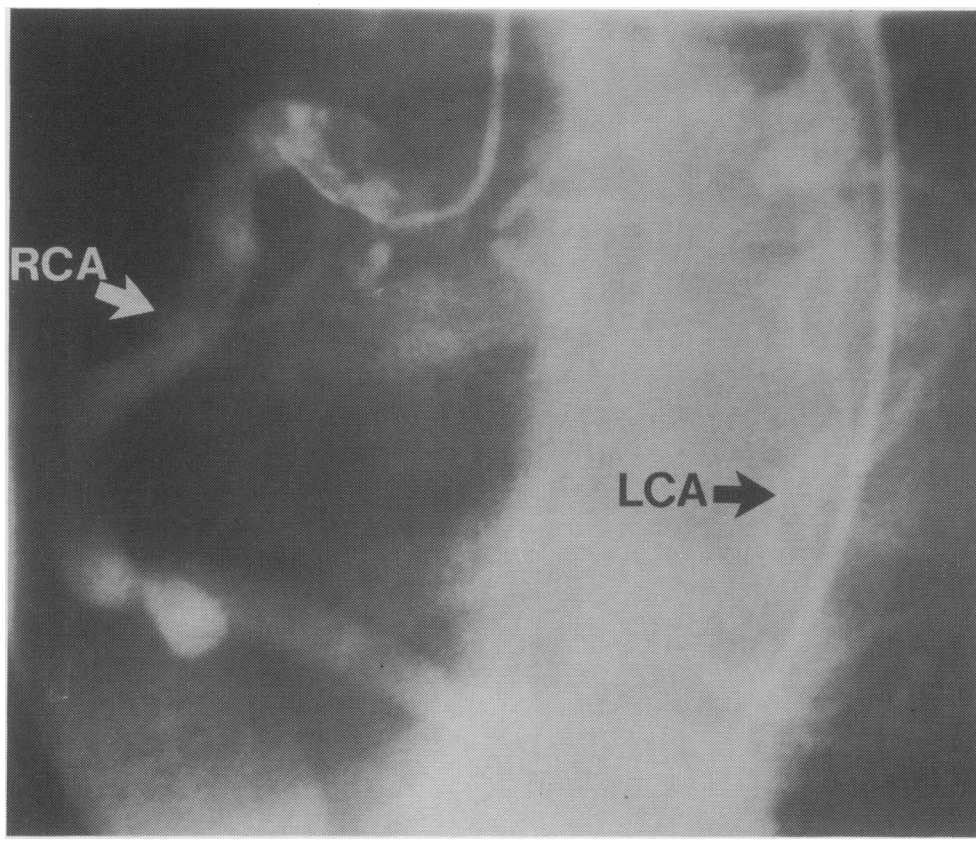


Figure.
Tortous, dilated right coronary artery (RCA) supplying collaterals to the left coronary artery (LCA).

with the left ventricular ejection fraction calculated at 52% (normal value 59% – 75%) and an elevated left ventricular end-diastolic pressure of 24 mmHg (normal value 3 – 12 mmHg). Both the pulmonary systolic pressure and mean pulmonary wedge pressure were elevated at 51 mmHg (normal value 15 – 30 mmHg) and 20 mmHg (normal value 1 – 10 mmHg) respectively. The oxygen saturation was 64·7% at the right atrial level, 62·9% at the right ventricular level, 68·3% at the pulmonary artery level and 92·6% at the level of the aorta. These findings would be consistent with a left to right shunt at the pulmonary artery level with almost 7% step up in oxygen saturation between the right ventricle and the pulmonary artery (criteria for left to right shunt at the great vessel level is $>$ 5% step up in oxygen saturation).

At operation, there was evidence of a full thickness infarct in the anterior area of the left ventricle. The anomalous origin of the left main coronary artery was ligated and the left internal mammary artery was grafted to the first diagonal artery. Her postoperative course was uneventful and at 6 weeks review appointment she reported symptomatic improvement, with diminution of the intensity of the systolic murmur.

DISCUSSION

The first case of anomalous origin of the left coronary artery from the pulmonary artery was reported by Abbott in 1908; this was a postmortem diagnosis in a 60-year-old woman who had met with an accidental death. The clinical syndrome in infancy was originally described by Bland, White and Garland when they assembled the clinical and postmortem findings from an infant whose symptoms were dyspnoea, sweating and crying with exertion.[2] Infants usually present with congestive heart failure, angina, failure to thrive, or for investigation of a murmur or cardiomegaly on chest X-ray. Those who survive to adult life may present with heart failure or angina, or may die suddenly without any symptoms.

Sudden death in adults with this condition is common and is usually related to exertion.[1] Our patient is unusual in that she was relatively asymptomatic in spite of a very active life-style and had undergone five pregnancies without complications. Several authors have suggested that the presence of collaterals from the right coronary artery is a prerequisite for survival[3] which we found in this patient.

The management of infants and children with this condition is controversial with rather conflicting results from several series.[3] There is, however, increasing evidence that early establishment of antegrade flow in the anomalous artery is beneficial. Laborde and colleagues reviewed results in 20 cases, comparing outcome in those operated on before a year old with those in whom the operation was delayed.[4] They concluded that if the operation was delayed until the patients are more than a year old the outcome is more favourable. Other series however have shown that with medical treatment alone, mortality in the first year of life ranges from 35%[5] to 80%[6] or even 90%.[7] This discrepancy could be explained by the fact that with the severity of the condition natural selection ensures that only low risk patients survive beyond one year of age.

The surgical treatment of choice in this condition remains controversial. Simple ligation has been associated with a higher mortality and a higher risk of sudden death. Patients who survive ligature are left with a one coronary artery system which is theoretically less satisfactory and is associated with greater risk from atherosclerosis. The establishment of a two coronary artery system has been associated with a more significant improvement in left ventricular ejection fraction postoperatively[7] and a less ischaemic electrocardiograph on treadmill exercise testing.

Several surgical techniques have been tried, but each has a drawback. Direct reimplantation of the left coronary artery into the aorta is often technically difficult especially in adults, due to the distance between the aorta and the anomalous orifice. Aorta-coronary bypass grafting using the saphenous vein has the potential risk of graft occlusion[7] and a review of several series by Laborde *et al*[4] gives an occlusion rate of 22% among patients operated upon under one year of age. A technique that has been found to be useful in children is the creation of a left coronary-to-aorta tunnel with a pulmonary artery baffle (Takeuchi procedure).[8]

The left internal mammary artery was used to graft the anomalous left coronary artery in our patient; this has been reported only in one other case.[9] It has satisfactorily established antegrade flow into the left coronary artery and should maintain patency better than a saphenous vein graft.

The authors would like to thank Mr H O J O'Kane, FRCS, for his surgical management of this case.

REFERENCES

1. George JM, Knowlan DM. Anomalous origin of the left coronary artery from the pulmonary artery in an adult. *N Engl J Med* 1959; **261**: 993-8.
2. Bland EF, White PD, Garland J. Congenital anomalies of the coronary arteries: report of an unusual case associated with cardiac hypertrophy. *Am Heart J* 1933; **8**: 787-801.
3. Bunton R, Jonas RA, Lang P, Rein AJJT, Castaneda AR. Anomalous origin of the left coronary artery from the pulmonary artery. Ligation versus establishment of a two coronary artery system. *J Thorac Cardiovasc Surg* 1987; **93**: 103-8.
4. Laborde F, Marchand M, Leca F, Jarreau M-M, Dequirot A, Hazan E. Surgical treatment of anomalous origin of the left coronary artery in infancy and childhood: early and late results in 20 consecutive cases. *J Thorac Cardiovasc Surg* 1981; **82**: 423-8.
5. Driscoll DJ, Nihill MR, Mullins CE, Cooley DA, McNamara DG. Management of symptomatic infants with anomalous origin of the left coronary artery from the pulmonary artery. *Am J Cardiol* 1981; **47**: 642-8.
6. Wesselhoeft H, Fawcett JS, Johnson AL. Anomalous origin of the left coronary artery from the pulmonary trunk. Its clinical spectrum, pathology and pathophysiology, based on a review of 140 cases with seven further cases. *Circulation* 1968; **38**: 403-25.
7. Arciniegas E, Farooki ZQ, Hakimi M, Green EW. Management of anomalous left coronary artery from the pulmonary artery. *Circulation* 1980; **62 (Suppl 1)**: 180-9.
8. Takeuchi S, Imamura H, Katsumoto K, *et al.* New surgical method for repair of anomalous left coronary artery from pulmonary artery. *J Thorac Cardiovasc Surg* 1979; **78**: 7-11.
9. McComb JM, Vincent R, Hilton CJ. Recurrent ventricular tachycardia associated with anomalous left coronary artery from the pulmonary artery in a child managed by revascularisation and map-guided endocardial resection. *Br Heart J* 1989; **62**: 396-9.

The Ulster Medical Journal, Volume 61, No. 2, pp. 179 – 181, October 1992.

Case report

Enterolith obstruction of the small bowel

W A McCallion, P M Higgins, T E B Dane

Accepted 16 June 1992.

Intestinal obstruction caused by an enterolith formed in a small bowel diverticulum is rare. In most cases the enterolith arises from a jejunal diverticulum in an elderly patient. When enterolith obstruction is diagnosed, the entire small bowel and gallbladder should be examined to determine the source. We present three cases and discuss the management options.

CASE 1. A 37-year-old female presented with a four hour history of abdominal pain, nausea and vomiting. There was no past history of surgery. Abdominal examination revealed slight distension, generalised tenderness and diminished bowel sounds. Hernial orifices were normal. Radiography showed multiple dilated loops of small bowel. There was a polymorphonuclear leucocytosis of $15 \cdot 0 \times 10^9/1$. At emergency laparotomy an inflamed Meckel's diverticulum was found, distal to which was an enterolith obstructing the terminal ileum. The remaining small bowel and gallbladder were normal. The diverticulum was excised and the enterolith milked back and removed via the enterotomy. Postoperative recovery was uneventful.

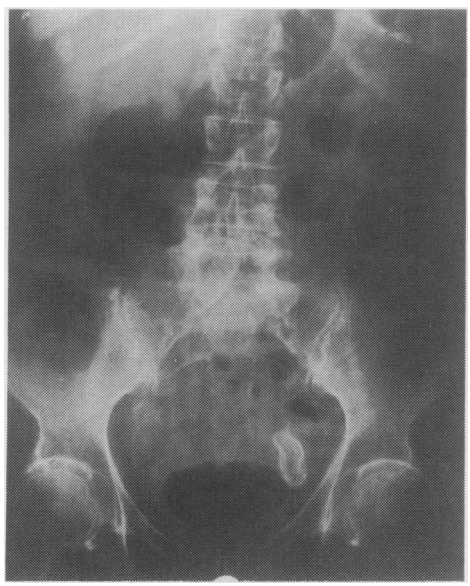
CASE 2. A 70-year-old female presented with a four week history of crampy central abdominal pain, followed by nausea and vomiting. Bowel habit was regular. Appendicectomy and hysterectomy had been performed in 1939 and 1961 respectively. Abdominal examination revealed marked distension and increased bowel sounds. Hernial orifices were normal. Abdominal radiography showed dilated small bowel and a large laminated opacity in the pelvis (Fig 1). There was no air in the biliary tree. Initial conservative management failed, and at laparotomy a hard enterolith obstructing the terminal ileum was removed via an enterotomy. The source of the enterolith was found to be a large jejunal diverticulum which was invaginated into the lumen. Postoperative recovery was unremarkable.

CASE 3. A 90-year-old man presented with a ten day history of crampy abdominal pain, followed seven days later by distension and vomiting. Four years previously, sigmoid colectomy had been performed for diverticular disease, and

Coleraine Hospital, Coleraine, Co. Londonderry, Northern Ireland.
WA McCallion, BSc, FRCS, Regional Senior House Officer.
P M Higgins, FRCR, Consultant Radiologist.
T E B Dane, MD, FRCS, Consultant Surgeon.

Correspondence to Mr McCallion, Royal Belfast Hospital for Sick Children, Belfast BT12 6BA.

jejunal diverticulosis was noted at that time. On examination the abdomen was distended and tender to the right of the midline. Bowel sounds were increased and hernial orifices normal. Radiography revealed dilated loops of small bowel but no abnormal opacities. Initially, symptoms and signs resolved with conservative management, but ten days following admission, distension and vomiting recurred. A small bowel series showed several large duodenal and jejunal diverticula and dilated proximal small bowel (Fig 2), but the nature of the obstruction was still uncertain. At laparotomy, a 3 cm diameter enterolith was found obstructing the distal jejunum. This was milked proximally and removed along with a 12 cm segment of jejunum containing multiple diverticula. Although initial recovery was satisfactory, he gradually deteriorated and died four weeks later.



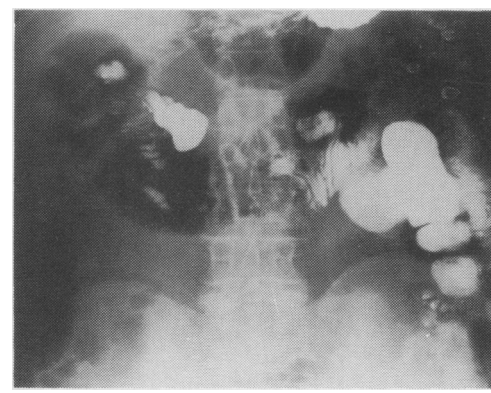


Fig 1 (opposite).
Laminated opacity in the pelvis with associated small bowel obstruction.

Fig 2 (above).
Small bowel series showing multiple duodenal and proximal jejunal diverticula.

DISCUSSION

Intestinal obstruction due to enterolith formation in a small bowel diverticulum is uncommon and has been the subject of occasional case reports.[1-3] This series of cases is presented to highlight this unusual cause of obstruction and to discuss the management options.

Unlike Meckel's diverticulum, jejunal diverticula are uncommon, acquired and usually occur in the elderly. They are false diverticula in that they lack a muscle coat. Both may be complicated by haemorrhage, inflammation and perforation. In addition, bacterial overgrowth in large jejunal diverticula is a recognised cause of malabsorption.[4] Chemical analysis of small bowel enteroliths has revealed mainly unconjugated bile acids.[5] It has been postulated that stasis, due to small bowel dyskinesia, results in bacterial overgrowth causing deconjugation of bile salts which precipitate to form a nucleus for enterolith formation. In the absence of small bowel diverticulosis enteroliths may form around a nidus such as fruit skins and stones, or rarely around ingested foreign bodies.[6]

In our first two cases removal of the enterolith was successfully combined with either invagination of a single jejunal diverticulum or wedge excision of a Meckel's diverticulum. In the third case a segment of jejunum containing multiple diverticula was resected and the enterolith removed. This elderly patient died postoperatively. Since jejunal diverticula usually occur in the elderly it has been suggested that in order to minimise postoperative complications the obstructing enterolith should be broken up manually and milked into the caecum,[7] thereby obviating the need for enterotomy. Recurrent enterolith obstruction has not been reported, and since jejunal diverticula are usually asymptomatic it is recommended that in the absence of diverticulitis or necrosis the diverticulum or diverticular segment should not be resected in frail elderly patients.

When enterolith obstruction is diagnosed radiologically or at laparotomy, the entire small intestine and gallbladder should be examined to determine the source.

REFERENCES

1. Herbetko J, Brunton FJ. Enteroliths of small bowel diverticula. *Clin Radiol* 1991; **43**: 311-3.
2. Beal SL, Walton CB, Bodai BI. Enterolith ileus resulting from small bowel diverticulosis. *Am J Gastroenterol* 1987; **82**: 162-4.
3. King PM, Bird DR, Eremin O. Enterolith obstruction of the small bowel. *J Roy Coll Surg Edinb* 1985; **30**: 269-70.
4. Drude RB, Finkelman D, Davis WD, Ferrante WA. Malabsorption in jejunal diverticulosis treated with resection of the diverticula. *Dig Dis Sci* 1980; **25**: 802-6.
5. Phillips J. Two cases of intestinal obstruction due to an enterolith: with reports of the chemical analysis. *Br J Surg* 1921; **8**: 378-9.
6. Gardiner KR, Maxwell RJ. Incidental enterolithiasis. *Ulster Med J* 1989; **58**: 196-7.
7. Shocket E, Simon SA. Small bowel obstruction due to enterolith (Bezoar) formed in a duodenal diverticulum: a case report and review of the literature. *Am J Gastroenterol* 1982; **77**: 621-4.

The Ulster Medical Journal, Volume 61, No. 2, pp. 182 – 184, October 1992.

Case report

Small bowel diaphragm disease — strictures associated with non-steroidal anti-inflammatory drugs

K H McCune, D Allen, B Cranley

Accepted 16 June 1992.

"Diaphragm disease" is a recently described entity seen in patients taking non-steroidal anti-inflammatory drugs in which strictures or diaphragms develop in the small intestine.[1] A case of intermittent small bowel obstruction is reported, in which the cause was a combination of such diaphragms and herniation of the small bowel through a mesenteric defect.

CASE REPORT. A 62-year-old woman, who had had rheumatoid arthritis for 20 years, was investigated over a period of three years from 1988 for episodes of central crampy abdominal pain. These attacks were usually precipitated by eating and lasted for two to three hours post-prandially. They were occasionally associated with vomiting but always settled spontaneously. Her rheumatoid arthritis had been treated for 12 years with simple analgesics alone, including aspirin, but in 1987 she was started on azapropazone 600 mgs twice daily and piroxicam 20 mgs at night. There was no other past medical history of note.

Upper gastro-intestinal endoscopy, barium meal and barium enema were normal. An ultrasound scan showed gallstones, and cholecystectomy was performed in 1990. Laparotomy findings at this time were normal. However, her symptoms failed to resolve; further investigation which included small bowel barium series, colonoscopy, ERCP and CT scanning gave normal results.

She presented again in March 1991 with an acute episode of abdominal pain and vomiting. Plain abdominal X-ray showed small bowel obstruction, and laparotomy was undertaken. At operation, a 38 cm loop of ileum was herniating through an adjacent 20 cm defect in the distal small bowel mesentery. The bowel was macroscopically normal and moved freely in and out of the defect. However, several strictures could be felt along the loop of bowel and this segment was therefore resected. The defect in the mesentery was closed.

Daisy Hill Hospital, Newry BT35 8DR.
K H McCune, MB, ChB, Senior House Officer.
B Cranley, MD, FRCS, Consultant Surgeon.

Craigavon Area Hospital, Craigavon BT63 5QQ.
D Allen, MD, MRCPath, Consultant Pathologist.

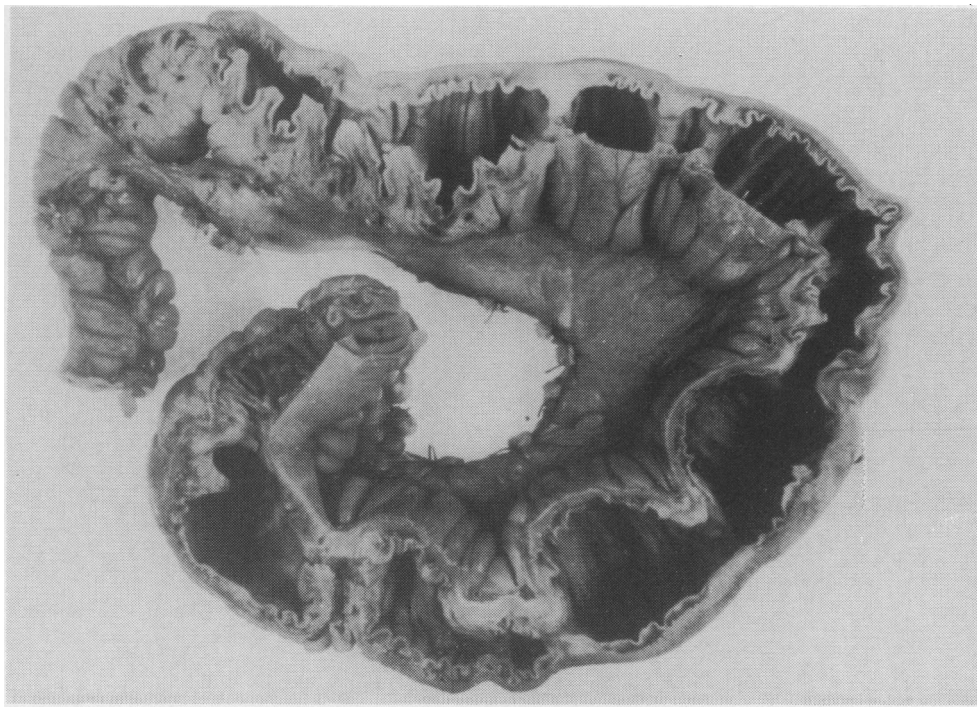


Figure.
Herniated ileum opened longitudinally showing the presence of several diaphragm-like strictures.

Pathological examination of the ileum revealed no gross external abnormality apart from slight episodic indentation of the serosal surface. On opening the bowel multiple concentric diaphragm-like strictures were found, compartmentalising the bowel into segments several centimetres in length. The strictures were between 0·5 cm to 1·5 cm long and had resulted in a stenosed bowel lumen measuring from 0·5 cm to 1 cm in diameter (Figure). Histological cross-sections showed a triangular core of prominent sub-mucosal fibrosis with a base of slightly distorted muscularis propria. The apex was covered by a cap of focally eroded mucosa and granulation tissue. There was no evidence of granulomatous inflammation.

DISCUSSION

"Diaphragm disease" has recently been described in a retrospective study of small bowel resection specimens from patients treated with non-steroidal anti-inflammatory analgesics.[1] All the seven cases reported have presented with sub-acute small bowel obstruction. The duration of usage of the non-steroidal analgesics was as short as 18 months, and strictures were mainly found in the mid to distal ileum. The macroscopic appearances of the luminal strictures is thought to be pathognomonic, and related to the ability of these drugs to alter the mucosal integrity of the bowel, causing cellular damage and scarring.[2] Although this effect is presumably systemically mediated,[3] it is not known whether another local factor may also be involved.

Our patient had an apparently congenital mesenteric defect with a well formed edge and evidence of hernia formation. Although the hernia was not causing obvious obstruction at laparotomy, we feel that it was probably responsible for the episodic pattern of her obstructive symptoms. Only the herniating loop of ileum appeared to be affected by "diaphragm disease", and the process of herniation may have increased the susceptibility of the mucosal cells to damage, perhaps by causing intermittent ischaemia.

This case illustrates the difficulty in diagnosis of "diaphragm disease". Strictures have been seen on small bowel barium series,[4] but are easily overlooked as they resemble the plicae circulares. At laparotomy the bowel is grossly normal and strictures are usually not detected unless specifically sought. Surgeons should be aware of the possibility of ileal diaphragm disease as a cause of small bowel obstruction in patients on non-steroidal analgesic therapy. A high degree of clinical suspicion is required as radiological investigation may appear normal.

REFERENCES

1. Lang J, Price AB, Levi AJ, *et al.* Diaphragm disease: pathology of disease of the small intestine induced by non-steroidal anti-inflammatory drugs. *J Clin Path* 1988; **41**: 516-26.
2. Bjarnason I, Zanelli G, Smith T, *et al.* The pathogenesis and consequence of non-steroidal anti-inflammatory drug induced small intestinal inflammation in man. *Scand J Rheumatol* 1987; **Suppl 64**: 55-62.
3. Bjarnason I, Williams P, Smethurst P, Peters TJ, Levi AJ. Effect of non-steroidal anti-inflammatory drugs and prostaglandins on the permeability of the human small intestine. *Gut* 1986; **27**: 1292-7.
4. Levi S, de-Lacey G, Price AB, *et al.* 'Diaphragm-like' strictures of the small bowel in patients treated with non-steroidal anti-inflammatory drugs. *Br J Radiol* 1990; **63**: 186-9.

The Ulster Medical Journal, Volume 61, No. 2, pp. 185 – 187, October 1992.

Case report

Diabetic amyotrophy in a teenage boy

A L Curran, O C Finnegan

Accepted 29 May 1992.

Diabetic amyotrophy is a rare complication of diabetes mellitus at any age, but it is almost unreported in the literature below the age of 30 years. We report the case of a 16 year old boy who attempted suicide due to the symptoms of diabetic amyotrophy.

CASE REPORT. A 16-year-old boy was admitted following an overdose of 48 tablets of diclofenac 100 mg, 20 tablets of codeine phosphate 30 mg and 40 tablets of paracetamol 500 mg. He was alert and felt well on admission. General examination was normal. His serum paracetamol level on arrival was 34 mg/l, which did not justify treatment with acetyl cysteine. Serum sodium, potassium and urea were normal. He was treated conservatively and observed overnight. On further questioning the patient revealed that the reason for his overdose was increasing clumsiness in his legs over the preceding six months which had halted a promising athletic career and had led to him being the focus of teasing at school. His home situation seemed to be supportive and caring, and no other cause for his attempted suicide was apparent.

He admitted to polyuria and polydipsia associated with weight loss of approximately 5 kg over the six months prior to admission. Detailed examination of his central nervous system revealed normal cranial nerve function, with normal optic fundi. A grade 2/5 weakness in his right extensor hallucis group of muscles was present, but no obvious weakness of the quadriceps or psoas muscle groups was detected, despite wasting of both thighs and calves. His gait was clumsy and broad-based, with hyper-extension of both knees. He was unable to stand on tiptoes. Tendon reflexes were absent in arms and legs, with downgoing plantar responses. There was no sensory deficit or impairment of joint position sense.

His random plasma glucose was 33·1 mmol/l. His haemoglobin concentration, white cell count, serum urea electrolytes and liver function tests were normal. Serum thyroxine, thyroid stimulating hormone, vitamin B12 and folate, complement, immunoglobulins and immune complexes were all normal. Serum auto-antibody tests, rheumatoid factor and porphyrin levels were normal. His serum aldolase was 3·5 iu/l (normal range 1·2 to 7·6) and creatine kinase 40 iu/l (less than 150). Radiograph of the lumbar spine was normal.

Coleraine Hospital, Co. Londonderry BT52 1JA.
A L Curran, MB, DCH, Senior House Officer.
O C Finnegan, MB, FRCP(Edin), Consultant Physician.
Correspondence to Dr Finnegan.

The diagnosis of diabetes mellitus was confirmed by the hyperglycaemia, and treatment was started with insulin. Within two weeks his plasma glucose levels were well controlled and he was allowed home. He was reviewed two weeks, six weeks, and four months later to assess the progress of both his diabetes and his muscle weakness. Exercise tolerance, assessed at two weeks and at four months by maximum running distance in ten minutes had increased from 760m to 1765m. By the end of four months the wasting of his legs noted on admission had resolved (thigh measurements increased from 41 to 49 cms and calf measurements from 15 to 25 cms), and his tendon reflexes had returned to normal. His diabetic control was good with average plasma glucose throughout the day 7·5 mmol/l and glycosylated haemoglobin (HbA1) 9·3% (normal $<7{\cdot}5\%$). He felt considerably better in himself and all depressive and suicidal ideas had gone.

DISCUSSION

Diabetic amyotrophy is an unusual presentation of diabetes mellitus, as too is attempted suicide. First described in 1890 by Bruns in Germany it was not until 1953 that Garland[1] again brought the condition to the notice of contemporary clinicians. Described as a disease associated with poor control of diabetes mellitus[2, 3] it usually presents with weakness and muscle wasting of the lower limbs, often associated with pain, and usually without loss of touch or joint position sense.[2] It is more common in the older diabetic and we have been able to find only one report of a patient under the age of 33 years in the past seven years.[3] The absence of pain in the legs does not fit with the more usual presentation of the condition but there are examples in the literature of pain-free diabetic amyotrophy.[2, 4] The involvement of the extensor hallucis group of muscles is of interest as Garland described a similar distribution in three of his 12 cases.[2] The term diabetic proximal motor neuropathy is sometimes preferred but is less appropriate in this case as there was no demonstrable proximal weakness. The abnormal gait and hyper-extension of the knees would, however, indicate some involvement of the quadriceps and psoas muscle groups.

The pathogenesis of diabetic amyotrophy remains obscure. Garland felt on the basis of nerve conduction studies that the primary lesion was in the lumbar spinal cord and he postulated a lower motor neurone neuropathy.[1] It was noted that as the diabetic control improved so also did the muscle weakness.[2, 3, 4] This is consistent with the metabolic theory, with accumulation of intraneuronal sorbitol and depletion of myoinositol. This results in nerve dysfunction and damage.[5, 6, 7] These metabolic pathways are dependent on the enzyme aldose reductase, and the dysfunction and biochemical abnormalities of diabetic neuropathy can be improved by the administration of aldose reductase inhibitors.[5, 6] Diabetic neuropathy is usually diffuse and symmetrical but there are often focal features, particularly in diabetic amyotrophy. This suggests that microvascular disease contributes to pathogenesis. The demonstration of resistance in a diabetic nerve to the effects of ischaemia, possibly due to an adaptive process, is further evidence of the vascular theory.[7] The precise aetiology, however, remains unclear, but a combination of factors is likely.

The differential diagnosis would include femoral neuritis, sciatica, carcinomatous neuropathy, intra-pelvic neoplasm, motor neurone disease, thyrotoxicosis and

Guillain-Barré syndrome. Electrophysiological studies and a lumbar puncture would have helped to confirm the diagnosis, but we are satisfied that our investigations, and the marked improvement with control of his diabetes, indicate that the correct diagnosis of this case is diabetic amyotrophy.

REFERENCES

1. Garland H, Taverner D. Diabetic myelopathy. *Br Med J* 1953; **1**: 1405-8.
2. Garland H. Diabetic amyotrophy. *Br Med J* 1955; **2**: 1287-90.
3. Seddon PC, Smith CS. Adolescent diabetic amyotrophy. *Acta Paediatr Scand* 1988; **77**: 937-40.
4. Leedman PJ, Davis S, Harrison LC. Diabetic amyotrophy: reassessment of the clinical spectrum. *Aust NZ J Med* 1988; **18**: 768-73.
5. Jaspan J, Herold K, Maselli R, Bartkus C. Treatment of severely painful diabetic neuropathy with an aldose reductase inhibitor: relief of pain and improved somatic and autonomic nerve function. *Lancet* 1983; **2**: 758-62.
6. Ward JD. Diabetic neuropathy. *Br Med Bull* 1989; **45**: 111-26.
7. Thomas PK, Ward JD, Watkins PJ. Diabetic neuropathy. In: Keen H, Jarrett RJ, eds. Complications of diabetes. 2nd ed. London: Edward Arnold, 1982: 109-36.

The Ulster Medical Journal, Volume 61, No. 2, pp. 188 – 192, October 1992.

Case report

Metabolic and respiratory consequences of a glucose load in a hypoxic patient with cystic fibrosis

J S Elborn, T Jagoe, D J Shale

Accepted 3 March 1992.

Patients with cystic fibrosis are often in a poor nutritional state and require dietary supplementation to meet the energy requirements of an increased basal metabolic rate.[1] As lung disease progresses, energy requirements increase further to meet the stress of pulmonary sepsis and increased work of breathing.[1-4] Dietary supplementation involves an overall increase in energy intake predominantly in the form of carbohydrate, the most efficient energy substrate.[5,6] We report the metabolic and respiratory consequences of a glucose load, of equivalent energy value to that usually given to such patients as an overnight supplementary feed by fine bore nasogastric tube, in a hypoxic patient with cystic fibrosis who complained of breathlessness and lethargy after meals.

CASE REPORT. A 28-year-old male with cystic fibrosis was referred for assessment of respiratory failure and cor pulmonale. Cystic fibrosis was diagnosed at the age of five months because of repeated respiratory tract infection and failure to thrive. Sweat sodium at that time was 78 mmol/l (normal < 40 mmol/l). He remained well until the age of 26 when after a severe pulmonary exacerbation he reported progressive shortness of breath.

At admission he was cyanosed, arterial oxygen tension (PaO_2) 5·9 kPa with hypercapnoea, arterial carbon dioxide tension ($PaCO_2$) 6·85 kPa. There was marked pulmonary disease with finger clubbing, and bilateral coarse crepitations on examination. The Chrispin-Norman score based on changes on a chest radiograph was 21 points (0 = no disease, 38 = most severe).[7] Sputum culture had regularly grown *Pseudomonas aeruginosa* for more than five years during which time he received intermittent courses of intravenous antibiotics. He was undernourished (47·1 kg; body mass index (BMI) 15·6 kg/m^2); his Shwachman score was 40 points indicating an overall poor clinical condition (100 points = excellent; 40 or less = very severe).[8] He had recently developed diabetes mellitus

Respiratory Medicine Unit, City Hospital, Nottingham NG5 1PB.
J S Elborn, MD, MRCP, Senior Registrar.
T Jagoe, MB, BCh, Senior House Officer.
D J Shale, MD, FRCP, Senior Lecturer.

Correspondence to Dr J S Elborn, now Lecturer in Respiratory Medicine and Communicable Diseases, Llandough Hospital, Penarth, South Glamorgan CF6 1XX.

which was controlled with glibenclamide 10 mg daily. He was initially commenced on a carbohydrate restricted diet, but due to weight loss the amount of carbohydrate was increased to provide extra energy and improve his nutritional state.

Subsequently he reported symptoms of breathlessness, particularly after high carbohydrate meals. To investigate this breathlessness, prior to beginning overnight enteral feeding, he had a 75 gram glucose tolerance test (300 kcal) following an overnight fast. The response of plasma glucose, the resting carbon dioxide production ($\dot{V}CO_2$), oxygen uptake ($\dot{V}O_2$), minute ventilation ($\dot{V}E$) and capillary blood gas tensions were determined. Respiratory gas exchange (Gould Electronics) was measured for 10 minutes prior to each blood sample at 0, 60, 90, 120 and 180 minutes. Minute ventilation, oxygen uptake, carbon dioxide production and respiratory exchange ratio (RER) were calculated from mixed expired gas sampled every 20 seconds.[9] The mean of measurement for the second five minutes of each period is reported. The coefficient of variation for each parameter (oxygen uptake, resting carbon dioxide production, minute ventilation and respiratory exchange ratio) was between 4–8% for each five minute period. The glucose tolerance test was repeated two days later while breathing oxygen (fraction of oxygen in the inspired air (FIO_2) = 28%) by face mask to determine whether this further elevated arterial carbon dioxide tension.

The glucose load while breathing air caused a substantial increase in resting oxygen uptake, carbon dioxide production and the respiratory exchange ratio after two hours (Table). Minute ventilation and respiratory rate also increased. Plasma glucose increased substantially at between one and two hours in parallel with an increase in arterial carbon dioxide tension. When repeated, breathing oxygen, a similar plasma glucose response occurred but with a higher arterial carbon dioxide tension and the patient was more dyspnoeic. The glucose tolerance test was repeated five days later with 12 units of insulin given 20 minutes prior to the glucose load. The rise in plasma glucose was less, increasing by 2·5 mmol/l after 120 minutes and arterial carbon dioxide tension increased by 1·2 kPa. There was no difference in the blood gas responses when insulin was given compared to when not.

DISCUSSION

We have demonstrated potentially deleterious effects of an oral glucose challenge on resting metabolism and capillary blood gases in a diabetic patient with hypoxic chronic obstructive lung disease due to cystic fibrosis. Patients with cystic fibrosis are often markedly underweight and require nutritional support.[1,5] Nutrition becomes a particular problem as lung function deteriorates and patients develop respiratory failure.[1] Such patients often require energy supplementation either by mouth or by fine bore nasogastric tube feeding. Many supplements are high in glucose and may represent a considerable metabolic load for patients already in respiratory failure.[10,11] In ambulatory patients with chronic obstructive pulmonary disease, high carbohydrate diets increase dyspnoea and reduce exercise tolerance[4] though these diets seem to have little effect on arterial carbon dioxide tension.[12] In patients with respiratory failure in an intensive care unit, intravenous total parenteral nutrition with high glucose content feeds increases both carbon dioxide production and arterial carbon dioxide tension.[6] In our patient with established respiratory failure the glucose load caused a similar increase in carbon dioxide production, arterial carbon dioxide tension and minute ventilation and resulted in symptoms of dyspnoea and sleepiness.

TABLE

Respiratory gas exchange, capillary blood gases and blood sugar responses to 75g glucose breathing room air and 28% oxygen

	Minutes				
	0	60	90	120	180
(Room air)					
Oxygen uptake ($\dot{V}O_2$) (ml/min)	225	235	228	240	242
Carbon dioxide production ($\dot{V}CO_2$) (ml/min)	175	187	181	201	207
Respiratory exchange ratio (RER)	0·79	0·79	0·80	0·84	0·86
Minute ventilation ($\dot{V}E$) (l/min)	8·9	9·5	10·0	10·6	10·2
Respiratory rate (RR) (b/min)	24	25	27	28	25
Plasma glucose (mmol/l)	6·4	8·1	11·8	14·2	15·0
Arterial oxygen tension (PaO_2) (kPa)	5·6	6·4	6·8	7·3	7·1
Arterial carbon dioxide tension ($PaCo_2$) (kPa)	6·7	7·0	7·4	7·4	6·9
pH	7·39	7·38	7·36	7·25	7·36
(28% O_2)					
Plasma glucose (mmol/l)	5·0	11·4	14·4	16·2	16·1
Arterial oxygen tension (PaO_2) (kPa)	7·5	7·5	7·6	7·5	7·5
Arterial carbon dioxide tension ($PaCO_2$) (kPa)	6·8	7·7	7·7	7·4	7·5
pH	7·41	7·41	7·41	7·41	7·40
Room air + insulin 12u					
Plasma glucose (mmol/l)	7·5	8·8	11·8	10·0	9·6
Arterial oxygen tension (PaO_2) (kPa)	5·0	5·2	5·4	5·4	5·5
Arterial carbon dioxide tension ($PaCO_2$) (kPa)	6·6	6·8	7·4	7·8	7·0
pH	7·45	7·45	7·44	7·40	7·42

A carbohydrate load, because of the high respiratory quotient ($\dot{V}CO_2/\dot{V}O_2$) of glucose (RQ = 1) is known to increase carbon dioxide production relative to oxygen uptake to a greater extent than fat (RQ = 0·7).[13] The increase in carbon dioxide production stimulates ventilatory receptors, increasing minute ventilation to prevent arterial carbon dioxide tension from rising. In patients with severe respiratory impairment the increase in ventilation may not be sufficient to remove the excess CO_2 produced and so, as in this case, an increase in arterial carbon dioxide tension occurs. The observed increase in arterial oxygen tension after oral glucose has been noted before and is most likely due to an increase in alveolar ventilation.[13]

Diabetes mellitus occurs in 5–10% of adult patients with cystic fibrosis, and impaired glucose tolerance is seen in up to 50%.[14, 15] Diabetes is usually controlled without much difficulty using oral hypoglycaemic agents or small doses of insulin. In our patient diabetes had not been a major problem and plasma glucose levels were never greatly elevated. The glucose load after 12 u of insulin did not result in a substantial difference in arterial tension of carbon dioxide when compared to glucose given alone. This suggests that diabetes did not contribute to changes in blood gases, and the abnormal pulmonary function was primarily responsible for the rise in arterial carbon dioxide tension. Although insulin did not normalise the plasma glucose responses to the glucose load, it is possible that the elevated glucose levels may have contributed to the patient's symptoms. However, it might be expected that insulin would increase carbon dioxide production by increasing the availability of intracellular glucose.

This report demonstrates the value of assessing the blood gas response to an oral glucose tolerance test which contains a similar carbohydrate content to commercially available supplements for a single overnight tube feed. We had hoped to provide overnight enteral feeding and oxygen therapy together, but in view of the rise in arterial carbon dioxide tension following this combination and the patient's increased symptoms, we decided to use a high fat feed. Reduction in the proportion of carbohydrate in his diet in favour of fat and protein improved his postprandial dyspnoea. This case illustrates the need to consider the metabolic consequences of diet in patients with respiratory failure, particularly when consideration is being given to intensive energy supplementation.

Dr J S Elborn was supported by the Cystic Fibrosis Research Trust. The authors thank Mrs Marie Loughran for typing the manuscript.

REFERENCES

1. Vaisman N, Pencharz PB, Corey M, Canny GJ, Hahn E. Energy expenditure of patients with cystic fibrosis. *J Pediatr* 1987; **111**: 496-500.
2. Elborn JS, Shale DJ. Lung injury in cystic fibrosis. *Thorax* 1990; **45**: 970-3.
3. Elborn JS, Norman D, Delamere FM, Shale DJ. *In vitro* tumour necrosis factor-α secretion by monocytes from patients with cystic fibrosis. *Am J Respir Cell Mol Brol* 1992; **6**: 207-11.
4. Norman D, Elborn JS, Cordon SM, *et al.* Plasma tumour necrosis factor alpha in cystic fibrosis. *Thorax* 1991; **46**: 91-5.
5. Chase HP, Long MA, Lavin MH. Cystic fibrosis and malnutrition. *J Pediatr* 1979; **95**: 337-47.
6. Goodchild MC, Dodge JA. Cystic fibrosis. London: Baillière Tindall, 1985.

7. Chrispin AR, Norman AP. The systematic evaluation of the chest radiograph in cystic fibrosis. *Pediat Radiol* 1974; **2**: 101-6.

8. Shwachman H, Kulczycki LL. Long-term study of 105 patients with cystic fibrosis: studies made over a 5 to 14 year period. *Am J Dis Child* 1958; **96**: 6-15.

9. Riley M, Elborn JS, McKane WR, *et al.* Resting energy expenditure in chronic cardiac failure. *Clin Sci* 1991; **80**: 633-9.

10. Gieseke T, Gurushanthaiah G, Glauser FL. Effects of carbohydrates on carbon dioxide excretion in patients with airway disease. *Chest* 1977; **71**: 55-8.

11. Brown SE, Wiener S, Brown RA, Marcarelli PA, Light RW. Exercise performance following a carbohydrate load in chronic air-flow obstruction. *J Appl Physiol* 1985; **58**: 1340-6.

12. Angelillo VA, Sukhdarshan B, Durfee D, Dahl J, Patterson AJ, O'Donohue WJ. Effects of low and high carbohydrate feedings in ambulatory patients with chronic obstructive pulmonary disease and chronic hypercapnia. *Ann Int Med* 1985; **103**: 883-5.

13. Covelli HD, Black JW, Olsen MS, Beekman JF. Respiratory failure precipitated by high carbohydrate loads. *Ann Int Med* 1981; **95**: 579-81.

14. Handwerger S, Roth J, Gordon P, *et al.* Glucose intolerance in cystic fibrosis. *N Eng J Med* 1969; **281**: 451-61.

15. Hodson ME, Norman AP, Batten JC. Cystic fibrosis. London: Baillière Tindall, 1983.

The Ulster Medical Journal, Volume 61, No. 2, pp. 193 – 197, October 1992.

Case report

Aggressive myxoid liposarcoma of mediastinum

B S Sharma, J A McGuigan, H Bharucha, I C Bailey

Accepted 15 June 1992.

Liposarcomata most frequently originate in the interfascial plane of the thigh, or in the retroperitoneal tissues.[1] Primary development in the mediastinum is a rare occurrence, with less than 60 cases reported in the world literature.[2, 3, 4, 5] Histopathologically, liposarcomata are classified into four basic types: well differentiated, myxoid, round cell and pleomorphic, and the clinical behaviour and prognosis correlate closely with the histological sub-type. The development of metastases in well differentiated and myxoid tumours is rarer than in the other sub-types, but myxoid liposarcomata can metastasize as a late manifestation of the disease.

We report a patient with a myxoid liposarcoma of the anterior mediastinum, which exhibited aggressive behaviour and an unusual pattern of metastases at rare sites, including the spinal epidural space, which caused spinal cord compression.

CASE REPORT. The patient was a 30-year-old female who was admitted with a five day history of sharp pain in the right abdomen with discomfort in the right shoulder tip. Clinical examination revealed soft and tender hepatomegaly. Ultrasound showed a 13 cm solid mass in the liver and the liver function tests were consistent with intrahepatic obstruction. Chest X-ray revealed a mass in the antero-superior mediastinum, adjacent to the aorta. These tumours were further visualized by computerised tomography (CT), (Fig 1) and an ultrasound-guided fine needle biopsy from the liver mass was suggestive of a liposarcoma.

One month later, at thoracotomy, the mediastinal mass and subpleural deposits were excised, and subsequently the liver mass was removed by formal right hepatectomy. Pathological examination of both the mediastinal tumour and the

Royal Victoria Hospital, Belfast BT12 6BA.

Department of Neurosurgery.

B S Sharma, MB, MS, MCh, Registrar in Neurosurgery.
I C Bailey, MB, BCh, FRCS, Consultant Neurosurgeon.

Department of Pathology.
H Bharucha, MB, BS, FRCPath, Consultant Pathologist.

Thoracic Surgical Unit.
J A McGuigan, MB, FRCS, Consultant Thoracic Surgeon.

Correspondence to Mr Bailey.

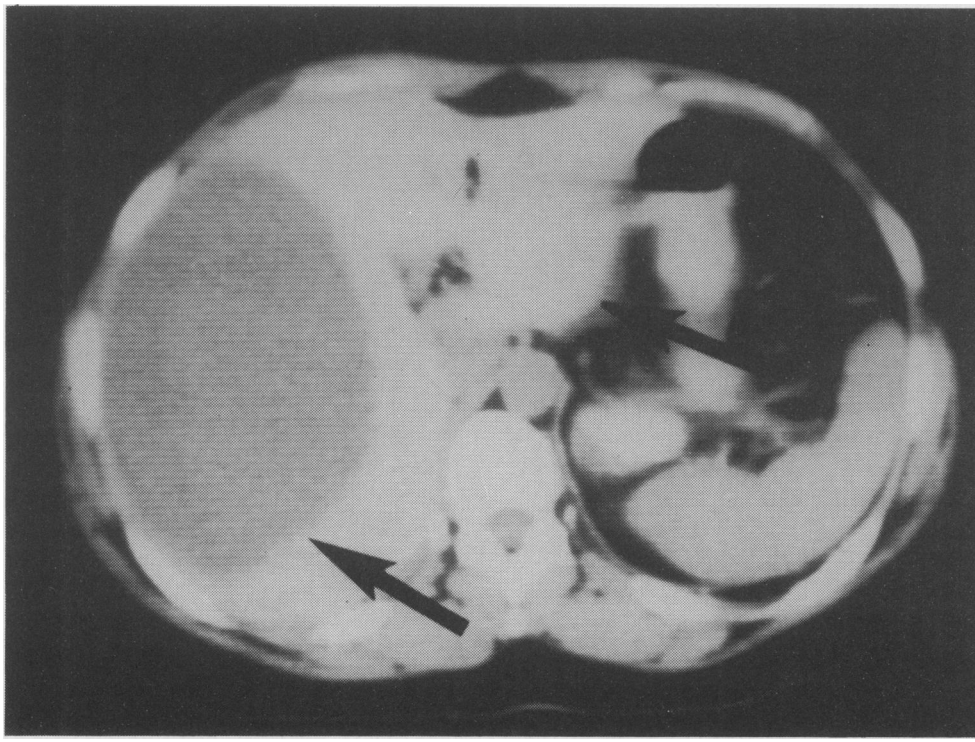


Fig 1. CT scan of abdomen. Arrows indicate tumours in the liver and the anterior mediastinum.

partial hepatectomy specimen revealed well-delineated fatty masses. In each case there was a good demarcation between tumour and normal tissue but no actual capsule was detected. It was concluded that the tumours were exhibiting expansile rather than infiltrative growth patterns. Histological examination of both tumours revealed a myxoid stroma with stellate cells and the characteristic plexiform capillary pattern seen in liposarcomata (Fig 2). The mitotic rate was not high and in view of the stromal change a diagnosis of myxoid liposarcoma was reached.

Immunohistochemistry showed a positive vimentin reaction in keeping with the mesenchymal origin of liposarcomata. Electron microscopy of tissue from the liver tumour showed a classical lipoblast with lipid droplets and a myxoid matrix surrounding the cell (Fig 3). Three months after operation, CT scan showed no recurrence in the mediastinum or liver, but additional lesions were present retroperitoneally in the region of the right adrenal gland, the crus of the diaphragm and the paravertebral gutter. Within the next year she developed three separate swellings, two in her groin and one in the posterior aspect of the left leg. These were excised and each specimen showed the same histological picture of a myxoid liposarcoma.

Ten months after initial presentation she was re-admitted complaining of pain in the right lower chest and a CT scan showed some increase in the size of the tumour in the right paravertebral region; this was excised through a right thoracotomy. Five days post-operatively she developed weakness of her legs,

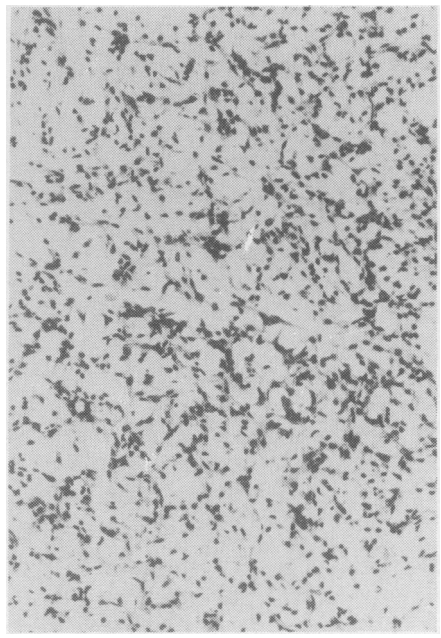


Fig 2. Liposarcoma showing myxoid stroma and prominent plexiform capillary pattern. Haemotoxylin and eosin. (× 25).

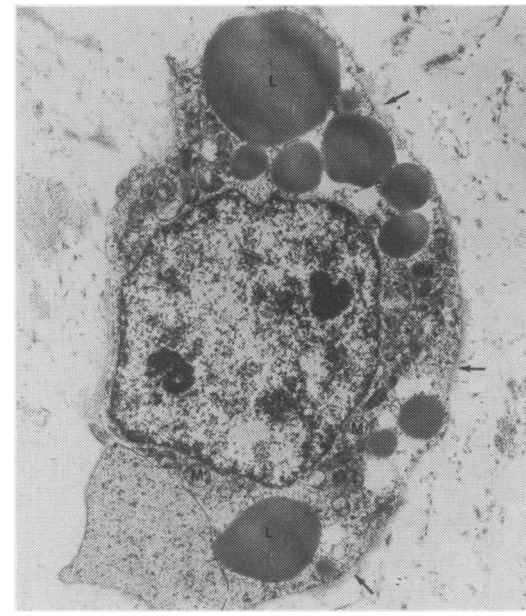


Fig 3. Electron microscopy, showing a lipoblast. Note the lipid droplets (L), mitochondria (M), and the focal external lamina (arrows). The cell is surrounded by a myxoid matrix. (× 200).

and retention of urine. Neurological examination showed a spastic paraparesis, with decreased pain sensation below the D8 dermatome. Myelography demonstrated complete extradural obstruction at the D6 level. At laminectomy, a 10 mm thick sheet of extradural tumour was found. The cranial and caudal limits of the tumour were defined and it was subtotally removed; there was no sign of vertebral body involvement. Post-operatively bladder function immediately improved, followed by complete sensory and motor recovery over the next week. Serial scans indicated that the liver had regenerated.

She later needed neurolysis of the intercostal nerves to control recurrent pain in the chest wall. Tumour in the anterior mediastinum, the paravertebral gutter and the retroperitoneal space recurred, but further surgery was thought to be of little avail. Subsequently, she developed gross ascites and oedema of the lower limbs, and died two years after presentation from pulmonary oedema and pneumonia. Autopsy was refused.

DISCUSSION

The age range of patients who have developed mediastinal liposarcoma is 14 months to 77 years, with a mean of 35 years.[2,6] There is no characteristic clinical history and the tumour may attain an enormous size before symptoms and signs of local compression occur. Our patient had no respiratory symptoms, and the lesion was suspected for the first time on chest X-ray. Slow growth with compression, rather than infiltration of the surrounding structures, together with the soft pliable nature of lipid tissue may be responsible for this very gradual clinical onset.[1,2]

Metastases from a liposarcoma depend on the size and location of the primary tumour, the histological differentiation and the mode of therapy used.[7] Mediastinal liposarcomata commonly metastasize to the lung, pleura and liver; rarely the kidney, adrenal, mediastinal lymph nodes, deltoid muscle or skin are involved.[8] Myxoid liposarcomata for unknown reasons tend to produce secondary lesions on the serosal surfaces of the liver, pericardium and the diaphragm, either alone or in combination with visceral metastases. Tumours of similar appearance may be found retroperitoneally, and in other soft tissues. There is no sharp distinction between multicentric and metastatic liposarcoma. Concurrent lesions in the mediastinum and liver of our patient, and the appearance shortly afterwards of additional lesions of similar histology in other unusual sites suggest that these were metastatic tumours. The time interval between the appearance of the primary and the secondary tumours is usually measured in years, and the rapid development of secondary tumours in this case is unusual. Metastases to the spinal canal, with neurological deficit, have been reviewed by Bogoth *et al.*[9] The absence of bony involvement, and the finding of a thick sheet of tumour extending over 3 vertebral segments, suggest an epidural metastasis, rather than direct extension from a paravertebral mass.

The CT appearance of a liposarcoma includes lack of clear demarcation from surrounding tissues, enhancement with intravenous contrast, and the low attenuation value of fat on the Hounsfield scale.[1, 5, 10, 11] In this case, CT scans were invaluable in determining the exact size and location of the tumours, and in the detection of recurrence at several sites.

Wide surgical excision is the treatment of choice, but the lack of discrete boundaries, and contiguity with the surrounding vital intrathoracic structures make this difficult.[6, 8, 12] Partial resection, followed by radiotherapy, may be useful, although the value of this approach is questioned by several authors.[2, 3, 4] Chemotherapy is ineffective, and radiotherapy is accepted as the standard treatment for metastases.[7] With spinal cord compression, rapid neurological deterioration is usually due to ischaemia of the neural tissues.

Decompression which must be carried out urgently, was successful in reversing the neurological deficit in this case, as it was in the previous report.[9] The prognosis in respect of a liposarcoma depends on the histological type and the topographical location of the tumour.[2, 7] The outlook is better in well-differentiated and in the myxoid variety than in other histological sub-types; the prognosis is less favourable when the tumour arises in the mediastinum.[3] Overall average survival is about 4 years.[12] The present case belongs to the group of liposarcomata supposed to have a good prognosis, but the early appearance of metastases implies unusually aggressive behaviour.

The authors wish to acknowledge the contribution made by the late Dr Patrick Watt to the original diagnosis of this case. The authors also wish to thank the Electron Microscopy Unit, Department of Pathology, Royal Victoria Hospital for their contribution to the diagnosis.

REFERENCES

1. DeSantos LA, Ginaldi S, Wallace S. Computed tomography in liposarcoma. *Cancer* 1981; **47**: 46-54.

2. Plukker JTM, Joosten HJM, Rensing JBM, Van Haelst UJGM. Primary liposarcoma of the mediastinum in a child. *J Surg Oncol* 1988; **37**: 257-63.
3. Shibata K, Koga Y, Onitsuka T, *et al.* Primary liposarcoma of the mediastinum — a case report and review of the literature. *Jpn J Surg* 1986; **16**: 277-83.
4. Standerfer RJ, Armistead SH, Paneth M. Liposarcoma of the mediastinum: report of two cases and a review of the literature. *Thorax* 1981; **36**: 693-4.
5. Yang R, Elliston L, Paterson R, Sahmel R. Dysphagia and cough in a patient with a posterior mediastinal mass. *Chest* 1987; **92**: 529-30.
6. Schweitzer DL, Aguam AS. Primary liposarcoma of the mediastinum. *J Thorac Cardiovasc Surg* 1977; **74**: 83-97.
7. Enzinger FM, Weiss SW. Soft tissue tumours. St Louis: CV Mosby, 1988: 346-82.
8. Razzuk MA, Urschel HC, Race GJ, Kingsley WB, Paulson DL. Liposarcoma of the mediastinum: case report and review of the literature.*J Thorac Cardiovasc Surg* 1971; **61**: 819-26.
9. Bogoth ER, English E, Perrin RG, Tator CH. Successful surgical decompression of spinal extradural metastases of liposarcoma. *Spine* 1983; **8**: 228-35.
10. Mendez G Jr, Isikoff MB, Isikoff SK, Sinner WM. Fatty tumours of the thorax demonstrated by CT. *Am J Roentgenol* 1979; **133**: 207-12.
11. Munk PL, Müller NL. Pleural liposarcoma: CT diagnosis. *J Comput Assist Tomogr* 1988; **12**: 709-10.
12. Prohm P, Winter J, Ulatowski L. Liposarcoma of the mediastinum: case report and review of the literature. *Thorac Cardiovasc Surg* 1981; **29**: 119-21.

The Ulster Medical Journal, Volume 61, No. 2, pp. 198 – 201, October 1992.

Case report

Lipoid pneumonia; an unsuspected diagnosis

J A McKnight, E McVeigh, J D Biggart, J R Hayes

Accepted 16 June 1992.

We describe a case of a woman who presented with a history suggestive of respiratory infection which was felt to be secondary to bronchial carcinoma, but was subsequently found to have exogenous lipoid pneumonia. In retrospect we wonder if this diagnosis could have been made preoperatively, and if this would have altered our management.

CASE REPORT. A 74-year-old female was admitted with a two month history of increasing breathlessness and malaise. Prior to this she had been fit and active. She had begun to feel unwell and developed a cough, initially producing pink sputum which later became green in colour. Two months prior to admission she developed severe right sided pleuritic chest pain on coughing. Initially she was breathless on walking half a mile but gradually became dyspnoeic at rest. Her appetite decreased but she had not noticed any loss of weight. She had smoked 20 – 30 cigarettes daily until the age of 66 years. There was no previous exposure to dust or asbestos. She had had pneumonia at the age of 16 but there was no previous history of tuberculosis.

On admission she was feverish (38·8 C) but had no anaemia, cyanosis, lymphadenopathy or finger clubbing. Her respiratory rate was 20/min. She had signs of decreased chest expansion, dullness to percussion and reduced air entry and vocal resonance in the right mid zone. Pulse rate was 100/min, blood pressure 170/60 mmHg. ESR was 100 mm per hour, haemoglobin 9·7 g/dl, mean cell volume 83 fl and white cell count 29·1 10^9/l (91% neutrophils). Serum albumin was 24 g/l (normal 35 – 50), alkaline phosphatase 474 μ/l (90 – 280) and gammaglutamyl transferase 96 μ/l (5 – 60). Arterial blood pH was 7·50 (7·35 – 7·44), pO2 9·73 KPa (12·0 – 14·6), pCO2 4·2 KPa (4·7 – 6·0) and standard bicarbonate 26·6 mmol/l (22 – 26). Repeated sputum microscopy revealed some polymorphoneuclear leucocytes, epithelial cells and gram positive cocci, but culture grew commensal organisms only. Sputum cytology and microscopy for acid and alcohol fast bacilli were negative. Chest X-ray (Fig 1) showed a large mass in the lateral part of the right midzone. CT scan showed the large mass to be in the right upper lobe extending out to the chest wall and

Belfast City Hospital, Belfast BT9 7AB.

J A McKnight, MD, MRCP, Senior Registrar in Medicine.

E McVeigh, MB, BCh, Senior House Officer.

J R Hayes, MD, FRCP, Consultant Physician.

J D Biggart, MD, FRCPath, Consultant Pathologist.

Correspondence to Dr Hayes.

involving it at one point. Enlarged lymph nodes were seen in the pre-cranial area. There was no enhancement with contrast. Bone scan was normal.

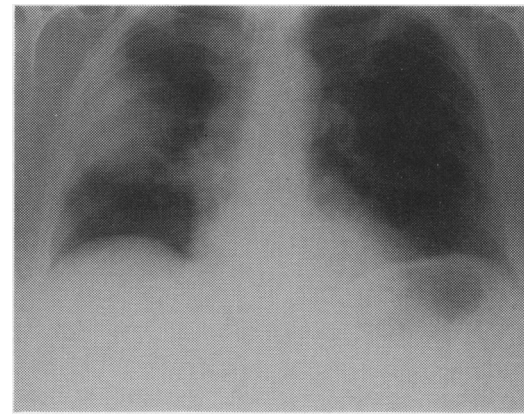


Fig 1. Chest X-ray on admission showing consolidated areas in the right mid zone.

She was initially treated with oral ampicillin. Bronchoscopy was attempted three days later, but mild laryngospasm caused immediate anoxia and the procedure had to be abandoned. Treatment was changed to intravenous erythromycin, her temperature fell after two days and remained normal. Three days later she had a large haemoptysis, and the chest X-ray had deteriorated. During the following week her condition gradually improved again and her haemoglobin rose to 11·6 g/dl, white cell count 8·3 10^9/l and all biochemistry had returned to normal. Bronchoscopy was repeated under general anaesthesia, and biopsy showed occasional inflammatory cells, but no dysplasia or malignancy.

Three weeks after her initial presentation a thoracotomy revealed a dense right upper lobe mass which was removed. She made an uneventful postoperative recovery and remains well with no symptoms.

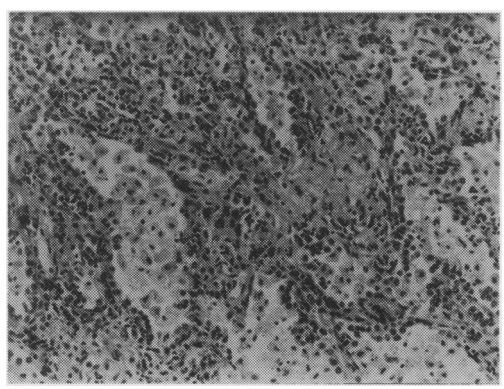


Fig 2. Area showing lipid-laden macrophages. The septa are infiltrated by chronic inflammatory cells. H & E × 630.

The right upper lobectomy specimen was virtually replaced by a solid greyish-white mass (7 × 4 × 4 cm) with overlying pleural thickening. The histological features were dominated by intra-alveolar collections of lipid-filled macrophages associated with marked interstitial fibrosis, and inflammatory infiltrates of lymphocytes and plasma cells (Fig 2). Many of the small arteries showed fibrosis. The major bronchi were not obstructed by malignancy or other lesions. The appearances were those of chronic lipoid pneumonia.

Subsequent questioning of the patient revealed that she had repeatedly used a liquid paraffin laxative at night when on holiday two months previously. She had noticed no dysphagia at that time.

DISCUSSION

Most diagnoses are reached in medicine following clinical history-taking and examination. In some cases patients initially fail to report events which may later

prove to be of importance. This is well demonstrated in the present case where a woman who presented with a respiratory illness and was found to have a mass in her right lung field, gave no history of aspiration, and was felt to have a lung neoplasm. The history of liquid paraffin ingestion was obtained only by specific questioning after the histological diagnosis was obtained.

Lipoid pneumonia was first described by Laughlen in 1925[1] who found consolidated areas of lung, which contained oil, in autopsies of children. This oil was situated in phagocytic vacuoles of mononuclear endothelial leucocytes and stained positively with Sudan III. The children had received oil preparations as part of their treatment. Laughlen then produced similar changes by innoculating rabbits with oil confirming the relationship of lipoid pneumonia with exogenous administration of oil. Since this first report others[2] have noted the diversity of possible presentations of lipoid pneumonia, and its ability to mimic other conditions. Patients may be asymptomatic or diagnosed with acute or chronic pneumonitis, or as in this case may simulate carcinoma of the lung.[2] The clinical presentation depends on the amount and the characteristics of the ingested oil. A high free fatty acid content and a high viscosity stimulates greater pulmonary damage.[3,4] Liquid petroleum is a mixture of refined hydrocarbons of high viscosity, which our patient took as a laxative on retiring to bed on holiday. Fox and Bartlett[5] noted that patients who take mineral oil at bedtime are particularly susceptible to lipoid pneumonia. A study of 389 chronically ill patients found 14·6% incidence of lipoid pneumonia.[6] The commonest reason for ingestion of oil in this group was for constipation. Other reports have described lipoid pneumonia caused by lip gloss,[7] aerosol lubricant[8] and inhalation of liquid paraffins in a female fire eater.[9]

Lipoid pneumonia may be difficult to diagnose. Specific questioning about oil ingestion may be required. Demonstration of a diffuse multilobar infiltrate or a well-circumscribed homogeneous infiltrate in the lower lobes[5] on chest X-ray may suggest the diagnosis, but other authors have required open lung biopsy to establish the diagnosis.[2,7,10,11,12] If lipoid pneumonia is suspected, microscopic examination of sputum or bronchial washings for fat and oil laden macrophages may be useful diagnostically.[6,13] More recently characteristic absorption on computed tomography,[14] or magnetic resonance scan[15] have been described.

Treatment of lipoid pneumonia is to avoid the offending agent. Repeated innoculation may otherwise result in recurrent acute respiratory inflammation.[2] Bacterial superinfection requires antibiotic treatment. Some authors have suggested a role for corticosteroid therapy[10,11] but others have found no benefit.[16,17] Diagnosis of this rare condition using some of the methods reviewed above might have resulted in gradual resolution of lipoid pneumonia and avoided thoracotomy and lobectomy.

REFERENCES

1. Laughlen GF. Studies on pneumonia following naso-pharyngeal injections of oil. *Am J Pathol* 1925; **1**: 407-15.

2. Sodeman WA, Stuart BM. Lipoid pneumonia in adults. *Ann Int Med* 1946; **24**: 241-53.

3. Pinkerton H. The reaction to oils and fats in the lungs. *Arch Pathol* 1928; **5**: 380-401.

4. Gerarde HW. Toxicological studies on hydrocarbon: the aspiration hazard and toxicity of hydrocarbons and hydrocarbon mixtures. *Arch Environ Health* 1963; **6**: 329-41.
5. Fox M, Bartlett JG. Lipoid pneumonia. In: Baum GL, Wolinsky E (eds). Textbook of pulmonary disease, 3rd ed. Boston: Little Brown and Company, 1983: 605-12.
6. Volk BW, Nathanson L, Losner S, Slade WR, Jacobi M. Incidence of lipoid pneumonia in a survey of 389 chronically ill patients. *Am J Med* 1951; **10**: 316-24.
7. Becton DL, Lowe JE, Falletta JM. Lipoid pneumonia in an adolescent girl secondary to use of lip gloss. *J Pediatr* 1984; **105**: 421-3.
8. Glynn KP, Gale NA. Exogenous lipoid pneumonia due to inhalation of spray lubricant (WD – 40 lung). *Chest* 1990; **97**: 1265-6.
9. Beermann B, Christensson T, Moller P, Stillstrom A. Lipoid pneumonia: an occupational hazard of fire eaters. *Br Med J* 1984; **289**: 1728-9.
10. Scully RE, Galdabini JJ, McNeely BU. Case records of the Massachusetts General Hospital, Case 19-1977. *N Engl J Med* 1977; **296**: 1105-11.
11. Ayvazian LF, Steward DS, Merkel CG, *et al.* Diffuse lipoid pneumonitis successfully treated with prednisone. *Am J Med* 1967; 930-4.
12. Cornacchia DJ, Snyder CH, Dupont DC, Yaron NS. Exogenous lipoid pneumonia. *J Am Osteopath Assoc* 1989; **89**: 659-63.
13. Weill H, Ferrand VJ, Gay RM, Ziskind MM. Early lipoid pneumonia. *Am J Med* 1964; **36**: 370-6.
14. Wheeler PS, Stitik FP, Hutchins GM, Klinefelter HF, Siegelman SS. Diagnosis of lipoid pneumonia by computed tomography. *JAMA* 1981; **245**: 65-6.
15. Carette MF, Grivaux M, Monod B, Capeau F, Lebreton C, Bigot JM. MR findings in lipoid pneumonia. *Am J Roentgenol* 1989; **153**: 1097-8.
16. Marks MI, Chicoine L, Legere G, Hillman E. Adrenocorticosteroid treatment of hydrocarbon pneumonia in children — a cooperative study. *J Pediatr* 1972; **81**: 366-9.
17. Steele RW, Conklin RH, Mark HM. Corticosteroids and antibiotics for the treatment of fulminant hydrocarbon aspiration. *JAMA* 1972; **219**: 1434-7.

The Ulster Medical Journal, Volume 61, No. 2, pp. 202 – 204, October 1992.

Case report

Iatrogenic hydrothorax complicated by reactive pleural effusion

A J Ritchie, Susan Atkinson, D L Coppel

Accepted 25 August 1992.

Central venous catheterization, an everyday procedure in intensive care units, has a number of well recognized complications.[1] Venous wall perforation resulting in hydrothorax is unlikely when the right internal jugular vein is cannulated by an experienced operator using a J-tipped guide wire and the Seldinger technique.[2] If perforation of the vein does occur, complications within the chest cavity can result in considerable morbidity, or death.[3,4] We report the result of propofol and atracurium infusion into the chest cavity and discuss factors related to malplacement of the venous catheter.

CASE REPORT. Following a neurosurgical operation an 80 kg caucasian male aged 17 years was electively hyperventilated. To facilitate anaesthesia and monitoring of cardiac filling pressure, a right internal jugular central venous catheter was inserted using the Seldinger technique and J-tipped guide wire. Although slight resistance was reported when the guide wire was inserted, and radiologically the catheter tip was positioned in an abnormal position (Fig 1), venous blood was readily aspirated from all ports of a 7FR triple lumen polyurethane catheter, and infusion of anaesthetic agents was commenced. In addition it was noted that a major problem, probably related to aspiration at the time of neurosurgical trauma, had occurred in the left chest. Initially high infusion rates of propofol (200 – 400 mg/hr) and atracurium (150 – 250 mg/hr) were required to facilitate ventilation. A total of 1800 ml was infused. Morphine was then infused peripherally at 3 – 7 mg/hr.

Haemodynamically the patient remained stable but oxygenation deteriorated over nine hours, pulmonary arterial oxygen concentration falling from 280 mmHg to 91 mmHg despite the fraction of inspired oxygen being 80% ($FI O_2$ 0·8) and the use of positive and expiratory pressure (PEEP). Bronchial lavage was undertaken using a fibreoptic bronchoscope to treat left basal collapse. Oxygenation improved and the left lung re-expanded after evacuation of multiple small clots and then the

Regional Thoracic Surgical Department, Royal Victoria Hospital, Belfast BT12 6BA.
A J Ritchie, BSc, MB, BCh, FRCSEd, Research Fellow.

Department of Clinical Anaesthesia, Royal Victoria Hospital, Belfast BT12 6BA.
Susan Atkinson, MB, BCh, FFARCS, Senior Registrar.
D L Coppel, MB, BCh, FFARCS, Consultant Anaesthetist.

Correspondence to Mr Ritchie, Department of Cardio-Thoracic Surgery, Aberdeen Royal Infirmary, Aberdeen.

catheter tip was partially withdrawn. Repeat X-ray then showed full re-expansion of the left base but persistent abnormal catheter tip position. In addition, the right pleural cavity demonstrated diffuse opacity which was consistent with fluid within the pleural space (Fig 2). Throughout, central venous pressure remained normal.

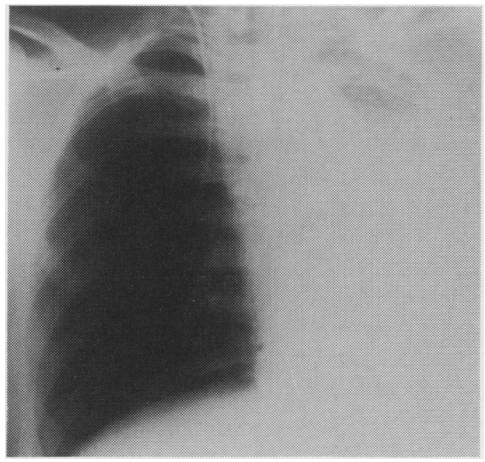


Fig 1. Initial chest X-ray. Note the position of the catheter is outwith the 'cannula tip positioning zone' in the right chest. The left side indicates collapse of the left base and possible effusion most likely related to aspiration in the bronchial tree.

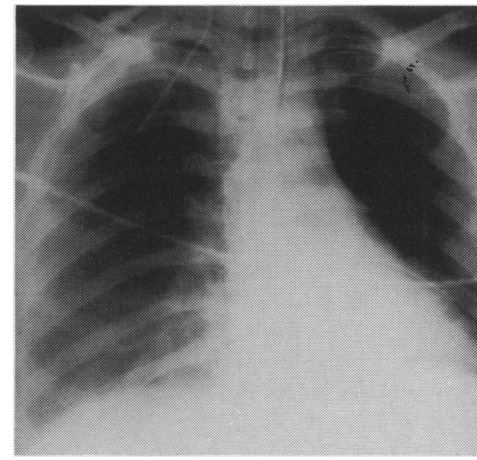


Fig 2. Subsequent to bronchoscopic aspiration of the left tracheobronchial tree and improvement in the left chest. Withdrawal of the cannula tip now shows it clearly in an abnormal position. Note the absence of any clear radiological sign of fluid in the right chest cavity — nine hours after insertion of the cannula and infusion of 1900 ml of fluid.

At 24 hours, further X-ray confirmed a right-sided hydropneumothorax on the same side as the venous cannula. This was associated with further deterioration in peripheral arterial oxygen tension to 143 mmHg despite increasing the inspired oxygen concentration to 90% and continued use of positive and expiratory pressure of 2·5. The central venous catheter was removed and propofol and atracarium were subsequently infused peripherally. Tube thoracostomy immediately drained 1900 ml of bloodstained fluid containing propofol and atracarium of similar volume to that infused. This allowed the right lung to re-expand and arterial oxygenation improved.

Over the subsequent 72 hours a total of 500 ml straw-coloured pleural effusion was evacuated. The effusion was found to be an exudate protein 3·7 g/l and neither it nor cultures from the tips of the central venous line or chest drain produced bacterial growth. Subsequently the chest drain was removed without further complication and, following tracheostomy, the patient was returned for long-term care to the neurosurgical unit.

DISCUSSION

A number of important points can be made from this case in which propofol and atracarium were inadvertently infused into the right chest cavity as a result of malplacement of a right internal jugular venous cannula.

Three consecutive clues could have suggested that the cannula was incorrectly placed. On insertion, a sensation of resistance indicates the need for replacement of the cannula. The withdrawal of blood does not guarantee correct placement because the most proximal portal may be inside the vein, allowing withdrawal of blood while the distal tip is outside the wall. Movement of a patient from the prone to supine position may result in sufficient movement of the tip to take it on through the wall. The X-ray taken to confirm the position of the catheter showed that the tip lay outside the 'catheter tip positioning zone'[4] and neck flexion and extension can move this between 1·5 and 3 cm. It is generally agreed that the tip should lie 3 – 4 cm above the superior vena cava, but the position in the present case was deemed acceptable since venous blood was readily aspirated even after initial suspicion had been aroused. At this time, attention was drawn to the significant complication in the left lung which was thought to be the major problem. The third clue came from the abnormally high dose of propofol and atracarium required to facilitate ventilation, and despite dealing with the major complication in the left lung, the arterial oxygen saturation did not improve as expected. Withdrawal of the cannula by several centimetres at this point still allowed venous blood withdrawal, and although the X-ray now showed a clearly abnormal position, there was no obvious sign of collapse or effusion into the right chest cavity. All the X-rays were taken in the supine position, and since up to 500 ml fluid can accumulate without detection on chest films, this delayed the diagnosis even further.

The effects of propofol and atracarium within the pleural cavity have not previously been reported. The thoracic surgeon should be aware of the potential problems, and removal of the offending fluid and re-expansion of the lung may not be enough. Despite being regarded as innocuous to endothelial cells when given intravenously, this combination of agents resulted in an exudative inflammatory process within the chest cavity. Infusion of hyperosmotic fluids, such as used in parental nutrition, can result in pulmonary oedema, chest wall abscess and recurrent pleural effusion[4] which may mandate continued drainage of the chest after the volume of the infused load has been removed.

There should always be a high index of suspicion for incorrect placement of venous catheters despite distraction by apparently more obvious and pressing complications elsewhere in the chest.

We thank Miss May Weller for typing this manuscript.

REFERENCES

1. Scott WL. Complications associated with central venous catheters. A survey. *Chest* 1988; **94**: 1221-4.
2. Belani KG, Buckley JJ, Gordon JR, Castaneda W. Percutaneous cervical central venous line placement: a comparison of the internal and external jugular vein routes. *Anesth Analg* 1980; **59**: 40-4.
3. Defalque RJ, Campbell C. Cardiac tamponade from central venous catheters. *Anesthesiology* 1979; **50**: 249-52.
4. Oakes DD, Wilson RE. Malposition of a subclavian line. Resultant pleural effusions, interstitial pulmonary edema, and chest wall abscess during total parenteral nutrition. *JAMA* 1975; **233**: 532-3.

The Ulster Medical Journal, Volume 61, No. 2, pp. 205 – 206, October 1992.

Case report

Allergy to the ethylenediamine component of aminophylline

M Toal, Anne Kinney, R Fulton

Accepted 28 July 1992.

Intravenous aminophylline is used as a first line treatment for bronchial asthma. This preparation consists of theophylline or a salt of theophylline dissolved in a solution of ethylenediamine, which confers greater solubility and reduces the alkalinity of the drug.[1] In clinical usage side effects of tachycardia, seizures and nausea are well known. We report on the much less common, but clinically important problem of allergy to the ethylenediamine component of aminophylline.

CASE REPORT. A 57-year-old lady was admitted with an infective exacerbation of chronic bronchitis and emphysema. Initial treatment was with nebulized bronchodilators, and intravenous aminophylline, hydrocortisone and ampicillin. Later on the day of admission the patient developed an erythematous macular rash, initially on the trunk and limbs, but becoming confluent with periorbital oedema. The rash was hot, itchy and uncomfortable, and the patient had paraethesia in her limbs. There was no clinical evidence of anaphylaxis and exfoliation did not occur. No history of drug allergy was given and medical notes were unavailable at this time. It was suspected the allergen was ampicillin, but discontinuation of this drug provided no relief. The patient remained on nebulized bronchodilators and an intravenous infusion of hydrocortisone and aminophylline.

When the medical notes became available, close scrutiny revealed attendance at the ENT Clinic with otitis externa three years previously. At that time, treatment with Tri-Adcortyl cream (Squibb; triamcinolone acetate, gramicidin, neomycin, nystatin, ethylenediamine) was commenced, but later discontinued when a rash occurred around the ear. She was referred to the dermatology clinic where patch testing was performed. A strong positive reaction was found to ethylenediamine (1%) which is a standard allergen in the European Standard Battery. She had not received aminophylline preparations in the past. The infusion of aminophylline was therefore discontinued and the rash subsided over the next few days. It is still possible that the allergen was ampicillin but a clear link to ethylenediamine is evident.

Altnagelvin Area Hospital, Londonderry BT47 1SB.
M Toal, MB, BCh, Senior House Officer.
Anne Kinney, MB, BCh, Senior House Officer.
R Fulton, MB, MRCP, Consultant Dermatologist.

Correspondence to Dr Toal.

DISCUSSION

Ethylenediamine is a substance used as a binding agent in a number of products, principally Tri-Adcortyl cream and aminophylline injection and enema. Reports of sensitization have been reported in hospital practice and industry.[2,3] In addition, cross reaction has been observed with antihistamines[4,5] and piperazine.[6] The allergic reaction can result in an exfoliative erythrodermic dermatitis which carries a significant morbidity and mortality. Neither the presence of ethylenediamine, nor the potential cross reaction are listed in the manufacturer's data sheet for Tri-Adcortyl cream, or for any of the commonly used aminophylline preparations. They are, however, mentioned in Martindale and the British Pharmacopoeia.

This case illustrates the importance of careful examination of medical notes for such allergies, and clear warnings on the front cover of notes. It is also important to warn general practitioners, and indeed the patients themselves, of the potential risk. It is also worth remembering that in cases of drug allergy, the most obvious suspect is not always the correct one.

We thank Dr J G Daly, Consultant Physician, Altnagelvin Hospital for permission to report this case.

REFERENCES

1. Martindale. The Extra Pharmacopoeia. 29th Ed. The Pharmaceutical Press, 1989: 1521-6.
2. Provost TT, Jillson OF. Ethylenediamine contact dermatitis. *Arch Dermatol* 1967; **96**: 231-4.
3. Petrozzi JW, Shore RN. Generalized exfoliative dermatitis from ethylenediamine. *Arch Dermatol* 1967; **112**: 525-6.
4. White MI, Douglas WS, Main RA. Contact dermatitis attributed to ethylenediamine. *Brit Med J* 1978; **i**: 415-6.
5. Fisher AA. Instructions for the ethylenediamine sensitive patient. *Cutis* 1976; **13**: 27.
6. Calnan CA. Occupational piperazine dermatitis. *Contact Dermatitis* 1975; **1**: 126.
7. Burry JN. Ethylenediamine sensitivity with a systemic reaction to piperazine citrate. *Contact Dermatitis* 1978; **4**: 380.

The Ulster Medical Journal, Volume 61, No. 2, pp. 207 – 208, October 1992.

Case report

Haemoperitoneum following gallbladder necrosis

Sigrid E Refsum, R H Wilson, G Blake

Accepted 20 September 1992.

Acute cholecystitis is usually treated conservatively, and surgery performed electively. Complications of cholecystitis such as empyema and perforation may be difficult to diagnose preoperatively. Delay in diagnosis and subsequent operative intervention results in increased morbidity and mortality. Prompt recognition and management are necessary to improve the outcome.

CASE REPORT. A 54-year-old mentally handicapped man was admitted with a one week history of vomiting, rigors and periumbilical pain. Initially he was well, haemodynamically stable and not clinically anaemic, jaundiced or pyrexic. His abdomen was soft, with slight tenderness in the right hypochondrium and 4 cm of hepatomegaly.

Four hours later he suddenly became shocked, pulse rate 120 per minute, blood pressure 70/0 mmHg. He developed abdominal guarding with rebound tenderness, and bowel sounds were absent. Haemoglobin was 5·6 g/dl, white cell 31,000. Serum sodium, urea, potassium and amylase concentration were normal. Liver function tests were mildly elevated, serum bilirubin 15 mmol/l (3 – 18), gamma glutamyl transpeptidase 141 mmol/l (7 – 64), asparate transaminase 140 mmol/l (10 – 42), and alkaline phosphatase 171 mmol/l (26 – 88).

Emergency laparotomy revealed a malodorous mass of necrotic tissue and fresh clot around the gallbladder bed and there were two litres of blood in the peritoneal cavity. No gallbladder tissue or gallstones were found. There was a brisk ooze from the cavity in the liver, but no definite bleeding point could be identified. The clot and free blood were removed, saline and tetracycline lavage was performed and the area packed. At a second laparotomy 36 hours later the packs were removed uneventfully. Histopathological examination of the necrotic mass showed no evidence of gallbladder tissue.

Preoperative blood cultures had grown *streptococcus faecalis*; he was treated with intravenous gentamicin, penicillin and metronidazole with good clinical response. Postoperatively he recovered well and went home three weeks later. On abdominal ultrasound scan six months later a gallbladder remnant was not visualised.

Daisy Hill Hospital, Newry, Co Down BT35 8BR.

Sigrid Refsum, BSc, MB, FRCS, Registrar in Surgery.
R H Wilson, MB, FRCS, Registrar in Surgery.
G Blake, MD, FRCS, Consultant Surgeon.

Correspondence to Miss Refsum, Department of Surgery, The Queen's University of Belfast, Institute of Clinical Science, Belfast BT12 6BJ.

DISCUSSION

We assume that the course of events underlying this case was acute cholecystitis with empyema formation, then subsequent perforation into the liver bed, abscess cavity formation and finally massive secondary haemorrhage into the peritoneal cavity. The underlying disorder may have been either vascular or calculous disease. Whether this was on the basis of vascular pathology or stones is unknown. His mental handicap and poor description of symptoms led to the delayed diagnosis.

Acute cholecystitis is one of the commonest causes of emergency admission to hospital.[1] While the vast majority resolve with conservative treatment, the reported incidence of gallbladder empyema is from two to nine percent,[2, 3] and for gallbladder perforation two to 17 percent.[4] The clinical signs and symptoms of empyema are virtually indistinguishable from acute cholecystitis[2, 5, 6] and result in diagnostic problems in the absence of ultrasonography. Physical signs are scanty or non-specific, with right hypochondrial pain, fever, nausea and vomiting as the main features.[5] Leucocytosis and non-specific deranged liver function tests are common.[2]

Correct preoperative diagnosis is made in less than half the cases,[4] and the reported mortality rate varies from six to 25 percent.[3, 5] Identified risk factors include increasing age, male sex and concomitant medical disease.[6] The delay in presentation of patients with an empyema of the gallbladder partly explains the high mortality rate.[2] Only 30 percent of patients had symptoms suggestive of gallbladder disease and 17 – 27 percent were acalculous.[3, 4]

Haemorrhage as the principal manifestation of gallbladder perforation has been reported in the literature only in 43 cases between 1858 and 1992.[7, 8] It is often impossible to identify the exact bleeding point at operation and haemostasis may only be accomplished by perihepatic packing.[9] There have been no previous reports of total necrotic disintegration of the gallbladder.

Awareness that complications of cholecystitis such as empyema and perforation of the gallbladder are more likely to occur in patients who present with a lengthy history should lead to urgent investigation with ultrasonography and early cholecystectomy.

REFERENCES

1. Krogh J. Empyema of the gallbladder: a case with unusual presentation. *Acta Chir Belg* 1989; **89**: 204-5.
2. Thorton JR, Heaton KW, Espiner HJ, Eltringham WK. Empyema of the gallbladder — reappraisal of a neglected disease. *Gut* 1983; **24**: 1183-5.
3. Fry DE, Cox RA, Harbrecht PJ. Empyema of the gallbladder: a complication in the natural history of acute cholecystitis. *Am J Surg* 1981; **141**: 366-9.
4. Lam KH, Wong J, Lim STK, Ong GB. Acute suppurative cholecystitis: a retrospective study of 173 cases. *Aust N Z J Surg* 1979; **49**: 23-8.
5. Chua CL, Cheah SL, Chew KH. Empyema of the gallbladder. *Ann Acad Med Singapore* 1988; **17**: 447-50.
6. Roslyn JJ, Thompson JE, Darvin H, DenBesten L. Risk factors for gallbladder perforation. *Am J Gastroent* 1987; **82**: 636-40.
7. Shiina S, Hisada T, Tagawa K, *et al.* Massive intraperitoneal haemorrhage due to rupture of the gallbladder. *ROFO* 1987; **147**: 568-9.
8. Syme RG, Thomas EJ. Massive haemoperitoneum from transhepatic perforation of the gallbladder: a rare complication of cholelithiasis. *Surgery* 1989; **105**: 556-9.
9. Becker WF. Haemoperitoneum as a complication of acute cholecystitis: a report of two cases. *Amer Surg* 1968; **34**: 88-93.

The Ulster Medical Journal, Volume 61, No. 2, pp. 209 – 210, October 1992.

Book reviews

1990 Public Health Matters. The Third Annual Report of the Director of Public Health. Dr G J Scally, MSc, FFPHM. Prepared by The Department of Public Health Medicine, Eastern Health and Social Services Board, Belfast, October 1991. (pp 82. £7.00).

The managerial revolution in the health services of the Thatcher years included the field of public health: thus from 1989[1] the DHSS (NI) required each Area Board to designate a consultant in Public Health Medicine as the Board 'Director of Public Health' to oversee 'not only efforts to preserve health . . . but also the provision of effective and efficient services to restore the sick to health . . . and to reduce suffering, disability and dependence'.[2] Each Director published his first annual report (for 1988) in October 1989: the subject of this review is the third annual report by Dr Gabriel Scally, Director of Public Health of the Eastern Board. These reports are not to be confused with the *Annual Reports* of the Boards, their various *Supplements,* nor the *Annual Report of the Chief Medical Officer* of DHSS (NI).

Few fields have benefitted more from reports of public officers than has public health: 'The establishment of the office of Registrar-General in 1837 marks a major turning point in the history of public health as well as of demography in Britain'.[3] It is no accident that Britain's most influential Victorian public health pioneers were public servants — William Farr ('compiler of abstracts' at the GRO), John Simon (medical officer to the Local Government Board and the Privy Council), and Edwin Chadwick (secretary to the Poor Law Commission). To emulate them their heirs must improve the present knowledge-base on the relationship between, on the one hand, the provision of health and other services and, on the other, population or individual health, and instigate those recommendations in the recent Green Paper[4] concerning monitoring, assessing and evaluating indicators of 'health gain' rather than of 'process' or 'provision'. This will require a conceptual grasp of basics, sound views on health and sickness determinants, intellectual courage, awareness of political imperatives, and a consistent strategy over the years.

Dr Scally understands all this. His first three reports are planned for this wider purpose as well as promulgating essential statistics on current issues. Thus his first report[5] provides *inter alia* a baseline for the measurement of future experience. His second[6] extends the database and focuses further on high profile disease rubrics — cardio-vascular disease, cancer, mental illness, maternal and child health, and accidents. While the present report further develops these themes, it also introduces the concept of 'health gain' now placed firmly in the new environment of the restructured health services, and courageously faces contemporary problems notably those of health and homelessness, and of unplanned pregnancy in teenagers, which our society (and its medical commentators!) conveniently though supinely choose to ignore. The report itself is 'reader-friendly' with generous type, clear and incisive prose, well-chosen tables and diagrams, and with core summary tables compiled in concert with those of the other three Area Boards so that "the rates given are directly comparable between Boards" — a laudable exercise indeed! There is a selective bibliography.

The value of this series will depend on the ability of the four Directors not to be swayed by shorter-term imperatives from their longer-term objectives; not to lose the central thrust. It would be unfortunate if failure of vision, resources, or resolve, turned the exercise into a bland commentary on annual compilations of easily retrieved statistics. Close collaboration will identify and prioritise matters of general concern; intelligence and resolve, such as Dr Scally and his colleagues show, will place these squarely on-stage and preserve a common strategy. This reviewer would not object to more challenge or controversy in subsequent reports: the contemporary Director may hardly match the classical allusions or ornate prose of a Farr or a Simon, let alone a Greenwood, (or have such uncharted territory to map), or rival the bellicosity of a Chadwick, but he could aim to match their sense of inquiry and zeal for discovery. Dr Scally and his group have made an excellent start.

P FROGGATT

REFERENCES

1. *Health of the Population: Responsibilities of Boards.* DHSS (NI), Circular (CHI/89) (1989).
2. *Public Health in England (Acheson Report).* London: HMSO, 1988.
3. Flinn MW (ed). Introduction to Chadwick, E (1842), *Report on the Sanitary Conditions of the Labouring Population of Great Britain.* Edinburgh: Livingstone, 1965, p.31.
4. *The Nation's Health: A Consultative Document for Health in England.* Cmnd. 1523. London: HMSO, 1991.
5. *1988 Public Health Matters. The First Annual Report of the Director of Public Health.* Eastern Health and Social Services Board (NI), 1989.
6. *1989 Public Health Matters. The Second Annual Report of the Director of Public Health.* Eastern Health and Social Services Board (NI), 1990.

Altnagelvin's thirty glorious years: Two hundred years of medical care in Londonderry. By Cahal Dallat. (pp 122. £3.00). Available from Altnagelvin Area Hospital, Londonderry, N. Ireland BT47 1SB.

This publication records the medical history of Londonderry from the creation of the Abbey of St Columba in 546 AD, with a brief reference to the first hospice founded by Princess Macha in 330 BC.

In the closing stages of World War II, a combined effort was made by municipal leaders to build a new Londonderry hospital under the able chairmanship of the late Anderson Piggot, OBE. The endeavour was to replace the outdated institutions, that is, the infirmary type of hospital and the lunatic asylum; the latter dating from 1829. The London firm of architects in hospital construction drew up plans to meet the requirements of modern medicine, surgery, dentistry, nursing and support services. This consisted of an eleven storey building on a high ground site two miles from the city centre to be named Altnagelvin.

A main article in the press in early 1959 recorded: "Is this Europe's finest hospital?". "Bedside phones, television, balconies with a view . . . and meals come by electric train" ran the sub-heading. "Stretching spectacularly and proudly above the Ulster countryside like a luxury Mediterranean hotel, Britain's first major post-war hospital waits to receive its first patient . . . It is a landmark both in architecture and modern surgery. For into this building has gone 14 years of planning . . . and every new idea to increase efficiency in a cause where efficiency means a disease beaten, a life saved."

It was to be the first completely new acute hospital to be built in Europe since the end of World War II. The project was ambitious. Economy enforced modifications in design. However, events progressed, and in July, 1960, Lord Wakehurst, Governor of Northern Ireland, unveiled the plaque at the opening of the hospital. This embraced 391 beds, the main medical and dental specialties, doctor instruction, nurse, radiographic and physiotherapy training and full support services.

The Area Hospital became noted throughout the UK, and further afield; being recognised by the Royal Medical Colleges, the Nursing Council and Universities for training. In 1967 the BMA decided to hold the National Annual Conference in the Altnagelvin site and related areas. This was a massive undertaking with visiting delegates from the main areas of the UK, the Americas, Europe, India, Pakistan and Japan.

In the mid 1980's two ophthalmic wards were re-equipped; a modern newly designed accident and emergency department with a major resuscitation area being provided, with two operating theatres, specialised examination rooms and allied facilities. In 1988 a new nucleus was built consisting of an ophthalmic ward and theatre facilities.

The final chapter of this record concludes with a message from the administrative staff. "The spirit that pervades Altnagelvin Hospital today is a product of the inspired and charismatic leadership which has existed from its beginning. The emphasis is on teamwork, recognising the uniqueness of the contributions to be made by the different professions and trades in a holistic approach to quality care. In the medical and nursing professions especially, we have had a succession of people of vision and stature co-ordinated by administrators of outstanding sensitivity and ability — all of them held in high esteem by their peers throughout the Province and further afield."

DG WHYTE