VOLUME 40 1971 PART 2

THE ULSTER MEDICAL JOURNAL

PUBLISHED BY
THE ULSTER MEDICAL SOCIETY

The Ulster Medical Journal

VOLUME 40

PUBLISHED BY
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1971

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THE ULSTER MEDICAL JOURNAL

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To DR. N. C. NEVIN,
DEPARTMENT OF MEDICAL STATISTICS,
INSTITUTE OF CLINICAL SCIENCE,
GROSVENOR ROAD, BELFAST BT12 6BJ.

.....19.....

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THE
ULSTER MEDICAL JOURNAL

PUBLISHED ON BEHALF OF THE ULSTER MEDICAL SOCIETY

VOLUME 40 1971 PART II

THE HISTORY OF THE ROYAL BELFAST HOSPITAL FOR SICK CHILDREN THE QUEEN STREET DAYS

By H. G. CALWELL, B.A., M.D., D.T.M.&H.

THE SIR THOMAS AND LADY EDITH DIXON MEMORIAL LECTURE

delivered before the Ulster Medical Society, 14th January, 1971

I MUST first acknowledge the great honour that has been done me by the invitation to deliver the Sir Thomas and Lady Edith Dixon Memorial Lecture. When I accepted the invitation I requested that I should be permitted to deliver the lecture under the auspices of the Ulster Medical Society so that I might continue the story of the Royal Belfast Hospital for Sick Children at the domestic hearth of Ulster medicine. I am grateful to the president and council for granting my request.

The history of the hospital is a proper subject for a Dixon lecture as both Sir Thomas Dixon and his wife were generous supporters of its work, giving not only money and goods but also of their time.

Brice Smyth, the hospital's first attending physician, testified in public in 1910 to Sir Thomas Dixon's active interest as a member of the Board of Management. Lady Dixon founded the Linen League in order to supply household linen for the hospital and on more than one occasion provided the New Year dinner and tea. She was also a member of the Board. Much of the story I have to tell is of events that were shaped by the Dixons and their contemporaries.

In the year 1879 Miss Lennox, the first matron of the (Royal) Belfast Hospital for Sick Children, received a letter from Miss Florence Nightingale in whose school of nursing at St. Thomas' Hospital she had once been a pupil. This letter reads:

April 21/79.
10 South Street,
Pak Lane. W.

My dear Miss Lennox

Many, many thanks for your letter.

I take the liveliest interest in your new Children's Hospital and I give you joy & your little patients & Belfast joy of their extension of a good work.

I am going to ask you a favour: could I have *tracings* of the plans. . . . In Germany they are going to build a new Children's Hospital at Heidelberg (Baden); & the Grand Duchess of Baden, who has done a great deal in Training Nurses, has written to me for plans of the best new Children's Hospitals in this country. I am collecting and having tracings of plans made to send to her. And I should be very grateful to add to them the new Belfast Children's Hospital.

Thanking you again for your letter, & again giving you joy, pray believe me, dear Miss Lennox, ever sincerely yours

FLORENCE NIGHTINGALE

The occasion of this letter was the opening of the new Belfast Hospital for Sick children in Queen Street.

In a postscript to the letter Miss Nightingale thanked Miss Lennox for a newspaper cutting which she said made her all the more eager to see the plans. This may have been from the Belfast Newsletter of 19 April 1879 in which there is a long report of the transfer of the hospital from King St. to the new building in Queen St. the previous day.

The reporter began by praising the work that had been done in the old hospital and then went on to give a detailed description of the new:

> The façade . . . is an elegant specimen of the English style of architecture of the reign of Queen Anne. . . . The entrance for patients is through an area in College Court. Immediately on entering from Queen Street is a spacious hall, having on the right a room for visitors and on the left the boardroom. In the design of the building light, quietness and ventilation have been attained in a remarkable degree. On the ground floor the kitchen, laundry and general consultation room are placed, the last having the doctor's room adjoining. Opening off the last named is the dispensary from which the patients will receive the medicine at a window. On the first floor apartments for the matron are situated over the boardroom. . . . The general medical ward having accommodation for upwards of thirty beds and an apartment for the nurses in attendance are behind this. A small ward with two beds opens off the general ward. On the second floor the arrangements are somewhat similar, the most noticeable feature being that a room has been fitted up as a theatre for operations. Between the theatre and the general surgical ward is a small room in which patients will be kept after operation. . . . There are five rooms in the attic and it is probable that they will be used for patients with infectious disease. Communication is obtained between the different parts of the house by means of speaking tubes, and a lift or hoist will convey the food to the different floors.

Brett (1967) described the Queen St. Hospital as "a distinguished building in Scottish Renaissance type . . . much more successful than the Belfast Town Hall (also designed by Thomas Jackson & Son).

The doors of the old hospital in King St. had hardly been opened when the Board began seeking more suitable accommodation. The search ended in 1876 when the Belfast Town Council offered for disposal by public tender a vacant plot of ground on the west side of Queen Street between the Gas Office and a dwelling house. The Board's tender of a yearly rent of £65 with a single payment of £50 down was accepted in April 1876, although, as the Town Clerk pointed out, another offer £5 higher had been received. The Board entered into an agreement to expend £500 on the premises within 3 years.

A letter was sent to subscribers seeking their aid, and the interest of the public at large was solicited by the erection of a board on the site intimating that the new Belfast Hospital for Sick Children was to be built there.

The *Witness* (a now defunct weekly journal which had a wide circulation and exerted much influence in the presbyterian community) gave the appeal powerful support in an editorial article. The editor began by castigating those who objected to special hospitals and who advocated that all the various agencies for the relief of suffering in Belfast should be under one central management. He went on:

> The Children's Hospital has seized hold of capacities for good, that are both powerful and widely diffused. It is now three years old, the promoters want a larger building. By all means let them have it and let the public of Belfast give generously. . . . To erect an adequate Children's Hospital in Belfast will, after all, only demand an imperceptible tax on our luxurious expenditure.

By the end of 1876 more than £2,000 had been contributed to the building fund, and the Board felt they could proceed. Their first step was to appoint an architect. They chose the firm of Thomas Jackson & Son, Corn Market, Belfast, a member of which had superintended all the building and sanitary arrangements in the King St. hospital.

This firm was well known in Belfast throughout most of the 19th century having designed among other buildings the Old Museum in College Sq. North, St. Malachy's Roman Catholic church in Alfred St., the Belfast Town Hall in Victoria St., St. Enoch's Presbyterian church in Carlisle Circus (Brett, 1967) and Glenmachan Tower, Strandtown, where Thomas Jackson lived. The architect visited the Evelina and other children's hospitals in London, and Miss Lennox visited the Pendlebury Children's Hospital (now the Royal Manchester Children's Hospital) in order to obtain ideas and suggestions for planning the new hospital.

Hospitals have not always been safe places for patients – indeed they have at times been positively dangerous. The Board recognized this and expressed their awareness of "the grave responsibility which is incurred by those who undertake to bring together in a hospital a number of persons, whether children or adults, without adopting every possible precaution for their safety, and thus reducing the risk of their being associated together."

They also recalled the words of Florence Nightingale in the preface to her book "Facts about Hospitals":

> It may seem a strange principle to enunciate as the very first requirement in an Hospital, that it should do the sick no harm."

Their aim was "to have a building especially built for its purpose and with all the arrangements carried out on the best principles." They followed the axiom *primum non nocere.*

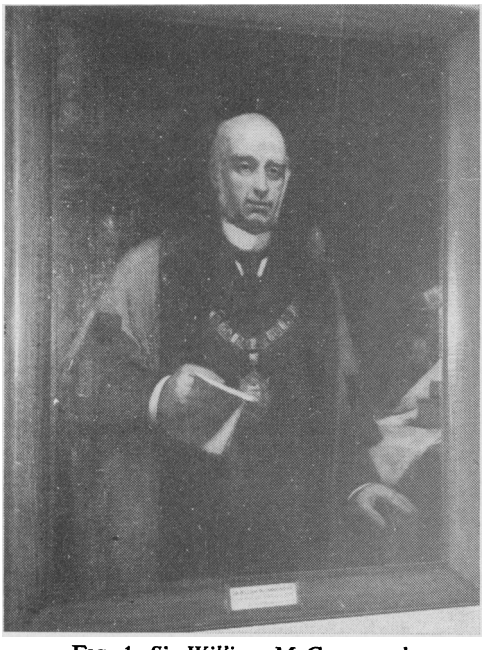

FIG. 1. *Sir William McCammond*

The plan finally adopted was for a complete hospital of 44 beds. The building contract was awarded to Messrs. William McCammond of which firm the head later became Mayor of Belfast and Sir William (Fig 1). The price was £4,012 and the date set for completion was 1st February, 1879.

There were many interruptions in the work – some due to natural causes such as severe weather and the softness of the ground and a prolonged one due to the encroachment on the site of the upper part of the Gas Office. This was only resolved after threat of legal action against the Town Council. Nevertheless all trials and tribulations were overcome, and in February 1879 the members of the Board and the Ladies' Committee were busy in the new hospital seeing to its furnishings.

When the time came for the lettering to be cut on the front of the building the architect was in doubt about what date should be inscribed. A drawing shows A.D. 1877 (Fig. 2) but the actual date on the building (which is still there) is 1878. Near the apex of the gable of the middle dormer the heraldic arms of Belfast were inscribed.

Little information remains about the actual furnishing and equipping of the hospital. Early in 1879 approval was given to John Fagan, the senior surgeon, to buy two sets of surgical instruments at a total cost of £5/10s, and a little later he was permitted to purchase additional instruments costing £9/3s/10d.

Inquiries were made in children's hospitals in London and elsewhere about cots. There was the choice of the "Ormond St." pattern at 30 shillings or a stronger one at 35 shillings or one with brass knobs at 42 shillings. The Board chose the last. Illuminated cards bearing the names of the donors of cots or of the persons they desired to commemorate were to be prepared for attachment to the cots. I have not found any of these cards although many brass plates of similar intent but of a later date remain.

It was decided to open with only 18 beds – the number in King St. Some members of the Board wanted a ceremonial opening which "might add prestige to the hospital and be a source of profit." The majority of the members were opposed to this, and the opening of the new hospital was announced by advertisement in the newspapers and a circular letter to clergymen.

Pride of achievement is expressed in Darbishire's speech at the annual meeting in 1880. To quote from the report:

> He said they had left their old, small inconvenient hospital in King Street, where they did so large a work, and did it so thoroughly, and had removed into an Hospital which would rank with any of the important hospitals of this kingdom. It had every appliance that they could bring into requisition for the alleviation of the suffering of the sick children of the poor.

Having launched the new hospital successfully Darbishire resigned from the office of honorary secretary and was succeeded by Edward Higgins. In commemoration of Darbishire's services it was resolved that the surgical ward should be given his name and also that the medical ward should be given the name of the president Lord O'Neill. The names were "not to be put up in any merely ornamental fashion but in such a solid manner as to become part and parcel of the building." They were engraved in marble tablets placed above the doors of the respective wards but in spite of the Board's resolution in 1931 that all the Queen St. names should be repeated in the new Falls Rd. hospital other and later names have replaced those of Darbishire and O'Neill.

The hospital was now serving a wide area of the province but the public were not making full use of it. The medical staff noted that there was much greater readiness to place in hospital a child with deformity than one with, for example, pneumonia. They asked the ladies who visited the sick poor to try to educate them in the importance of in-patient treatment in acute medical conditions.

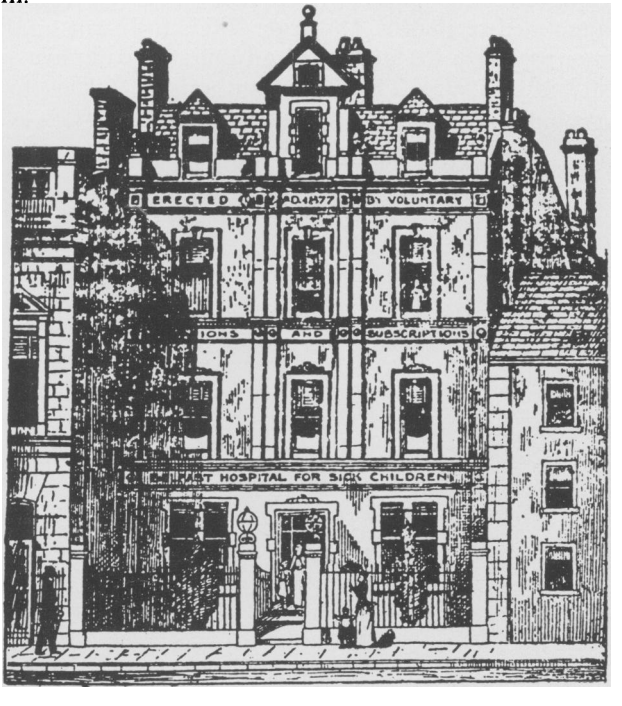

FIG. 2. *The Belfast Hospital for Sick Children, Queen St.*

The term "sick poor" is familiar. We have already heard it in Darbishire's farewell speech, and it recurs time and again. The first object of the hospital was to help the sick children of the poor, not sick children in general. Children other than those of the poor were not admitted unless the admission of poor children had to be restricted because of lack of money.

Here is how a writer in the Children's Sunday Album saw a children's hospital in 1881:

> Look at this picture well, you bright happy children who are well and

strong, or even any afflicted like these, and be grateful for the cheerful home, the loving friends, the comforts which surround you! Good generous people pitying and loving little children, have sent enough money to support them. . . . Bad nursing in their babyhood, joyless unchildlike lives in crowded dirty streets, cause the children of the London poor to be wretched sufferers; and it is a piteous touching sight to visit the hospitals which have been built for these poor little creatures. Everything is done for them that skill and kindness can do; but it is not like you at home in your beautiful nurseries . . [with] your loving mother, and healthy little brothers and sisters making merry round. In each little bed is some poor, suffering child, tended by kind nurses certainly, but no mothers. Think of this, little ones, when inclined to be fractious and cross and bless God who has made your lot so bright.

The first break in the original attending medical staff came in 1883 when Brice Smyth resigned and was elected a consulting physician. The new honorary attending physician was John McCaw (Fig. 3), who is regarded as the first specialist in diseases of children in Belfast. The first edition of his book "Aids to Diseases of Children" was published in 1893 (Fig. 4). The hospital possesses his personal annotated copy of it. He wrote "Aids to Infant Feeding and Hygiene" in 1905, and was probably the author of an undated pamphlet on the care of infants which is still in existence. The Board authorised McCaw to have 10,000 copies of such a

FIG. 3. *John McCaw*

AIDS

TO

THE DIAGNOSIS AND TREATMENT

OF

DISEASES OF CHILDREN

(MEDICAL).

BY

JOHN McCAW,

M.D., L.R.C.P., ETC.,

PHYSICIAN TO THE BELFAST HOSPITAL FOR SICK CHILDREN;
VICE-PRESIDENT OF THE ULSTER MEDICAL SOCIETY.

STUDENTS' AIDS SERIES

LONDON: BAILLIÈRE, TINDALL & COX,
King William Street, Strand.
EDINBURGH: LIVINGSTONE. DUBLIN: FANNIN & CO.,
GLASGOW: SMITH & SON.

FIG. 4

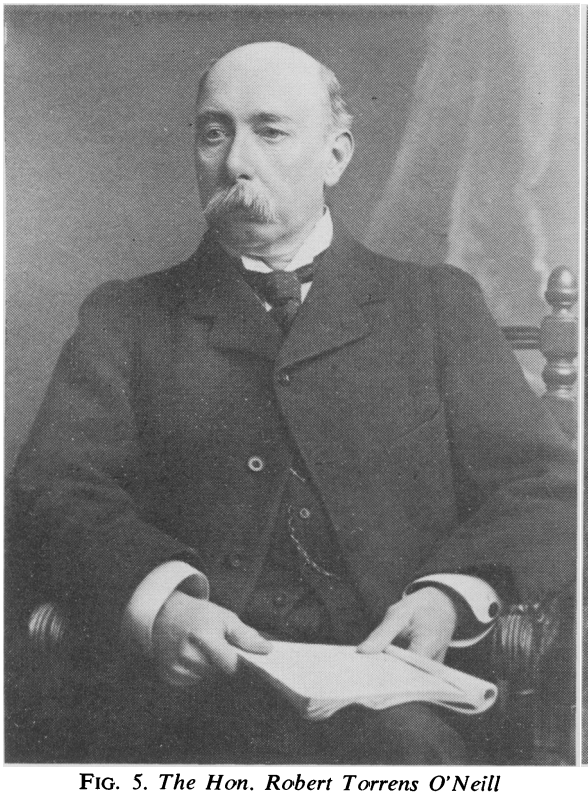

FIG. 5. *The Hon. Robert Torrens O'Neill*

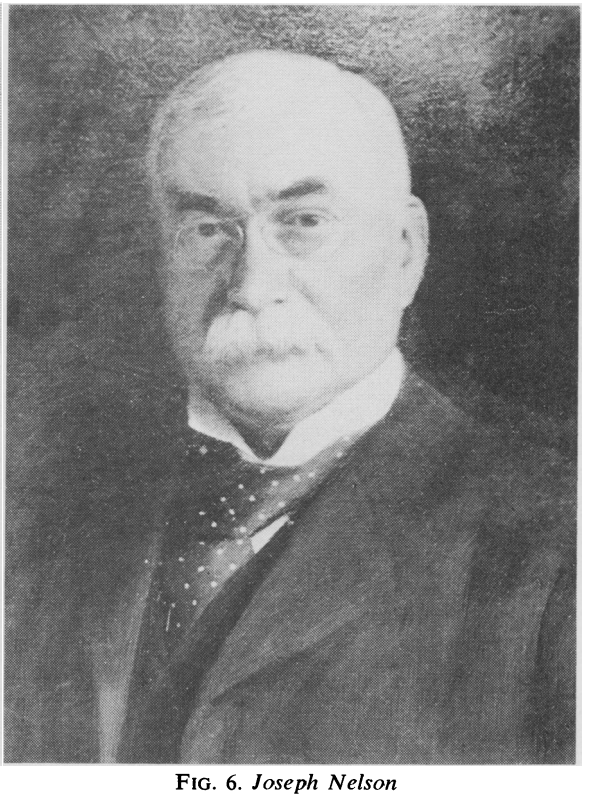

FIG. 6. *Joseph Nelson*

pamphlet printed in 1899 but whether that was the one of which I possess a copy I do not know.

The Reverend William, Baron O'Neill the hospital's first president, died in 1883. There is ample documentary evidence of his keen interest in the hospital and of the wise counsel he gave. He was succeeded in the presidency by his third son, the Hon. Robert Torrens O'Neill (Fig. 5) who was to hold the office for the next 27 years.

The claims of specialism gradually made themselves felt, and in 1885 the Board established two new posts – that of oculist and aurist combined and of dental surgeon. Those appointed were Joseph Nelson (Fig. 6) and John J. Andrew respectively. Nelson's letter of acceptance has survived. Much has already been written and spoken of his career.

There had been a move to appoint a dental surgeon as early as 1875 but nothing came of it. In 1883 an unnamed dental surgeon had offered to treat free any children in connection with the hospital but the Board had declined his offer stating that there was no necessity for such a service. The views of the medical staff on this cannot be known now as there are no records of their meetings before 1885. Andrew had received his dental training at the London Dental and the Middlesex Hospitals. He was President of the British Dental Association in 1908.

When Nelson joined the staff they recommended that he be given a special ward of four beds for his patients but the Board would not agree and instead provided an isolation ward for children with infectious disease awaiting transfer to the Union Fever Hospital.

In 1888 the Board increased the medical staff by appointing an additional assistant physician and an assistant surgeon "to take up the work in the enforced absence of other members of the staff." This is the first use of the designation "assistant". Francis Howard Sinclair (Fig. 7) and John St. Clair Boyd were elected.

The duties of the new posts were defined:

> To take charge of such extern patients as the Board shall appoint.
>
> To assist the physicians and surgeons in the wards and act for them.
>
> To be summoned to all staff meetings, consultations and operations (at which they shall assist and give the anaesthetics).

FIG. 7. *Francis Howard Sinclair*

Sinclair served until 1899. He is best known for his interest in pulmonary tuberculosis. He was a physician in the Consumption Hospital in Fisherwick Place (the predecessor of the present Forster Green Hospital), and his contributions to medical literature include a paper on the treatment of tuberculosis with tuberculin. Sinclair was a distinguished offshore yachtsman; for three years he held the London Cruising Clubs Challenge Cup for the best amateur yachting cruise in the British Isles (Dewar, 1900).

Boyd is better known as a gynaecologist than a children's surgeon. He joined the staff of the Ulster Hospital for Children and Women in the former capacity in 1889, remaining also on the staff of the Children's Hospital until 1891. A sidelight on him appears in a book by Bulmer Hobson (1968).

> At this time [1901] I had been a member of the Gaelic League in Belfast. Dr. St. Clair Boyd was the chairman and Treasurer . . . and being well off, he paid all our bills.

Boyd's grave is marked by a Celtic cross inscribed with his name, degrees and dates of birth and death in old Irish lettering. This is further evidence of his interest in Gaelic culture.

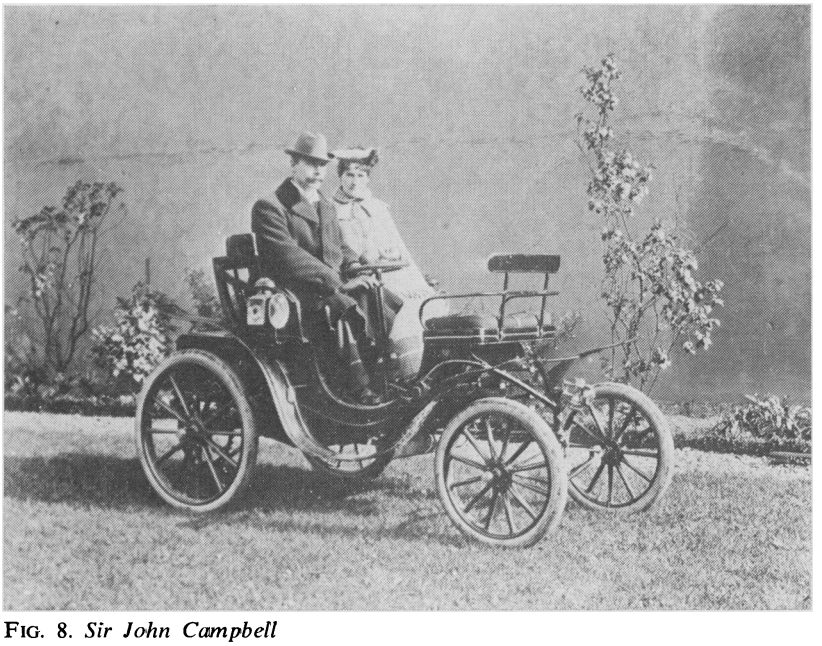

FIG. 8. *Sir John Campbell*

When Boyd resigned in 1891 his successor as assistant surgeon was John Campbell (Fig. 8) (later Sir John). He left at the end of 1892 to pursue a successful career as a gynaecologist in the Samaritan Hospital, Belfast.

Miss Lennox was compelled by ill health to resign in 1891 having been matron for 18 years. There was great difficulty in replacing her. There were three matrons between 1891 and 1894 when at last one was appointed who stayed for more than a few months. She was Miss Beatrice Colborne.

The rapid turnover of matrons suggests that perhaps their relationship with the Ladies' Committee was not always cordial. When Miss Winder resigned in 1893 after only 13 months' service her resignation was accepted without any recorded request to her to change her mind, and it may be only coincidence that it was exactly at that time that the ladies made a new rule "that the matron shall wear indoor uniform consisting of Netley Cap, plain blue serge dress, apron, etc. Perhaps Miss Winder did not agree.

Miss Lennox had probably made her own rules of conduct and duty as she went along but after her departure the Ladies' Committee laid down rules:

> The Matron shall not absent herself . . . for a night without intimation to the Lady President. She shall have at least one free day each month . . . and an annual holiday for one month.
>
> She shall be responsible for the good order and government of the household affairs.
>
> She shall visit every part of the hospital once every day. . . . She shall be responsible for the welfare of the patients. . . . She must be present at all operations.

To these duties were added the charge of all instruments and stocks, the engagement of nurses and servants (subject to the approval of the Ladies' Committee), the keeping of household accounts and the oversight of the Convalescent Home.

There was one commodity of stock which caused trouble in the period when matrons were coming and going with alarming frequency. A minute of the Ladies' Committee reads:

> The ladies would suggest an amendment in the arrangement about stimulant *viz* that it should be kept in the Dispensary and not in the matron's room.

A minute of the Board follows:

> Lady President remarks that none are kept in the Hospital but have to be sent out for when required, yet that an unusual consumption is going on. The Board of Management recommend that the matron be authorised to keep a bottle of brandy in her own room under lock and key for use in urgent cases, that she keep a book and shew dates when and quantities used for the information of the Ladies' Committee who will order more when required.

Another minute of the Board follows:

> The Ladies' suggestion approved of that this shall be kept in the Dispensary and not in the Matron's room, it being understood that a small quantity is always in the operating room.

On Fagan's appointment to the consulting staff in 1893 Edward Churchill Stack succeeded him as honorary attending surgeon, and Sidney Brice Smyth (Fig. 9), a son of Brice Smyth, was elected assistant surgeon. The unsuccessful candidate was Arthur Brownlow Mitchell. Smyth did not remain long on the surgical side for in the same year Byers resigned, Sinclair was promoted attending physician in his place and Smyth (with the agreement of his colleagues on the staff) became assistant physician.

Smyth's transfer left a vacancy for an assistant surgeon. The successful candidate was John Smith Morrow (Fig. 10), and the unsuccessful candidate was again Mitchell whose talents were soon to be put to the service of the Ulster Hospital.

FIG. 9. *Sidney Brice Smyth*

FIG. 10. *John Smith Morrow*

FIG. 11. *Thomas Sinclair Kirk*

FIG. 12

The public demand on the hospital was now so great that the medical staff recommended the creation of additional posts of assistant physician and assistant surgeon. The object was to have the extern fully staffed every day. The Board approved and in 1895 elected William C. Steen assistant physician and Thomas Sinclair Kirk (Fig. 11) assistant surgeon. Kirk was to serve the hospital for forty-one years. He is commemorated by a mural tablet (Fig. 12). The unsuccessful candidate for the surgical post was Andrew Fullerton.

The reorganization of the out-patients' department included the appointment of the first extern sister, at a salary of £23 per year. Not only did she carry out the ordinary duties of such a post but it is recorded that "some of the children also return in the afternoon when Sister Janet puts them through a course of physical exercise and massage." The physiotherapy department of the hospital has a respectable antiquity.

The accommodation was also enlarged so that it became possible to perform many minor surgical operations in the extern instead of as hitherto only after the admission of the child to hospital.

The first honorary pathologist was appointed in 1896. He was James Lorrain Smith (Fig. 13) who later became the first Musgrave professor of pathology in the Queen's College, Belfast, and was then lecturer. The circumstances of his appointment to the hospital were unusual. At a meeting of the medical staff in 1896 the members discussed the question of adding a pathologist to the staff. McCaw suggested that such an appointment should be in the hands of the staff and that they should defray all expenses connected with it, the post being an honorary one. This was agreed to, and Kirk was deputed to call on Smith and convey the staff's invitation to him. Smith refused the invitation at the hands of the staff but sent word through Kirk that if the Board created the post of honorary pathologist to the hospital with full staff status he would become a candidate for it. The Board in due course created the post and appointed Smith.

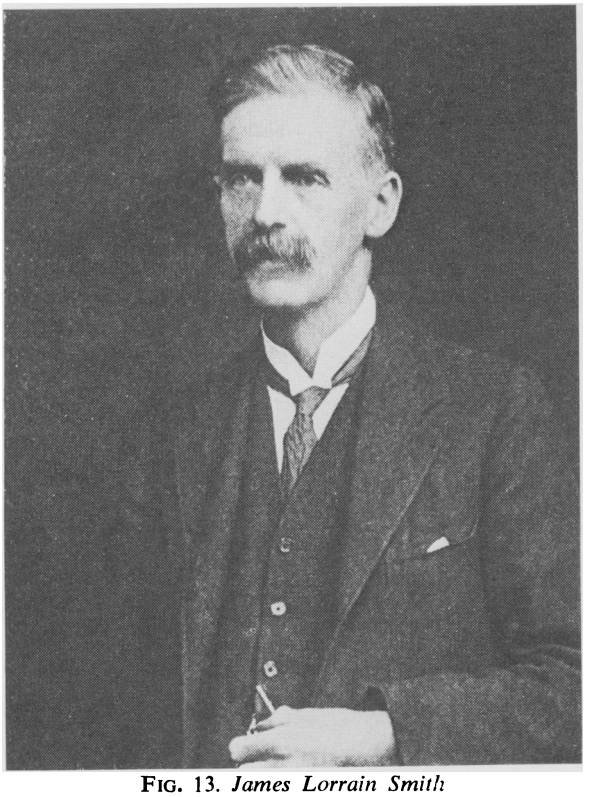

FIG. 13. *James Lorrain Smith*

Funds were now falling so far behind needs that the Board decided that free medicines would not be supplied to out-patients after 1st July 1897. It was estimated that this measure would save £100 annually. Instead patients were

to be given prescriptions which they could have dispensed at reduced rates in certain pharmacies. A list of these was displayed in the extern. An alternative measure proposed was that each patient should be charged one shilling for consultation and medicine. The medical staff rejected this and chose the other, although with misgivings.

The immediate result was that the work of the medical extern almost stopped. There had been 7,121 new medical out-patients in 1896; there were 2,538 in 1898. This catastrophic decline drew public comment from the Hon. Robert O'Neill who deplored the decision to withhold free medicine because "the decline in numbers represents thousands of children whose poverty obliges them to abstain from the benefits of the hospital." He was to repeat this stricture on other occasions.

It was one of the original rules of the hospital that no child under the age of 3 years should be admitted as an in-patient. Discretion was allowed to the medical staff in the matter but the Board sometimes enforced the rule. An example occurred in 1891. Byers as honorary secretary of the medical staff brought to the Board a request from one of the surgeons that a child under 3 might be admitted for operation, accompanied by the mother. The Board decided not to sanction any alteration of the rules.

In 1896 the ladies drew attention to the admission of so many infants but added "the infant mortality in Belfast is so great that a little latitude . . . seems to meet a badly-felt want. We have had babies in as young as three months. . . ."

The infantile mortality rate in Belfast at the time was 147 per 1,000 live births (compared with 24 in 1969). That is to say 147 infants died within a year of their birth. The medical officer of health commented (Medical Officer of Health, 1894):

> The law which precludes women from working in the mills for one month after child bearing is practically a dead letter. Millworkers, the majority women and children, go to work at 5.30 a.m. indifferently clad, with but little nutritious food and working at high pressure. What chance have their babies?

Morrow tried to regularize the admission of infants by proposing that the rule be changed. He wanted infants to be admitted "in especially urgent cases, children with hare lip to be nursed if possible by the mother during the stay in hospital" The Board stuck to the criterion "extreme urgency", but later relented and allowed the staff to use discretion subject to reporting to them the circumstances of every under-age admission. Finally when the rules were being redrawn on the hospital being incorporated in 1908 the lower age limit disappeared and there only remained an upper age limit of 12 years.

On Stack's resignation in 1897 Morrow became full surgeon, and Robert Campbell (Fig. 14), younger brother of John, was elected an assistant surgeon but within a few months there were more changes when Mackenzie left the active staff to joint the consulting staff. Kirk was promoted full surgeon and succeeded as assistant surgeon by James Lynass. The unsuccessful candidate was again Andrew Fullerton (Fig. 15), this time an aspiring surgeon. He did not have to wait long however for another opportunity, for in 1898 Morrow departed from the world of surgery to pursue a career in medicine, Campbell was promoted in his stead and Fullerton was elected an assistant surgeon.

FIG. 14. *Robert Campbell*

FIG. 15. *Andrew Fullerton*

FIG. 16.
Miss A. I. McTaggart

A new matron was being sought again in 1898 when Miss Colborne left to become matron of a hospital in Bristol. She had won golden opinions from the medical staff during her 4 years' service in Belfast and in 1896 had had the honour of receiving together at the hospital Lord Kelvin and Lord Lister. There was some talk of Kelvin giving a lecture in connection with the hospital (probably to raise money) but I have not found any record of such an event.

The new matron was Miss Amy Isobel McTaggart (Fig. 16) who had been trained in the Westminster Hospital and came now from the Royal Edinburgh Hospital for Sick Children where she was surgical sister and sister in charge of the operating theatre.

The century was drawing to its close, and the last change in the medical staff to be recorded in the 1800s is the promotion of Sidney Brice Smyth to succeed Sinclair as attending physician in 1899. The new assistant physician was Robert R. L. Leathem (Fig. 17). The first election in the 1900s was that of Brian O'Brien to be assistant surgeon in place of James Lynass who had resigned.

The Board marked the birth of the twentieth century by a daring innovation. They invited the Ladies' Committee to nominate five lady life governors to be associated with them and attend their monthly meetings. Hitherto communication between the Ladies' Committee and the gentlemen of the Board had been by letter. The ladies were however not yet actually members of the Board. Full membership was not accorded them until 1904.

In 1901 Steen resigned from the staff and was succeeded as assistant physician by W. M. Beatty who wrote the chapter on diseases of the blood in the third edition of McCaw's Aids to Diseases of Children which was published in 1907.

Between 1880 (the first complete year in Queen St.) and 1900 the annual total of in-patients had risen from 294 to 572 but that of out-patients had fallen to 5,235 from its peak of 9,212 in 1896. The decision to withhold free medicine was still having its effect on attendances of medical out-patients.

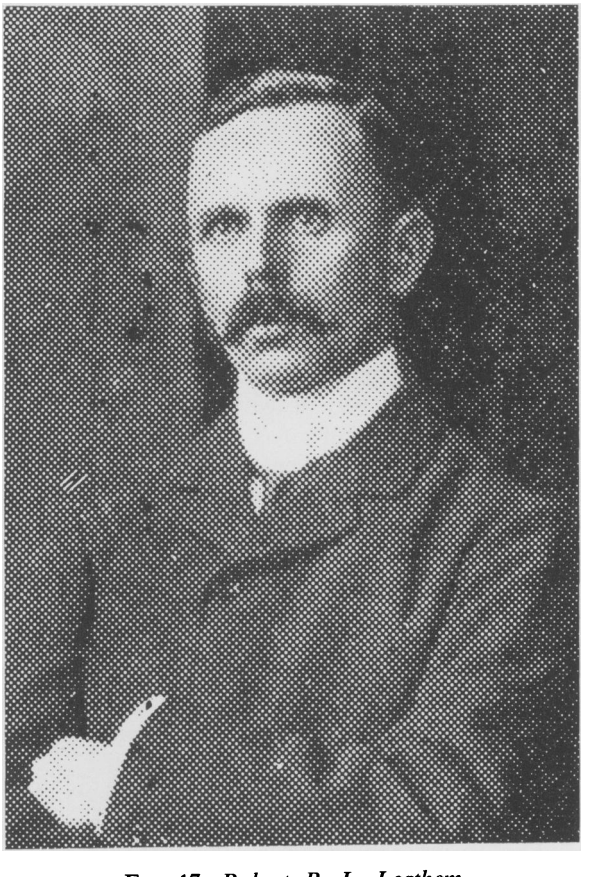

FIG. 17. *Robert R. L. Leathem*

There was an annual deficit of £200 which was a continual worry to the Board, and things became worse after the outbreak of the South African war. In 1900 the cost of drugs and dressings rose to double the previous cost but the rise in food prices was less marked. The daily cost per in-patient rose from 7d in 1899 to 8d in 1900.

Advances in the surgery of childhood led to increased admissions to the surgical ward. The annual number rose from 109 in 1880 to 248 in 1900. The "acute abdomen" was beginning to feature in the reports. A child was admitted with intussusception in 1898 and another in 1899 (this noted as "acute"). The first mention of appendicitis was in 1902; I have not even found the older term "typhlitis' in earlier reports. It may be that some children with acute abdominal conditions were still being admitted to the Royal Hospital.

The development of the practice of out-patient surgery in the Children's Hospital owes much if not all to Robert Campbell. It accorded well with his dictum "the best nurse for a child is its mother and the best place for it to be ill is at home." (Higgins, 1955).

At the annual meeting of the British Medical Association in Belfast in 1909 Campbell stated that he was in the habit of operating in the out-patient department on children with hernia. Fullerton followed with a note of warning about the possible legal consequences if there were post-operative complications and an outside doctor told the parents that the children should not have been sent home from the hospital immediately after operation but he ended by telling the meeting that his own practice largely agreed with Campbell's and he was sure more work would be done in the out-patient theatre.

Fullerton's prediction was fulfilled; he saw to it that it was. The result was such an increase in out-patient surgery that the Board was obliged to provide a new operating theatre in the out-patient department. The total cost of this was £260 which had to be taken from capital. The new theatre came into use in 1912. It was described as "absolutely the last word in up-to-date surgical operating rooms and replete in every particular". The number of operations on out-patients that year was 702, "every one of which was successful", the Board reported, "every one of these had been attended with success in the complete restoration to health of the patient," the President announced at the annual meeting. The report of the medical staff merely recorded the number of operations.

However successful the work may have been, one member of the Board expressed grave doubt about the wisdom of carrying out "severe" operations and discharging the patients immediately. The question was referred to the medical staff for their opinion which was that no untoward circumstances had arisen in the past . . . and that the same vigilance would continue in the future.

Fullerton appeared before the Board with McCaw at its next meeting and stated that in the opinion of the medical staff the practice was safe. He continued:

> By operating on children as out-patients the hospital is left free to do a larger work without extra cost. In the case of young children it is an advantage to have them nursed by their mothers. A majority of the operations are for hernia and in my own series the cure rate is 95 per cent.

The Board thereupon resolved that there should be no curtailment of the surgeons' "magnificient" work in the out-patient department.

A post-script to these events is a letter Fullerton wrote in 1913 in which he stated that he had greatly extended the scope of his out-patient operations since 1909. He now included hare-lip, cleft palate, genu valgum and varum, tonsils, naevi, tuberculous joints of the upper extremity, enlarged glands and cysts in the neck, and he had lately operated successfully on an occipital meningocele. He ended: "The surgeon in charge of out-patints, instead of being a glorified finger-post to the wards has now opportunities for practice and research denied him."

In telling how Fullerton escaped from the tedious and passive role of "glorified finger-post' I have departed from chronological sequence and must go back some years. In 1902 the hospital was extended by the provision of two additional wards – a medical and a surgical, each with six beds. One was named "Dudley" in honour of the Earl of Dudley who was Lord-Lieutenant of Ireland and performed the opening ceremony in the course of a viceregal progress; the other was named "Craig" in honour of Mr. Vincent Craig, an architect and brother of Viscount Craigavon the first Prime Minister of Northern Ireland. Mr. Craig had contributed one hundred pounds of the total cost of £225. Alas funds were so low that it was not possible to use the wards for patients, and they were used as playrooms. To have admitted patients to them would have meant additional expenditure of £150 per year which was a burden too great to be borne.

The increase in the hospital's surgical practice led to the appointment of the first anaesthetist. This appointment was solely in the hands of the staff as the title of the post "honorary anaesthetist to the staff" shows. The first to be appointed was Isaac Davidson who soon turned to ophthalmology in the Benn Ulster Eye and Ear Hospital. The unsuccessful candidate was a woman (Dr. Sproull) who must have been one of the earliest of her sex to seek a hospital appointment in Belfast.

The perilous state of the finances caused the Board to inquire in 1903 into the comparative costs of some of the principal children's hospitals in the United Kingdom. The results were:

Name of Hospital	No. of beds	Daily average occupied	No. of in-patients	No. of out-patients	Average cost per bed excluding out-patients	Average cost per bed including out-patients	Average cost of in-patient	Aveaage cost of in-patient per day	No. of patients to each bed per year
Victoria London	42	38	523	16,869	£116 16 0	£150 5 0	£8 10 0	6/3	14
Evelina, ,,	66	47	1,017	16,190	89 10 0	111 15 0	5 5 0	4/11	17
N.East ,,	57	47	773	19,116	104 10 0	135 0 0	6 5 3	5/9	16
Paddington Green	46	37	592	16,203	81 5 0	114 15 0	5 0 0	4/5	16
Birmingham	62	54	1,003	—	81 5 0	—	4 10 0	4/5	19
Derbyshire	40	21	175	1,594	44 15 0	50 10 0	5 5 0	2/9	8
Aberdeen	85	78	712	918	42 10 0	43 5 0	4 15 0	2/8	9
Glasgow	74	65	810	—	61 0 0	—	5 0 0	3/11	12
Belfast	44	44	826	5,376	40 10 2	49 13 0	2 13 0	2/8	18

It was found that the average cost per bed including out-patients was lowest in Belfast. Moreover the cost of laundry per patient per year was 6/3d in Belfast compared with 8/10d and 10/6d in two English hospitals. There were fewer nurses per bed in the Belfast hospital than in any of the others, and Belfast came second to Birmingham in the number of patients admitted to each bed per year.

There was thus ample evidence of good management but the financial situation was so bad that there was talk of reducing the bed complement. This disaster was avoided only by the generosity of the Jaffé family. Sir Otto Jaffé, who had been honorary treasurer of the hospital since 1891, announced at the annual meeting in 1903 that his wife would contribute one hundred pounds if ten others would do the same. The challenge was readily accepted, and the sum of £1,371 was contributd within six months. It was in the same year that Sir Otto's gift of £3,000 saved the Better Equipment Fund of the Queen's College from perishing. Many members of the Ulster Medical Society will remember studying physiology in the Jaffé Laboratories in Queen's.

Sir Otto Jaffé continued to serve the hospital until early in 1916 when he was forced to resign his office because of anti-German feeling. He had been born in Germany but had lived in Belfast for 57 years. One who was described as "a large subscriber to the funds" informed the Board that she would not contribute further "until something is done about striking off the name of any German who may be on any of the Committees". The Board did not actually strike Sir Otto's name from their membership but they accepted his resignation without any recorded protest.

When Lorrain Smith left Belfast in 1904 for Edinburgh he was succeeded in the chair of pathology in the Queen's College by William St Clair Symmers (Fig. 18) who also succeeded him as honorary pathologist to the Children's Hospital. Andrew resigned in the same year and was succeeded as honorary dental surgeon by T. Norman Whyte. The Board appointed Andrew to the consulting staff but his name does not appear as such in the published lists. The omission is now rectified *in memoriam.*

FIG. 18. *William St. Clair Symmers*

Miss McTaggart resigned from the nursing staff in 1906 to marry Robert Campbell. In addition to her multifarious duties as matron she had been a great organiser of fund-raising activities. It was due to her that electric light was installed in the hospital at no cost to the Board. She had also been successful in getting her salary raised in 1901 when she let it be known that she had been offered a post elsewhere. The Board at once resolved

FIG. 19. *Miss Constance Rome*

that her salary be raised from £70 to £85 and expressed their very high appreciation of her services.

The new matron was Miss Constance Rome (Fig. 19) from the East London Hospital for Children. The salary was again reduced to £60. Miss Rome resigned in 1908 to marry T. S. Kirk and was succeeded by Miss Lockwood from the Pendlebury Children's Hospital. A newspaper photograph of 1911 (not reproducible) shows Miss Lockwood with a staff of eleven nurses. There had been two nurses when the hospital moved to Queen St.

In 1906 the Ladies' Committee expressed concern about the large number of tuberculous children in the hospital. They saw no hope of improvement before the millennium. However we have not had to wait so long, and tuberculosis in children is almost a rarity now in Belfast

The ladies have left a picture of the condition of poor children in Belfast at the time:

> As a sample of the judgment of the mothers, the children come to hospital with practically no underclothing . . . the groundwork of their toilet being a huge roll of felt. . . .

They also mentioned a child being brought into the hospital dying of starvation.

Labourers were then being paid one penny for shovelling a ton of iron ore, the corresponding rate in Britain being four pence. Grain workers had to lift 100 tons a day for five shillings. A social historian writes that the pace was such that they could not stand it for more than three days and "by labour which strained every muscle to the breaking point" and "feverish recklessness menacing life and limb" they could contrive to earn fifteen shillings a week.

What of the mothers? The Sweated Industries Act laid down a minimum wage of three pence per hour for women workers but it was not being observed in Belfast. In 1909 the Medical Officer of Health reported that the ordinary rate for outworkers was one penny per hour and often less.

What of the children? It was remarked (Greeves, 1961) that the aspect of Belfast most astonishing to the visitor would be the part-time system of little children working from early morning.

FIG. 20. *Malcolm Brice Smyth*

The appointment of Malcolm Brice Smyth (Fig. 20) as assistant physician on the resignation of Beatty in 1907 established a record. He was the third member of his family to be elected to the honorary medical staff. A contemporary photograph shows the staff at the time (Fig. 21) and another (Fig. 22) one of the wards. Sidney Brice Smyth died in 1908 after 15 years service, Leathem was promoted full physician, and the new assistant physician was James Colville (Fig. 23).

The death of Robert O'Neill in 1910 removed an outstanding friend of the hospital. He had been president for 27 years and had played a very active part, notably in the "Word of God" controversy with the Ulster Hospital and in the establishment of the Queen Victoria Convalescent Home for Children – a project

FIG. 21. *Medical Staff of B.H.S.C.*, 1907.

Standing
(l. to r.):
T. S. Kirk,
R. Campbell,
S. Brice Smyth,
A. Fullerton,
R. R. Leathem,
W. M. Burnside.

Sitting:
John McCaw,
J. Nelson.

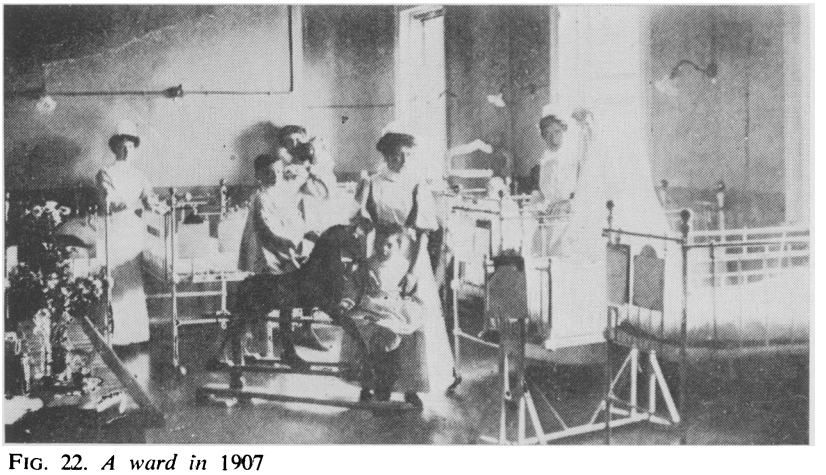

FIG. 22. *A ward in* 1907

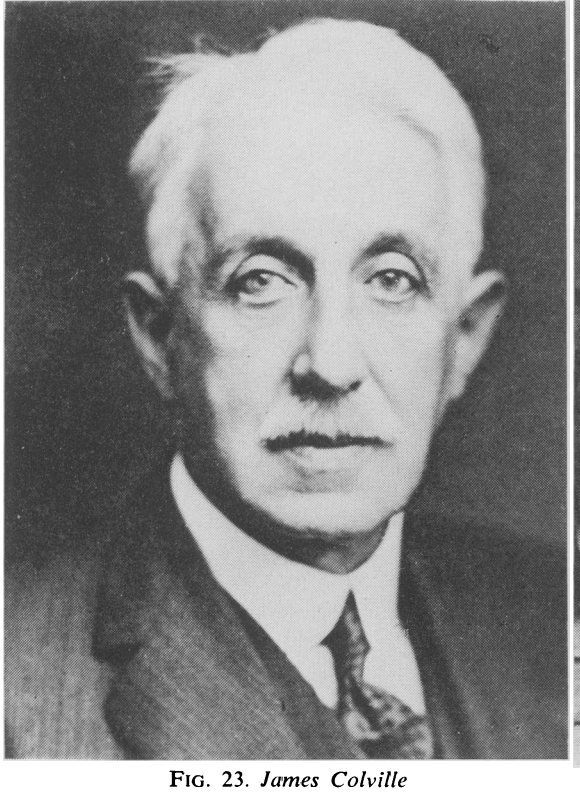

FIG. 23. *James Colville*

FIG. 24. *Capt. the Hon. Arthur B. O'Neill, M.P.*

he had advocated with vigour for some years before the Board could be persuaded to take it up. He was succeeded as president by his nephew, Captain the Hon. Arthur B. O'Neill, M.P. (Fig. 24), father of Lord O'Neill of the Maine.

Joseph Nelson died in 1910, and his widow presented his instruments to the hospital. His successor as oculist and aurist was Wiclif McCready (Fig. 25). In the same year Percival Templeton Crymble (Fig. 26) left the anaesthetist's end of the operating table where he had been officiating for some time and became honorary assistant surgeon in succession to Brian O'Brien who had joined the medical staff of the Local Government Board in Dublin, thus following in the footsteps of John Fagan. One of the unsuccessful candidates for the surgical post was Rowland Hill (Fig. 27) who took Crymble's place as an anaesthetist until 1912 when he was elected an assistant physician in place of Colville who had been forced to resign on account of ill health.

FIG. 25. *Wiclif McCready*

FIG. 26. *Percival T. Crymble*

The hospital had now been in Queen Street for 30 years, the district had become less residential and more industrial, and there was talk of moving again. However, Kirk and Campbell told the Board that the medical staff was satisfied with the existing location. It took the Great War to make

FIG. 27. *Rowland Hill*

give anaesthetics because "Miss Knox had been giving anaesthetics for many years and we wish to relieve her of this duty as she has too much to do." In order to prevent misunderstanding, McCaw added that he did not wish the Board to think that any reflection was being cast on Miss Knox by the staff's request. Miss Knox (now M.B.E.) died in the Children's Hospital in 1966 at the age of 84 years. One of the wards in the Falls Road hospital bears her name.

When Captain O'Neill was killed in action in 1914 (the first member of the House of Commons so to die) his

FIG. 28. *Miss Annie P. Knox*

them discontented with Queen Street.

The last change of matron that I shall have to record occurred in 1913 when Miss Annie P. Knox (Fig. 28) succeeded Miss Lockwood, again at a salary of £60 per annum. Miss Knox was the first locally trained nurse to be appointed matron of the Children's Hospital. She held the post for 35 years (still the record), serving until 1948 in which year the hospital's independent life ended. During the Great War Miss Knox worked in very difficult conditions; a shortage of anaesthetists meant that she must do their work as well as her own. As late as 1923 we find the medical staff asking for a second house surgeon to

FIG. 29. *Hon. Hugh O'Neill*

younger brother the Hon. Hugh O'Neill (Fig. 29) (now Lord Rathcavan) was invited to succeed him in the presidency. He was unable to do so as he was on active service but in 1918 at the end of the war he assumed the office, thus becoming the fourth member of his family to be president of the Children's Hospital. He held the post until it was abolished in 1948 when the hospital was taken into the National Health Service.

The medical staff was seriously depleted in 1915 when Crymble, Fullerton, Hill and McCready left for service in the Forces. Mackenzie came back from retirement to help with the surgical work, and Campbell took upon himself the largest part of the burden of the numerous operations performed both in the intern and extern departments.

Income fell and expenses rose but in spite of the cost of commodities being doubled household expenditure between 1915 and 1918 rose by only 34 per cent – a tribute to Miss Knox's economical management. Nevertheless the average annual cost per bed rose from just under £16 to over £33.

By 1919 the medical staff was again at full strength but the mood of contentment of 1910 had vanished. They now expressed concern about the length of the surgical waiting list, about the need for X-ray apparatus and about the need for a new hospital. The only action the Board took at the time was to create the post of extern house surgeon. The first to hold it was a woman.

The death of Campbell in 1920 left the way open for Fullerton to become full surgeon after serving as assistant surgeon for 22 years. Compare this with Campbell's promotion after only one year and Kirk's after only two. Fullerton's successor as assistant surgeon was Henry Potter Hall (Fig. 30).

Early in 1924 John McCaw died. He had been intimately connected with the hospital for 42 years and shortly before his death had been elected vice-president of the hospital. Malcolm Brice Smyth was promoted full physician and succeeded as honorary attending physician in charge of out-patients by Frederick Martin Brice Allen, the post of assistant physician having been abolished. In the same year McCready resigned and was succeeded as honorary oculist by Frederick Jefferson

who served only for a short time and was succeeded in 1926 by Frederick Alexander MacLaughlin.

The staff continued to grow, and in 1924 the first honorary bacteriologist was appointed. He was Joseph Tegart Lewis. In 1925 Ivan Henry McCaw (Fig. 31) was appointed the first honorary physician in charge of the skin department. He was the son of John McCaw.

In 1927 the medical staff recommended that the Board should create an additional post of honorary attending assistant surgeon in charge of out-patients. There were two candidates for the post, and the Board took the unprecedented step of discussing the

FIG. 30. *Henry Potter Hall*

suggestion by one of its members that they should make two appointments. This was deemed irregular and Ian James Fraser was elected to the post. The Board then asked the medical staff if the services of another assistant surgeon could be made use of but the answer was in the negative. The records of this appointment refer to the post as that of "assistant surgeon" but this is a misnomer as in the staff lists it always appears as "surgeon". The designation "assistant" had in fact been abolished by the Board in 1911.

Fullerton resigned in 1928 after over thirty years service

FIG. 31 *Ivan Henry McCaw*

and was succeeded both in the hospital and in the chair of surgery in Queen's by Crymble. The latter's post of out-patient surgeon was not filled because the medical staff advised the Board that the existing staff could carry out the work adequately without addition.

On Leathem's resignation in 1929 Hill moved from the extern to the wards and was succeeded as out-patient physician by Thomas Howard Crozier. The story of the X-ray department should begin in the same year with the appointment of Richard McCulloch as its first honorary physician. However, there never was an X-ray plant in the Queen St. hospital and so there was no X-ray department for McCulloch to preside over.

There had been no dental surgeon on the staff since the death of Whyte in 1919. In 1931 J. C. McNeill who had been doing dental work in the hospital for some time was appointed honorary dental surgeon. He was to serve for over 30 years.

The dissatisfaction of the medical staff with the hospital continued to grow and to be expressed more vigorously. They said that in some cases they were even recommending that children should seek advice elsewhere, and in 1925 the medical report remarked:

> For many years the Belfast Hospital for Sick Children occupied the premier position . . . but now it is surpassed by other charitable organizations and even by Poor Law Institutions in Northern Ireland.

It was therefore with much relief that the new hospital on the Falls Road was opened in April 1932 – exactly 53 years after the opening of the Queen St. hospital. Crymble gave a warning about the harmful effects of the inertia that might follow promotion from the cellar to the palace. The old building was sold for £6,500 – a a profit of almost £2,000. It became a station of the Royal Ulster Constabulary.

This briefly is the story of the Queen St. days. The "old, small, inconvenient hospital in King St." gave way to a hospital "perfect in all its details and second to none in the Kingdom" as it was described in 1879 but "pitifully inadequate" as it was described fifty years later.

When Darbishire spoke in 1879 he referred to the "many good men who have gone from us . . . leaving in our hospital a monument which is more valuable than any pillar erected in market square or mountain top, which is merely of stone, while our hospital is a living stream of usefulness." That is how it was; that is how it is. The abode changes; the work continues.

Acknowledgements

I am indebted to the authorities concerned for access to the records and reports of the Royal Belfast Hospital for Sick Children and to the following for the loan of photographs and portraits: Reverend A. L. Agnew, Mr. W. J. Bell, Dr. J. W. Browne, Mrs. K. D. Campbell, Sir Ian Fraser, Mrs. I. H. McCaw, Lord O'Neill of the Maine, Lord Rathcavan, Dr. R. W. M. Strain, Professor William St. C. Symmers sen., the Ulster Medical Society and Mrs. D. Walmsley.

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CHRONIC PERITONITIC ENCAPSULATION OF THE SMALL INTESTINE DUE TO TUBERCULOSIS WITH PROLONGED HIGH JEJUNAL OBSTRUCTION

by

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PERITONEAL ENCAPSULATION of the small intestine is an anomaly in which the whole small intestine is found in the mid-abdomen inside a large sac of normal peritoneum (Lewin and McCarthy 1970). Lewin and McCarthy described in their case a small non-constricting aperture in the sac in the lower right quadrant of the abdomen from which the terminal ileum emerged. Superiorly the sac enclosed the duodeno-jejunal junction. Hardas (1970) reported a case associated with malposition of the gut.

Chronic peritonitic encapsulation is a different disorder in which a globular or ovoid sac encloses the small intestine, but the membrane is thick, though not adherent to the surface of the small intestine loops, which themselves are aggregated, and adherent to each other. Presumably this may rarely be the result of chronic peritonitis in a patient with congenital peritoneal encapsulation, but it may more usually be the consequence of the laying down of an inflammatory membrane on the surface of a hitherto normal small intestine. In either case it may be tuberculous, but in many cases no aetiology can be determined. Albot *et al* (1970) have described a case in which jejuno-ileitis of unknown origin led over six years to encapsulating peritonitis with high jejunal obstruction and death. There was no histological evidence of Crohn's disease, nor any of tuberculosis. Albot's case was explored in the second year and the peritonitis was seen in the more acute stage when there was not yet a capsule. Pezzi (1967) described two cases of encapsulating peritonitis with high jejunal obstruction. Both were explored and both survived. There was no histological evidence of any particular disease. However negative histology may not necessarily exclude tuberculosis.

Case Report

The patient, a single woman, aged 34 at the onset of this illness, had been admitted to the Royal Victoria Hospital 14 years before for pleural effusion. Mycobacterium tuberculosis was isolated from the pleural fluid at that time. She made a good recovery from the pleurisy. The present admission was for a continuous fever which had lasted ten days. In spite of the high fever she did not look very ill. There were in the beginning no helpful physical signs. Haemoglobin was 66 per cent. Serum iron was 37 micrograms per cent. E.S.R. 72 mm/one hour. There was an occasional little dry cough. There was no sputum. The chest X-ray showed a small discrete soft shadow in the left upper zone of uncertain significance. Because the fever continued and there seemed no likelihood of establishing a certain diagnosis, anti-tuberculous drugs were begun on the 7th hospital day. Thereafter the fever declined slowly and she became afebrile on the 28th hospital day.

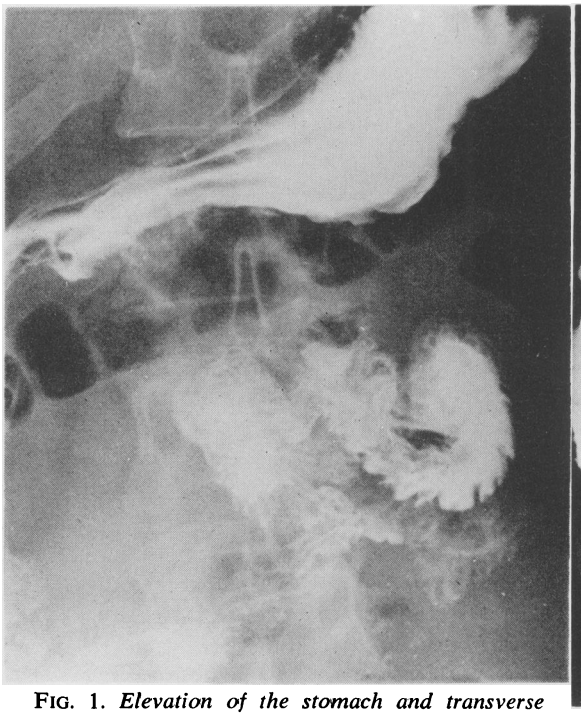

FIG. 1. *Elevation of the stomach and transverse colon by the pseudo-cyst of encapsulating peritonitis. The barium is passing freely into and through the duodenum and upper jejunum.*

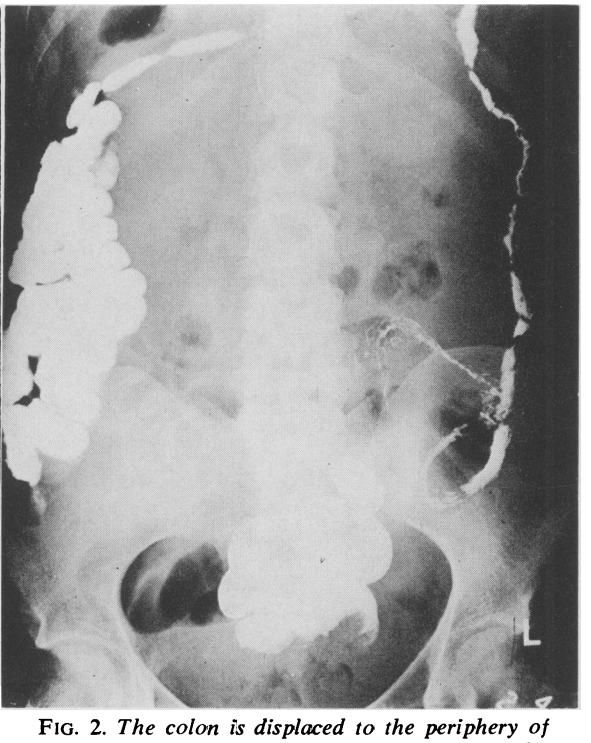

FIG. 2. *The colon is displaced to the periphery of the abdomen by the central abdominal pseudo-cyst.*

On the 20th hospital day the abdomen was noted to be a little full or tumid, and not wasted, as the rest of the body was wasted. The haemoglobin had fallen to 60 per cent. On the 21st hospital day the barium meal showed appearances which in retrospect we realise are consistent with encapsulating peritonitis. The stomach is elevated (Figs. 1 and 3) and the colon is displaced to the periphery (Fig. 2) by the central mass, which contains the small intestine, its loops gathered into a central position (Fig. 3). At that time the barium passed freely enough through the small intestine, though there may have been some delay in the terminal ileal loops. There was no dilatation of the upper jejunum or duodenum (Fig. 1).

About the 25th hospital day abdominal pain and vomiting of obstructive type began, and continued, and grew worse. On the 28th day (when the patient was afebrile) X-ray examination with a Gastrografin meal showed dilatation of the duodenum, and the contrast did not pass beyond the 4th part (Fig. 4).

Because of the obstruction the abdomen was explored on the 29th hospital day. A large pseudo-cyst was found centrally, containing the small intestine (Fig. 5). The cyst wall, which was very thick and white, was opened and partially removed, the contained small bowel was seen to be inflamed, red and friable, and the loops were bound together with adhesions. The cyst contained a small quantity of serous fluid. A narrow compressed transverse colon was found encased superiorly in the

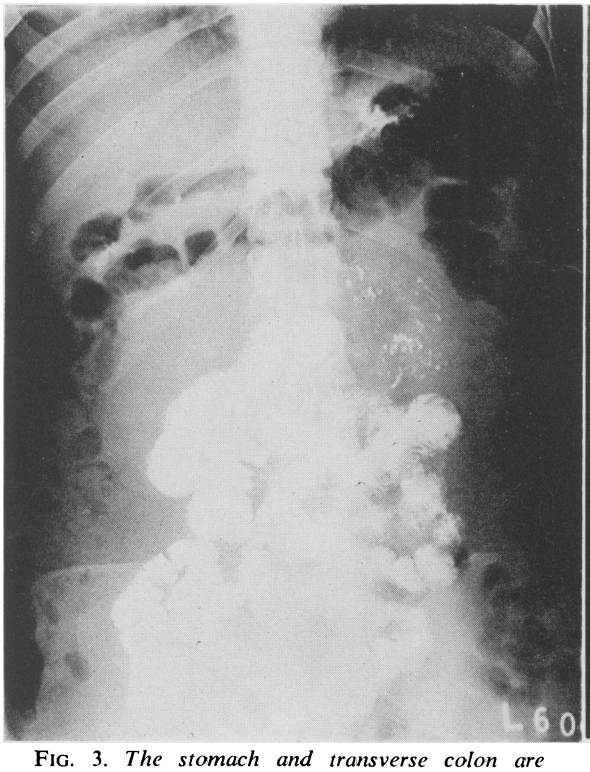

FIG. 3. *The stomach and transverse colon are elevated. The small intestine is gathered into the centre of the abdomen and retained there by the encapsulating pseudo-cyst wall.*

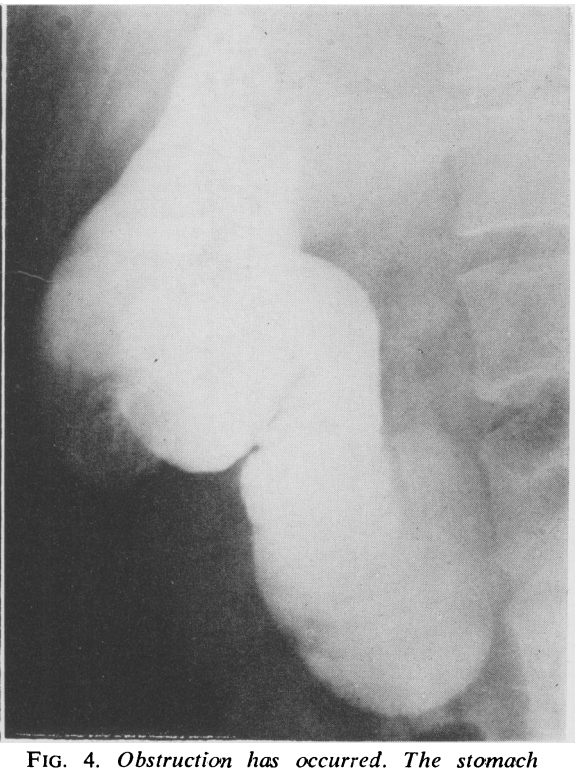

FIG. 4. *Obstruction has occurred. The stomach and duodenum are much dilated. Barium is not passing from the megaduodenum into the jejunum.*

cyst wall. The site of obstruction was found to be thickening in the mesocolon around the duodeno-jejunal region. A surgical by-pass was not possible owing to the thick oedematous jejunum making a gastro-jejunostomy hazardous. Above the cyst the liver and adjacent structures were found to be free of peritoneal thickening.

The obstruction continued unchanged after the operation. The patient was maintained with intravenous nutrition, and the anti-tuberculous drugs were continued. The obstruction continued till the 60th hospital day (it had begun on the 25th), and then the gastro-duodenal aspirations diminished. By the 64th hospital day she was able to eat. At the time the obstruction ceased, after five weeks of intravenous nutrition, staphylococcal bacteraemia with fever occurred. It yielded to treatment with cloxacillin and withdrawal of the intravenous cannulae. However, a septic arthritis led to a stiff right knee.

COMMENT

This note draws attention to encapsulating peritonitis as a manifestation (in this instance) of abdominal tuberculosis. The histological report on specimens of the cyst wall recorded "numerous granulomata with Langhans and foreign-body type giant cells, epithelioid cells and chronic inflammatory cells. In some granulomata

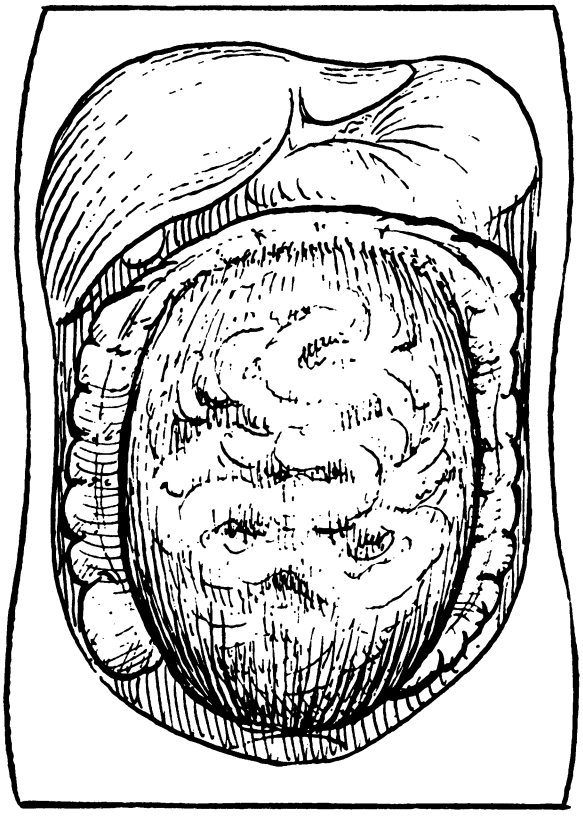

FIG. 5. *Diagram of the appearance of the encapsulated small intestine at operation. The small intestine loops are indicated not because they could be seen through the wall of the unopened sac but to show their situation in the sac.*

there are central areas of necrosis". This seems consistent with tuberculosis. No mycobacteria were seen in Ziehl-Neelsen staining.

In this case anti-tuberculous drugs were of great benefit. When no drug could be taken by mouth, she was treated with the injection of isoniazid as well as the injection of streptomycin. The anti-tuberculous drugs were continued for a year. The patient did well, and is in good health and at work. It is not clear why the obstruction set in when the tuberculous infection was beginning to improve, nor is it clear in what way the obstruction resolved in the end.

High jejunal obstruction seems a common complication of encapsulating peritonitis whatever the aetiology. However, obstruction at the duodeno-jejunal junction by a tuberculous stricture in patients with abdominal tuberculosis, but without encapsulating peritonitis, seems to be well-known in areas where abdominal tuberculosis is still common. Desai and Shah (1970) report nine cases. The patients complained of upper abdominal pain and loss of weight. On X-ray examination there was distension of the duodenum. At operation the stricture at the duodeno-jejunal junction was found.

The possibility of resolution, be it spontaneous or drug-induced, makes necessary resolute, prolonged, intravenous nutrition. It is best to rely for calories on glucose, aminoacid and fructose-ethanol-aminoacid solutions. The serum potassium and

magnesium should be maintained at normal levels. One may use the injection of potassium chloride and dextrose B.P.C. intravenously, and the injection of magnesium sulphate, 50 per cent solution, intramuscularly or intravenously. Hydroxocobalamin and folic acid should be injected, as well as ascorbic acid and the B group of vitamins. A normal iron state, obtained by the injection of Jectofer, helps to support the small intestine mucous membrane, as well as correcting iron-deficiency and anaemia. Infection of the intravenous cannula and thence of the blood stream is a serious danger. The sooner intravenous maintenance can stop, the better.

The X-ray appearance even when there is no obstruction (Figs. 1, 2 and 3) are suggestive enough of encapsulating peritonitis, and may make a pre-operative diagnosis possible. When the case is one of high jejunal obstruction (Fig. 4) encapsulating peritonitis should be considered as a possible cause.

Summary

A case of encapsulating peritonitis of tuberculous origin, complicated by high jejunal obstruction, is described. The obstruction resolved after 35 days of intravenous nutrition. Anti-tuberculous treatment restored the patient to health and full work. The X-ray appearances are helpful in diagnosis, and are reproduced.

Acknowledgements

We are indebted to Dr. F. Alexander for the histological report. The reproduction of the x-rays is by the Royal Victoria Hospital Department of photograhy. The diagram is by Mr. G. Smith, artist at the Royal Victoria Hospital.

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SUDDEN UNEXPECTED DEATH IN INFANTS ('COT DEATH') REPORT OF A COLLABORATIVE STUDY IN NORTHERN IRELAND*

by

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INFANTS HAVE always been known to die suddenly and unexpectedly in bed, their deaths being formerly ascribed to 'mechanical suffocation' by clothing or by 'overlaying' – of which there are presumptive examples from early literature (I Kings, 3:19). Early investigators (e.g. Templeman, 1892) stressed this danger and in Ireland the powerful temperance lobby obtained a clause (sect. 13) in the 1908 Children's Act under which an adult, if 'at the time of going to bed [be] under the influence of drink', could be prosecuted for 'overlaying' a child. Current views are different: sudden unexpected death in infants ('cot death') is now considered to be a clinical syndrome and the majority of cases are thought to have an identical – though as yet unidentified – 'final common pathway' of death. Victims, essentially healthy throughout life, die seemingly because during a developmental stage of physiological vulnerability some combination of intrinsic and extrinsic factors proves fatal. Among the many hypotheses advanced those postulating (i) asphyxia from laryngospasm or nasal obstruction, (ii) cardiac conduction disturbance, and (iii) some hypersensitivity or aberrant immunological reaction, are currently tenable.

Little was know about 'cot death' when we initially planned this study; in fact the report of the 'First Conference on Sudden Death in Infants' (Wedgwood and Benditt, 1965), which first brought together pertinent findings from diverse disciplines, was not then published. Accordingly, we aimed for breadth of knowledge through wide fact-finding; we neither specifically tested formulated hypotheses nor restricted our enquiry to a detailed study in any one discipline. Furthermore, because of the general dearth of data on this condition we described our results piecemeal as they became available (Froggatt, Lynas and Marshall, 1968; Froggatt, 1970a, b; Marshall, 1970; Froggatt, Lynas and MacKenzie, 1971). Some of the findings, however, have not yet been reported; documenting these, and presenting a coherent synopsis of our study are the purposes of the present paper.

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*Members of the working party were Dr. T. K. Marshall (chairman), Professors I. J. Carré, K. B. Fraser and P. Froggatt, and Drs. W. Bamber, D. J. L. Carson, Margaret Lynas, J. A. McLaughlin and A. L. Walby. We thank our colleagues for their confidence in entrusting us with the description of this study.

To minimise citation of the now extensive literature we reference specific work only when essential or when not covered in recent thorough reviews (Valdés-Dapena, 1967; Froggatt *et al.*, 1968; Bergman, Beckwith and Ray, 1970; and Froggatt *et al.*, 1971). For brevity these four publications are not always cited when they are the source for unreferenced facts. Throughout the text the description of a difference as 'significant' means that it or a greater difference was unlikely to occur by chance in more than 5 per cent of repeated trials. χ^2 is everywhere calculated on absolute numbers and, where appropriate, corrected for continuity.

Many terms and acronyms describe this condition: 'sudden unexplained death in infancy' (SUD), 'sudden unexpected infant death' (SUID), 'sudden unexpected, unexplained death in infants' (SUUD), and 'sudden infant death syndrome' (SIDS) – and their variants – are all used and in practice are synonymous with the colloquial 'cot death' ('crib death' in U.S.A.). In this article we use for convenience the acronym SUD.

Ascertainment

Selection of Cases

Ascertainment was through the Northern Ireland Forensic Pathology Service (NIFPS) – a government service run exclusively for the use of the country's 16 coroners – and covered the two years 1st August 1965 to 31st July 1967. Strictly, SUD infants would be coroners' cases under law (Coroners Act, 1959), but to maximise ascertainment we apprised general practitioners and the Northern Ireland General Health Services Board and one of us (T.K.M.), as State Pathologist, asked the coroners to demand an autopsy on young children as a routine. As a result, 297 children aged under five years were autopsied by one of five similarly-trained forensic pathologists in the NIFPS to a standard routine.

Since there are no recognisable positive autopsy criteria of SUD we reached our study group by exclusion. From the 297 children we excluded 78 who died from unnatural causes, e.g. violence and drowning; 29 in which there was an adequate natural cause for death other than respiratory inflammation (which some consider to be the cause of SUD), e.g. peritonitis; 21 in which respiratory inflammation co-existed with other lesions, e.g., mongolism, renal vein thrombosis, and congenital heart disease, which were considered adequate to explain death; and five which were difficult to appropriately allocate because of administrative problems. All but two of the remaining 164 children were in the 'conventional' age group 2–103 weeks (respectively four days and 144 weeks), and these were excluded leaving 162 'unexplained' cases for study. These are termed below 'index cases'. Interviews were conducted by one of us (M.A.L.) with the families (nearly always the mother) of 148 (91.4 per cent) of these index cases: of the 14 failures, eight families refused, two were untraced, three had left the country, and one case was *sub judice*.

Selection of Controls

There is no satisfactory control group for SUD. Accordingly two (Groups A and B) were selected each to serve in the analyses where appropriate. Group A comprised the (chronologically) next like-sexed birth (identified from birth notifications) in the same administrative area or sub-area division as that of each of the 148 index cases whose families were interviewed. Matching was therefore by

sex, county or county borough of domicile – or (in two counties) sub-area division – and date of birth. Only two second-choice controls were necessary. No family declined interview. Group B comprised the 42 infants, aged 2–103 weeks, from among the 107 (78+29) children under five years, mentioned above, whose death was unnatural or due to certain natural causes. A third control group was selected for some multivariate analyses the results of which are described elsewhere (Froggatt, 1970a).

Completeness and Bias

The nature of SUD precludes an accurate assessment of the completeness of the ascertainment, but we can reach a working estimate as follows. The records of children of Belfast-resident mothers dying in Belfast C.B. aged under one year yielded, for the study period, 91 infants aged 2–51 weeks with principal and, when documented, also contributory certified cause of death 'pneumonia' or associated conditions *viz.* ICD (Seventh Revision) categories 470–475, 480–483, 490–493, 500–502, 525 and E921. Because the NIFPS certify SUD in one or other of these categories, these cases should contain the Belfast index cases. Of these 91 infants, 13 had died in hospital with clinical bronchopneumonia (confirmed in eight at autopsy) and five had died (none autopsied) either at home, in the admission ward, or in transit to hospital in each case being under treatment for at least 12 hours for respiratory symptoms, often severe. None of these 18 was considered as notifiable to the coroner. We found that the remaining 73 comprised 72 of our index cases and one other infant death reported to the coroner on which an autopsy had been carried out by a pathologist not in the NIFPS. No child who died suddenly and unexpectedly for an 'unexplained' cause was missed and our Belfast ascertainment is therefore some 94–100 per cent 'complete' depending on whether only 13, or all 18, of the non-notified 'bronchopneumonias' above be accepted as 'explained'. Similar information is not available outside Belfast: but from the NIFPS figures (90 SUD outside Belfast) and the numbers of registered death 4–51 weeks in the Registrar General's tabulations under the above ICD categories (98 in Belfast C.B.; 151 in other areas), and accepting the Belfast ascertainment as 94 per cent complete, we can, *certeris paribus*, estimate the ascertainment outside Belfast as at least 83 per cent complete $\left(\frac{94 \times 98 \times 100}{151 \quad 73}\right)$ with an overall figure of, say, 85–90 per cent for Northern Ireland.

With this high level of 'completeness' important bias would be introduced only by the mechanism of ascertainment and the criteria themselves. The following are the most pertinent.

(a) Due to the selection criteria – whereby respiratory inflammation as a sole finding was not a criterion of exclusion – index cases are biased towards displaying pathology of the respiratory as compared to other systems. The respiratory inflammation was, however, slight (see below) and in only some 10 per cent would death have been considered 'explained' or 'possibly explained' in the terminology of Beckwith's (1970) rigorous criteria.

(b) Some index cases were ill for (usually short) periods pre-mortem yet their deaths were 'unexplained' at autopsy. These could have been excluded on the strict interpretation of the term 'unexpected' in SUD, but in fact few investigators exclude

such cases since they judge them as sufficiently 'sudden' and 'unexpected'. Bias and over-ascertainment should therefore be small.

(c) Some children who were excluded because of autopsy findings considered adequate to 'explain' death, may have actually been SUD cases. This bias operates in all surveys and leads to universal under-ascertainment of an unknown degree.

(d) Ascertainment through the NIFPS could bias selection against neonates. An autopsy on a neonate dying 'unexpectedly' in a maternity hospital would be carried out by a *hospital* pathologist. In addition the death of a neonate at home would be less likely to be reported to the coroner – death in a neonate being more common and less remarkable than in an older child. On the other hand, neonatal deaths are generally unlike SUD, and autopsies usually show presumptive non-SUD conventional causes of death. We consider, therefore, that missed cases should be few.

Materials and Methods

We contacted the families of the bereaved and control group A children through local health authority staff. The interviews, timed optimally for two weeks after the death, were conducted by one of us (M.A.L.) to a set scheme. Information, recorded on a standard schedule using mostly pre-coded responses, specified (index cases and controls are collectively designated 'subjects'): essential ante-natal, birth, and medical data on subjects and their sibs and the medical data also on their parents: legitimacy, father's social and economic status; parental ages, stature, consanguinity, and marriage date; specific history of (for the index case) the week preceding death and (for the control) the week preceding interview; feeding regime and medicine intake since subject's birth; date, day-of-the-week, time, and detailed circumstances of death of index cases; sleeping and feeding routine of subjects; state of housing, maternal care, and indices of domestic crowding; and electro-cardiograms (bipolar limb leads) of parents of index cases. A two-month pilot study preceded the main survey, cases ascertained being later discarded.

Only those results not previously documented, pertinent to the more plausible hypotheses of causation, or which are essential to coherently define the entity SUD are presented here: other findings are detailed elsewhere (Froggatt *et al.*, 1968; Froggatt 1970a, b; Marshall, 1970; Froggatt *et al.*, 1971).

Autopsy Results

Gross pathology

Most of the 162 infants seemed well-nourished (see later). A nappy rash, rarely severe or extensive, was found in 47 (29 per cent). None had any internal injury and only seven (4.3 per cent) bore external marks of trauma and these were always trivial. Hypostasis was unremarkable and it was rare to find any contact pallor over the nose and mouth such as could have indicated occlusion by pressure on the pillow or mattress.

The deaths of 35 infants (21.6 per cent) were associated with some escape of fluid from the nose or mouth and fluid of one kind or another – watery, mucus-like blood-tinged froth, milk curds – generally in very small amounts was also found in the trachea of 112 cases (69 per cent). Sometimes the fluid came from oedematous lungs; sometimes it had been regurgitated. However, there was no indication that

fluid from the stomach played a role in causing death; and microscopy neither revealed aspirated foreign material in the medium sized bronchi nor areas of aspiration autolysis. It probably entered the air passages in the agonal period or even post-mortem.

Petechial haemorrhages were present on the thymus, heart and lungs, in 48 cases (30 per cent); in 29 (18 per cent) they were on two of these organs and in 33 (20 per cent) on only one, most commonly the lungs (17 cases). This frequency (110 of 162 cases) is significantly greater than that of group B controls (12 of 42 cases) – $\chi^2=21.46$, d.f.$=1$, $P<0.001$ – confirming the experience of notably Beckwith (1970). These petechiae were more common in cases found unequivocably dead (83 of 109) than in those allegedly seen to die or 'collapse' (20 of 39) – $\chi^2=7.26$, d.f.$=1$, $P<0.01$ – but they are unrelated both to age at death and whether or not the subject had symptoms or was allegedly perfectly well during the week before death. Their role in SUD is discussed later.

Microscopic findings

The main changes concern the respiratory apparatus, the typical appearance of which is briefly described, more details being in Marshall (1970).

The submucosa of the trachea and main bronchi were often infiltrated by lymphocytes and plasma cells but neutrophils were uncommon. Peribronchiolar mononuclear cells were present, sometimes amounting to 'cuffing'. This picture, however, is often seen in infants who die from injuries or carbon monoxide poisoning and so it was considered unspecific as was also the 'fibrinoid necrosis' of the vocal cords carefully studied by Pinkham and Beckwith (1970).

The lung parenchyma showed a variable degree of patchy oedema and areas of alveolar collapse often affecting only a few alveoli in each locus and in such a way that what on cursory examination seemed normally aerated tissue really consisted of strands of collapsed tissue interspaced with large air spaces comprising alveoli and alveolar ducts which had undergone compensatory dilatation. Such collapse can produce an apparent increase in cellularity of the alveolar tissue, a change usually seen in SUD lungs, but in our cases there were parts where thickening and cellularity could be shown to involve single alveolar septa thus favouring a true reaction rather than an artifact. Even so, the significance of an increased cellularity is difficult to assess. The lungs of like-aged infants dying suddenly from carbon monoxide poisoning or head injury are not obviously different. However, a detailed comparison showed SUD lungs to contain more alveolar cells (pneumocytes and septal cells) and their nuclei more often vesicular and deformed. Many of these cells had entered the alveoli where some had ruptured and provided much of the granular material within the alveoli. This picture is consistent with a reaction of the lung to some local or bloodborne stimulus.

Special Examinations

Blood groups

A syndrome causing sudden death in children shows, in Northern Ireland, segregation of the rare C^w allele in two families (Fraser, Froggatt and Murphy, 1964) and possible linkage of the abnormal gene with the Rh locus. Accordingly,

TABLE I

Blood group phenotype frequencies. Observed numbers of SUD cases and expectations based on stated controls

Phenotype	*Belfast*		*Extra-Belfast*		*Phenotype*			*Phenotype*		
	Obs.	*Exp.*[1]	*Obs.*	*Exp.*[2]		*Obs.*	*Exp.*[3]		*Obs.*	*Exp.*[4]
O	26	33.01	53	54.45	M	46	41.15	R_1r	46	47.44
A	24	19.82	29	28.05	MN	76	71.88	rr	28	21.61
B	8	5.67	11	9.35	N	23	31.97	R_1R_2	22	20.97
AB	2	1.50	1	2.14				R_1R_1	18	23.43
								R_2r	18	18.13
								Other	13	13.43
χ^2 (d.f.)	3.49 (3)		0.97 (3)			3.32 (2)			3.26 (5)	
P	0.30–0.50		0.80–0.90			0.10–0.20			0.50–0.70	

[1]Kopec (1970), pp. 98-99. [2]Kopec (1970), pp. 98-99 and Dawson (1964).
[3]Race and Sanger (1968), p. 91. [4]Heiken and Rasmuson (1966).

SUD cases were grouped for Rh (and also ABO and MNS) phenotypes and the frequencies compared with expectation derived from controls (Table 1). (Weighting the expectations on regional or 'postal district' frequencies seemed an unnecessary refinement). The differences were nowhere significant though the ratio $A_1/A_2 = 1.7/1.0$ is lower than that (3.7/1.0) suggested for southern England by Ikin, Prior, Race and Taylor (1939). Only one child had the C^w gene. These findings should be interpreted with caution because of the geographic variation in phenotype frequencies and the small number of SUD cases.

Serum proteins and immunoglobulins

In SUD cases and group B controls age-specific levels of serum albumin, α_1-globulin, and β-globulin were unremarkable but α_2-globulin was consistently elevated – which we interpret as due to autolysis. Immunoglobulin (IgA, IgM, IgG) levels, which had not previously been measured in SUD, were estimated using Hyland immunoassay plates to a standard technique (Collins-Williams, Toft, Generoso and Moscarello, 1967). The readings of two observers, and replications, were taken and, for IgG and IgA (where dispersion should *a priori* be minimally affected by freezing and thawing) the results were averaged, but for IgM (where such dispersion could be marked) the first reading was taken. Non-systematic inter- and intra-observer variation, the dependance of the real level on such factors as maturity and post-natal experience (Hobbs and Davis, 1967), the disproportionate effect of a few aberrant experiences, and the effect on molecule dispersal of repeated freezing and thawing, could all influence the results, but nevertheless the age-specific levels show reasonable concordance with those of other series of which the results of Stiehm and Gold (1968) are representative (Table II). Collation of individual readings with e.g. case history was not attempted because of the high error of the individual estimates.

TABLE II

Age specific immunoglobulin levels in mgm. per cent. SUD cases and results from Stiehm and Gold (1968)

Age (months)	*Immuno-globulin*	*N. Ireland SUD cases (median)*	*Stiehm and Gold* (1968) *SUD cases (mean)*	*Living infants (mean)*
1–3	IgA	28.5	17±12	21±13
	IgG	530.0	388±138	430±110
	IgM	41.0	27±14	30±11
4–6	IgA	38.5	44±31	28±18
	IgG	580.0	659±370	427±186
	IgM	57.0	42±19	43±17
7–12	IgA	49.0	36±18	37±18
	IgG	850.0	694±207	661±219
	IgM	44.0	43±17	54±23

Amino-acid chromatograms

'Nephrosis peptide' and increased excretion of sulphur-containing amino-acids have been described in SUD (Stowens, Callahan and Clay 1966). We attempted two-dimensional chromatography on 57 subjects but protein autolysis reduced confidently interpretable papers to 12 SUD cases and three group B controls. Of the former, six were normal, three showed cystathionine (or, possibly, phosphoethanolamine), two β-amino isobutyric acid, and one taurine; of the controls, one was normal, one showed 'nephrosis peptide' (in a child with left atrial agenesis), and one cystathionine. All abnormal constituents were in small quantities. Indole papers showed, in both groups, occasional aspirin metabolities, the tryptophan metabodities kynurenic acid and kynurenine, and (in one SUD case) 5-hydroxyindole acetic acid*. It is unlikely that these results are significant to the problem of SUD.

Blood urea

This was estimated on later cases in the series. The levels varied considerably but there were many over 100 mgm. per cent in both SUD cases and group B controls. In some it may have been agonal, and in some again it probably reflected a metabolic disturbance during the last few hours of life. The highest figures are usually associated with dehydration from vomiting or diarrhoea. This field might re-pay further study.

Bacteriology

This is described in Marshall (1970). Briefly, tracheal swabs were examined from 159 SUD cases and, after exclusion of contaminants, growths were obtained in 24 per cent compared to 20 per cent among control group B. Predominant

*We thank Dr. N. A. J. Carson for conducting and interpreting these tests.

organisms in the former were coagulase positive *Staph. aureus* (21 cases), haemolytic *Streptococcus* (12 cases) and *Pneomococcus* (5 cases). The controls produced mainly haemolytic *Strept.* Pleuropneumonia organisms were also sought in 76 SUD cases. None was detected: in 65 cases the results were negative while in 11 bacterial contamination made the plates uninterpretable.

Virology

This was restricted to a search for respiratory syncytial virus (RSV) and is described in Froggatt (1970b). Briefly, using tracheal swabs, impressions of lung, heart and kidney, and a fluorescent antibody technique, there was no single positive finding. This result must be considered at least in part artifactual – a few positives would have been expected irrespective of the cause of death and total failure is perhaps partly ascribable to antigen distintegration over the period (up to 18 months) of tissue storage. Other workers (e.g. Ray, Beckwith, Hebestreit and Bergman, 1970; Brandt, 1970) have described higher levels of successful isolation of most strains of virus from SUD cases than from various 'controls'; but interpretation is equivocal.

Other examinations

Other gross and microscopic examinations, e.g. otitis media, and special tests, e.g. presence of milk antibodies and nuclear sex chromatin content of buccal mucosal cells, are generally uninformative and have been described elsewhere (Froggatt *et al.*, 1968; Froggatt, 1970a, b; Marshall, 1970; Froggatt *et al.*, 1971).

Epidemiology

General Factors

Incidence

Adjusting the first estimate on the basis of overall 90 per cent ascertainment gives a 'corrected' incidence of SUD as 2.8 per 1,000 live births, 11 per cent of total infant (<1 year) mortality, and 33 per cent of postneonatal (4–51 week) mortality. These accord generally with the consensus urban experience in North America, Europe, Scandinavia and Australia; disparities with some series can probably be attributed to differences in diagnostic criteria, population structure, and survey methods. An incidence of 2.0–3.0 per 1,000 live births can therefore be accepted for Europeanised communities in temperate zones. Nothing is known of SUD in 'underdeveloped' countries: no surveys have been published and cases would most likely be swamped by the high infant mortality rates.

Regional variation

Authors differ as to whether or not there is an urban excess in SUD. Table III summarises our results which are confined to post-neonatal deaths to allow population comparisons. (This restriction excludes only nine cases). Columns (a)–(c) show our ascertainment and columns (d)–(f) are the rates per 1,000 live births based on our finding (above) that a 'complete' ascertainment in Belfast would yield not more than 75 post-neonatal SUD cases. Columns (g)–(i) are the estimates for SUD cases based on registered deaths in combined 'respiratory' ICD (Seventh Revision) categories, again on the Belfast experience, and columns (j)–(l) give

TABLE III

Regional incidence of SUD per 1,000 live births. Post-neonatal (4-51 weeks) cases

Administrative Area	Number of Ascertained Cases (1 Aug. 1965—31 July 1967)			Incidence of SUD based on 'Adjusted' Numbers of Ascertained Cases†		
	Males (a)	Females (b)	Total (c)	Males per 1000 Male Live Births (d)	Females per 1000 Female Live Births (e)	Total per 1000 Total Live Births (f)
County Borough						
Belfast	47	23	70	5.79	3.09	4.49
Londonderry	5	3	8	3.24	2.08	2.66
County						
Antrim	14	14	28	1.94	2.14	2.03
Armagh	4	6	10	1.39	2.29	1.80
Down	14	9	23	2.44	1.72	2.09
Fermanagh	0	1	1	—	1.09	0.53
Londonderry	4	5	9	1.36	1.83	1.58
Tyrone	4	5	9	1.26	1.71	1.47
Total	92	66	158**	2.82	2.21	2.54

† Adjustment factors, based on Belfast experience, are: Males=50/47; Females=25/23; Total=75/70 (see also text).

**Includes 5 cases ascertained outside the NIFPS.

the corresponding rates per 1,000 live births. Except for Belfast the two sets of rates are independently reached.

Th overall rates (colums (f) and (l)) are disparate only for Fermanagh, Londonderry County, and Tyrone. Taking the estimate in column (l) we reach, for the six counties, a weighted average of 2.29 per 1,000 live births (χ^2 of heterogeneity= 0.80, d.f.=4, $0.95>P>0.90$) which is significantly lower than the Belfast figure of 4.49 ($\chi^2=20.13$, d.f.=1, $P<0.001$) but not that (2.61) for Londonderry C.B. This Belfast excess is significant for males ($\chi^2=35.9$, d.f.=1, $P<0.001$) but not for females ($\chi^2=1.63$, d.f.=1, $0.30>P>0.20$), and it seems not to be a general factor of urbanity since there is no difference in the rates per 1,000 live births for pooled rural districts (2.35), and for pooled urban districts and metropolitan boroughs (2.25).

Sex

Males predominate in only four of some 60 post-war series (Froggatt *et al.*, 1971). Our study has 95 (58.6 per cent) males and 67 (41.4 per cent) females, the male excess being ascribably mainly to the atypical Belfast experience (Table III). We show elsewhere (Froggatt *et al.*, 1971) that this overall male excess probably patterns that for deaths in infants generally.

TABLE III—*continued*

Administrative Area	Estimated Number* of Cases based on Registered Deaths (1 Aug. 1965—31 July 1967)			Incidence of SUD given Estimated Number of Cases based on Registered Deaths		
	Males (g)	Females (h)	Total (i)	Males per 1000 Male Live Births (j)	Females per 1000 Female Live Births (k)	Total per 1000 Total Live Births (l)
County Borough						
Belfast	50.00	25.00	75.00	5.79	3.09	4.49
Londonderry	3.23	4.86	8.42	1.96	3.09	2.61
County						
Antrim	17.00	15.28	32.91	2.20	2.16	2.23
Armagh	6.45	7.64	14.54	2.10	2.67	2.45
Down	17.00	7.64	24.49	2.77	1.34	2.08
Fermanagh	0.81	2.79	3.83	0.80	2.79	1.91
Londonderry	6.45	8.33	15.31	2.06	2.82	2.50
Tyrone	8.09	8.33	16.84	2.37	2.64	2.57
Total	109.03	79.87	191.34	3.14	2.47	2.85

* Calculated by applying factors, based on Belfast experience, to number of registered deaths in pooled ICD (Seventh Revision) categories 480-483, 490-493, 500-502, 525, and E921. The factors are: Males=50/62; Females=25/36; Total=75/98. Thus in Londonderry C.B. there were 8.42 x (98/75)=11 deaths 4-51 weeks in the above categories. (See also text).

Age

This study confirms the unique age distribution, similar for each sex, which characterises SUD, *viz.* 75 per cent of cases between four weeks and six months, a mean of 18 weeks and a median of 14 weeks, and only 5 per cent of cases under one month and 15 per cent aged 6–11 months. This is highly dissimilar to overall infant mortality where the decrement curve is negative exponential.

Seasonal variation

SUD is commonest in the colder, rarest in the warmer months, again different to the distribution of other causes of infant death (Froggatt *et al.*, 1971). This winter peak, present also for the 40 per cent of cases described as symptom-free over the week pre-mortem, is more marked in the first year of the study (Fig. 1) – coinciding with an epidemic of type B influenza in January and February and type A2 in February and March 1966. This suggests epidemicity; we have not, however, been able to confirm this by time-series analysis nor by space-time clustering techniques (Froggatt *et al.*, 1971).

Social and economic status

Most authors have noted that SUD is commonest among underprivileged children. Our data confirm this: compared to four acceptable control groups (no

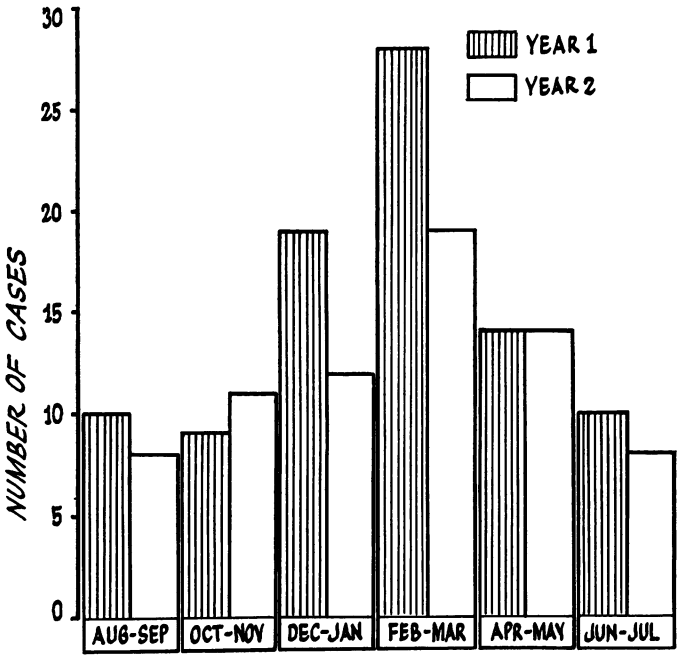

FIG. 1. *Seasonal incidence of 161 cases of SUD in Northern Ireland during the periods 1st August, 1965—31st July 1966 (Year 1) and 1st August 1966—31st July 1967 (Year 2). Reproduced by courtesy of the Editor from Amer.J.Cardiol.*, **22**, 457-468

single group is wholly appropriate for families ascertained through a SUD child) families of SUD cases are deficient in social classes I and II and over-represented in class V. Moreover, the fathers of 21 per cent of SUD cases compared to 8 per cent of group A controls ($\chi^2=8.51$, d.f.$=1$, $P<0.01$) were unemployed, or off work through sickness for more than six months; 7.4 per cent of SUD cases but no group A controls were illegitimate ($\chi^2=9.44$, d.f.$=1$, $P<0.01$); while room occupancy – 'crowding' - within social class was generally higher for the families of SUD cases than for those of group A controls. (Details are in Froggatt *et al.*, 1971).

To assess the independent importance in SUD of the above and other factors, we used a discriminant analysis, the two groups being Belfast singleton SUD cases and a random sample of Belfast surviving singletons, and the factors being sex, social class, parity, number of sibs claiming family resources, father's and mother's ages, and birthweight. Results (Froggatt, 1970a) showed a significant ($F=3.38$, d.f.$=7$ and 1,000, $P<0.01$) though unimportant difference between the mean discriminant scores but predictive power was too weak to be of practical importance.

Day of the week and time of the day

Cases occurred equally on each day of the week, but over the 24 hours the percentage distribution of the 'estimated' (Froggatt, 1970a) time of death in the three eight-hour periods from midnight, is 50.0, 36.4 and 13.6 (tested against equal frequencies, $\chi^2=32.93$, d.f.$=2$, $P<0.001$) which is similar to that (51.7, 33.3, and 15.0) for the sub-group of 60 index cases allegedly symptom-free throughout the week before death. More significantly, most infants were supposedly asleep at

TABLE IV

Distribution of SUD cases and Group A controls by maternal age and parity

Age of Mother (years)		*Birth Order of Child**										*Total*
		1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10+**	
⩽20	cases	3	7	1	—	—	—	—	—	—	—	11
	controls	8	1	—	—	—	—	—	—	—	—	9
20–	cases	11	16	12	11	3	—	1	—	—	—	54
	controls	15	10	6	1	1	—	—	—	—	—	33
25–	cases	2	5	8	8	6	7	3	1	1	2	43
	controls	10	8	9	12	4	3	—	—	—	—	46
30–	cases	3	6	3	5	3	2	2	1	—	1	26
	controls	4	3	8	6	7	3	4	2	1	1	39
35–	cases	—	—	—	2	4	—	1	1	1	1	11
	controls	1	—	3	3	1	1	2	1	2	—	14
40–44	cases	—	—	—	—	—	—	—	—	1	1	2
	controls	1	—	—	1	—	3	—	—	2	—	7
Total	cases	19	35	24	26	16	9	7	3	3	5	147†
	controls	39	22	26	23	13	10	6	3	5	1	148

*(Number of previous livebirths, stillbirths and miscarriages)+1.
†Birth order of one child unknown.

the time of death; certainly very few were being handled or patently awake at the time of the terminal event.

Maternal age and parity

These highly correlated factors entered the discriminant function (mentioned above) – where neither proved to be a significant determinant of SUD – but they were also examined, as follows. Table IV sets out the appropriate data and, on testing the null hypothesis that the two bivariate distributions are not discordant, we obtain a large sample χ^2 of 117.67 (d.f.=29, $P<0.001$) and the two distributions are thus significantly different. Partitioning this χ^2 value into its interaction components we find 10.43 (d.f.=4, $0.05>P>0.02$) ascribable to differences in parity, 9.50 (d.f.= 2, $P<0.01$) to differences in maternal age, 99.29 (d.f.=8, $P<0.001$) to the age/parity interaction, and a negligible component due to the second-order interaction. Though the differences are small SUD cases are on average of significantly higher parity and born to younger mothers; but inspection suggests that these are particularly marked in mothers under 30. These results parallel findings on infant mortality in Belfast for all categories other than congenital malformations (Elwood, 1969, pp. 96-7).

Factors Related to Birth and Pregnancy

Birth weight and gestation period

Most authors note a lower average birth weight or gestation period in SUD than

among surviving live births though not generally than among infants dying in the first year. These are found also in the present study, but when birthweight is more rigorously tested by the multivariate methods of discriminant function (Froggatt, 1970a) and multiple regression (Froggatt *et al.*, 1971) it appears less important. We conclude that birthweight *may* be a correlate with, but is not an important *determinant* of, SUD: as such it is likely to be merely a manifestation of general vulnerability of the premature since binary multiple regression shows birthweight to be a comparatively powerful determinant of all infant deaths (Elwood, 1969, ch. VI).

Multiple births

SUD is common among twins, equally so for mono- and dizygous pairs. When both partners are SUD cases their deaths have usually been coeval (Geertinger, 1968). In the present study 11 index cases were twins and two others were members of one like-sexed (female) but presumptive dizygous (by blood grouping) twin-set who died on the same day aged 10 weeks. Of the 12 sets, five were FF, four MM, and three MF; and the SUD twin was the heavier at birth in six. We argue elsewhere (Froggatt *et al.*, 1971) that this high representation of twins may be ascribable to their low birth weight (mean is 5.1 lb.) and the coeval death in twins (when both die) possibly interpretable in terms of a common environmental agent. We may also speculate that it is the direct effect on one twin by the death of his partner possibly mediated through shock or fear, factors known to cause sudden death (Engel, 1971).

Maternal health during pregnancy

During the pregnancies producing the 148 SUD cases with family interview data and the group A controls, respectively 27.7 and 23.6 per cent of mothers had conditions which could have endangered pregnancy, e.g., pyelitis, PET, APH, respectively 52.7 and 58.1 per cent alleged that their health throughout was good, while the remainder (19.6 and 18.3 per cent) had complaints either minor or conventionally unassociated with risk to the foetus. These differences are unimportant.

X-ray exposure

During the above pregnancies respectively 83.8 and 85.8 per cent of mothers had no pelvic or abdominal X-rays, and the number, and stage in pregnancy, of X-irradiation to the remainder were approximately equal.

Presentation and mode of delivery

Of the 148 SUD cases and group A controls, respectively 94.5 and 94.0 per cent were vertex presentations, most others being breech; while respectively 80.3 and 78.3 per cent were spontaneous deliveries, equal numbers (12) were induced, and 11 and 13 per cent respectively were delivered in other ways, about half being by Caesarian section.

Evidence of Specific Inheritance

A recessive Mendelian hypothesis would require sibship aggregation (within Mendelian expectations) and increased parental consanguinity, while an inherited autosomal anomaly would likely be recognisable on karyotyping and would produce

increased foetal loss. We show elsewhere (Froggatt *et al.*, 1971) that (i) the SUD case rate amongst sibs of singleton propositi is 11.0–22.0 per 1,000 live births, i.e. 4–7 times the population and control group A figures, but still far too weak an aggregation for a Mendelian interpretation, (ii) no parents of SUD cases admitted kinship, and (iii) there was no unusual foetal loss. (Tissue for karyograms was at the time difficult to culture: subsequently Weinberg and Purdy (1970) have shown some minor chromosomal abnormalities in SUD though of uncertain significance). From this, and autopsy evidence, we conclude that specific inheritance must play at most a small part in SUD.

Post-Natal Health and Environment

Post-natal weight increment

Drillen's (1964) work shows parallel population weight increment curves over the first year of life for each sex and all birth weights: thus Figure 2 validly compares weight increment of pooled SUD singletons with combined population series (Tanner, 1958; Watson and Lowrey, 1967). (Each SUD autopsy weight is 'corrected' to an estimate for exactly 1, 2, ... months, whichever is nearest, on the basis of Stuart and Meredith's tables (Watson and Lowrey, 1967)). The curve for SUD cases lies between the 10th and 50th percentile and, allowing for small-number instability at older ages, at a probably constant relationship to both. For twins, mean weight gain was 1.39 lb. per month which compares reasonably with Drillien's (1964, Appendix IIa) figures for weight increment in 'healthy' twins. These results suggest that SUD cases thrived normally.

Post-natal health

Arbitrarily dividing the period between birth and death (of an SUD case) or interview (of a control) into (i) from birth to one week before death (or interview) – which may be relevant to the child's underlying fitness, and (ii) the week preceding death (or interview) – which may be relevant to the terminal event, and dealing exclusively with illnesses for which medical attention was sought, we obtain the following experience for 148 SUD cases and group A controls.

During period (i), and omitting twins and premature baby unit admissions, 25 cases and 19 controls were admitted to hospital at least once. The principal cause of the most recent admission was: pneumonia, bronchitis, and other pulmonary conditions (ten cases, eight controls); failure to thrive (five cases, no controls); rhesus incompatability (two cases, two controls); gastro-intestinal conditions (four cases, four controls); and others (four cases, five controls). For other medical consultations and omitting twins, two cases with infantile ecezema, and the episodes leading to hospitalisation (as above), 62 (46.6 per cent) of the remaining SUD cases had at least one episode of illness compared to 57 (38.8 per cent) of controls. The episode rate (episodes per person sick) was approximately 1.3 for cases and 1.4 for controls, and 'upper respiratory infections' accounted for the majority (70 per cent among cases, 60 per cent among controls). There were no unusual diagnoses. These findings confirm the weight increment results (Fig. 2) indicating basic fitness.

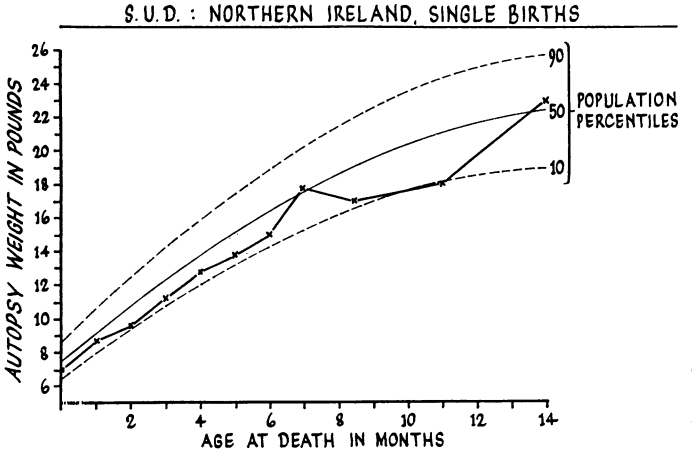

FIG. 2. *Mean weights at death of singleton SUD cases in the present study compared with population limits for mean weight of normal singletons at birth, 1, 2 . . . months of age. (See also text).*

During period (ii), seven SUD cases were discharged from hospital after treatment for pneumonia (three cases), otitis media (one case), negative investigation of failure to thrive (one case), breathless episodes (one case), and intermittent vomiting (one case). Six SUD cases were admitted *in extremis* and died. No controls were in hospital during the period. In addition, a doctor was consulted or was sought for 30 other SUD cases (other than those *in extremis*) compared with 15 controls ($\chi^2=5.61$, d.f.$=1$, $P<0.05$), the cause being respiratory infection (17 cases, 12 controls), gastro-intestinal conditions (10 cases, 3 controls), and general pyrexia, listlessness, and convulsions (one case each). Furthermore, an additional 42 SUD cases had been allegedly 'unwell' (mostly minor coryzal or digestive symptoms) – though medical advice was not sought – during this period compared to zero controls. Unless there was gross re-call bias the picture is of increased minor illness during the week, particularly the 24 hours, before death.

Feeding and medicaments

SUD cases have often seemingly differed from controls in feeding regimens, and breast feeding, specially during the first two weeks (to avoid sensitisation), has been advocated (Ministry of Health, 1965).

In the present study 18 SUD cases were wholly breast-fed for at least one week after birth, eight were partially breast fed from birth, and 121 were never breast-fed. Corresponding frequencies for matched controls were 22, six and 119 ($\chi^2=0.70$, d.f.$=2$, $0.98>P>0.95$). One further case and control had been wholly breast-fed for five and three days respectively. Two of the 18 cases were exclusively breast-fed throughout life, and among the remaining 16 the time-interval between the first introduction of other types of feeding and death ranged widely (1–38 weeks. Mean$=11.1$; median$=7$). The strengths and formulae of feeds were not dissimilar for cases and controls and in only five instances did the last feed contain a previously untried substance *viz.* Lucozade, glucose, Ostermilk 2, Farex, and a

cereal. Only three infants were known to die or collapse within one hour of feeding. Generally milk antibody titres were unremarkable (Froggatt *et al.*, 1971). These findings were not conclusive but they do not seemingly indicate an important numerical role in SUD for hypersensitivity to cow's milk protein.

Most infants are given medicines at some time. Among the SUD cases 10.8 per cent allegedly received none, 46.6 per cent allegedly received only household medicaments – aspirin, gripe water, nose drops, teething powder, etc. – while 42.6 per cent received other drugs usually antibiotics. Corresponding frequencies for group A controls – 10.1, 53.4, and 36.5 per cent – are not significantly dissimilar to these ($\chi^2=1.40$, d.f.$=2$, $0.50>P>0.30$).

Sleeping position

Suffocation by bed clothes and 'overlaying' were long accepted as major causes of sudden death in infants (Templeman, 1892; Froggatt *et al.*, 1968). Most authors now accept that suffocation by external agencies or by aspiration of gastric contents is unimportant, but some recommend discontinuance of soft pillows (e.g., Ministry of Health, 1965).

Though we cannot interpret the relevance of the results, we record that there were significant differences between SUD cases and group A controls in their 'normal' sleeping position, i.e., the position in which the mother allegedly 'usually' finds the child (Froggatt, 1970a), but in the 111 cases found unequivocally dead there was no difference, in as many as 68 per cent, between 'normal' sleeping position and position found. Where there was a difference the terminal position was: face down (nine); face up (eight); face to side (five) – i.e. there is no evidence of an excessive 'face down' position. Among 144 cases, 55.4 per cent slept without a pillow (25.3 per cent with neither bedding nor pillow) and only 17.6 per cent of those found dead or *in extremis* were face down on a pillow or with nose and/or mouth covered by bedding. Again there is no strong evidence for smothering. Other terminal findings are described by Marshall (1970).

Discussion

These results provide a pathological, epidemiological, and clinical profile of SUD. They have, however, only limited value in specifically assessing current hypotheses of causation though we can adduce some evidence on the coherence of certain general theories.

We can confirm the consensus view (Bergman *et al.*, 1970) that SUD victims do not have some underlying 'disease' as yet unrecognised: the unrevealing special autopsies, the lack of evidence for the segregation of abnormal genes, the satisfactory thriving and absence of significant clinical findings (other than terminally), and the narrow age range all argue against it. Cases appear in fact to be essentially 'normal' infants, and the social and biological factors correlating with SUD can be shown to have very poor predictive power (Froggatt, 1970a). The characteristic age range is the most important factor and there seems little doubt that these infants die during a period of increased physiological vulnerability because some critical combination of intrinsic and extrinsic factors proves lethal: what *is* in doubt is the mechanism, or 'final common pathway', of death.

Our results must ascribe some role to infection, mainly respiratory infection. Greatest incidence is in Belfast among the lowest socio-economic groups and the most crowded houses, in the coldest months, with serial correlation between SUD and major virus epidemics, and with 'season'/'city' contingency – cases in Belfast in the winter being disproportionately prevalent; a history of minor symptoms, usually coryzal or respiratory, in the week and notably the 48 hours pre-mortem is common; there is increasing relative incidence with increasing parity, at least in mothers under 30 years of age; the clinical picture is of seeming respiratory infection in those few cases seen *in extremis* by a doctor; when both twin partners are SUD, death is simultaneous; and the autopsy findings, though conventionally accepted as 'normal' (except perhaps for the petechiae – see below), usually show in common with other studies (Valdés-Dapena, 1967; Beckwith, 1970) an increased cellularity of the lungs sometimes with cellular infiltration of peri-bronchial tissue (Marshall, 1970). Furthermore, virus and bacteria isolations, unremarkable in our study (Marshall, 1970; Froggatt, 1970b), have been considered relevant in others (see Bergman *et al.*, 1970). As against this is the failure to demonstrate space-time clustering – though the tests will not always detect epidemicity especially if, as seems likely, there be several partly independent infective agents (Ray, Beckwith, Hebestreit and Bergman, 1970) – and the fact that some of the above findings could be expected on other hypotheses (Froggatt *et al.*, 1971).

The precise role played by any such infection is equivocal. It may be primary *viz.* in overwhelming the infant during an innate vulnerable immunological state, in sensitizing (by a first challenge) then overwhelming (in a second challenge) the now sensitized child, in inducing lethal pulmonary reflexes or occlusive laryngospasm, or by other mechanisms (Valdés-Dapena, 1967; Froggatt *et al.*, 1968; Dawes, 1968; Bergman *et al.*, 1970; Ray *et al.*, 1970); or it may be secondary, increasing the child's susceptibility to some other, perhaps unrelated, lethal process e.g. cardiac conduction disturbance, or making such a process itself more likely (e.g. James, 1968). Whatever the mechanism even quite trivial respiratory infections may seemingly predispose to SUD in a child aged 1–6 months.

Apart from findings in the conduction system of the heart (James, 1968) the only significant autopsy finding is the presence of numerous petechiae on the lungs, heart and thymus. They occurred on one or more of these organs in two-thirds of cases. These haemorrhages, formerly interpreted as 'asphyxial', were largely responsible in the past for the diagnosis of suffocation and recently they have been interpreted as evidence of terminal laryngospasm by Beckwith (1970) who argues that the strict intrathoracic distribution of the petechiae provides compelling evidence of markedly elevated intrathroracic negative pressure due to a terminal episode of high airway obstruction. We are reluctant, however, to accept this argument; petechial haemorrhages occur in many kinds of death in adults and whatever the cause of death, they favour certain sites of which the lungs and heart are prominent; and they are not particularly numerous in children suffocated by a plastic bag over the head or by a foreign body in the larynx, when elevated intrathoracic negative pressures ought also to operate. Whilst we are unwilling to ascribe any diagnostic significance to intrathoracic petechiae, we recognise that they are found more regularly and more conspicuously in SUD than in other infant deaths and that interest in them should be maintained.

Sleep seems also to have significance. It is impossible to be certain that an unobserved infant is asleep, but the characteristic distribution of cases over the 24 hours – unless time-of-day is itself a factor in SUD – and the rarity of the terminal event while the child is being handled, are suggestive. This does not help towards establishing the 'final common pathway' of death: bodily changes during sleep are theorised to increase the likelihood of SUD by, specifically, laryngospasm (Bergman *et al.*, 1970, p. 210), cardiac conduction disturbance (James, 1968), respiratory centre failure (Guntheroth, 1970), and lethal cardiopulmonary reflexes (Steinschneider, 1970).

The age distribution is characteristic and is the main cornerstone of the concept of developmental physiological vulnerability in SUD. This concept receives support from the case histories and family data. One aspect should be emphasised: the occasional history of previous 'fainting', cyanotic or apnoeic episodes, or periodic breathing, perhaps especially in multi-case sibships (Froggatt *et al.*, 1971). Such a history could be elicited in a few of our cases (only twice did it lead to hospital investigation – which was negative) and has also been documented by e.g. Stevens (1965), and Steele (Bergman *et al.*, 1970, p. 74). It may in fact be even more common since such episodes may easily go unnoticed (or unrecorded) in the very young infant. Episodes of this type occur in infants generally and for variously interpreted causes (James, 1968; Steinschneider, 1970; Guntheroth, 1970); if they be more common in SUD victims than others then they should be considered a sinister symptom requiring investigation of the child. They may on the other hand indicate causal heterogeneity: if so the mechanism of SUD in such infants may be different to that in others.

Undoubtedly the outstanding problem is to identify the 'final common pathway' of death; a subsidiary one is to distinguish sub-types in the entity (or entities) SUD. Meanwhile SUD will remain a disease of theories with investigators continuing to favour explanations in their own specialty. Thus virologists see the importance of respiratory viruses, immunologists the dangers of immature immune mechanisms, cardiologists the hazards of the unstable infantile conduction system or myocardial electrolyte disturbance, and cardiopulmonary physiologists the role of the baby's labile autonomic system. Tests which may, for example, discriminate 'pulmonary' from 'cardiac' death e.g. terminal blood-gas composition (Mithoefer, Mead, Hughes, Iliff and Campbell, 1967) unfortunately seem to be equivocal in interpretation (Bergman *et al.*, 1970, pp. 130-2). Nevertheless experimental and basic sciences offer now the best avenues to advance knowledge. Case reduction would at present seem to depend upon general realisation of the vulnerability of the infant between one and six months – especially if he be an underweight twin or a child with respiratory symptoms – and the possibility that the crucial episode is not irreversible and that a proportion of infants could be saved by cardiopulmonary resuscitation. Such methods may not need to be by machine; simple handling and first-aid techniques may arrest or reverse the process long enough to allow hospitalisation. Prevention of known environmental trigger mechanisms e.g. respiratory infection, would have only slight effect, but round-the-clock surveillance (or monitoring) of *all* infants would certainly reduce case incidence and could elucidate causation; in view of the infrequency of SUD and the weak predictive power of the currently recognised 'at risk' factors (Froggatt, 1970a), anything short of this would have a

numerically small effect. Such a procedure is impracticable, but further studies may disclose factors which would allow surer identification of 'at risk' children and so make such a surveillance scheme feasible. Anyone working for reduction in the incidence of SUD must hope that this will prove to be the case.

Summary

This article describes a collaborative study of sudden unexpected death in infants ('cot death') in Northern Ireland based on the 162 cases ascertained through the Northern Ireland Forensic Pathology Service from 1st August 1965, to 31st July 1967. These represent some 90 per cent of an estimated complete ascertainment, and clinical, epidemiological, and pathological data are presented on them. After full discussion of theories and interpretation of the data it is concluded that 'cot death' victims are essentially healthy throughout life; they die because during a developmental stage of physiological vulnerability – mainly between one and six months – some combination of intrinsic and extrinsic factors proves fatal. Despite intensive research the 'final common pathway' of death is still unknown though social and biological risk factors have been identified. Basic scientific and experimental studies now offer the best opportunities for further work.

Acknowledgments

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INDUCTION OF PROGRESSIVE NEPHROPATHY IN RATS BY ELUTED γ-GLOBULIN AND FREUND'S ADJUVANT

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INTRODUCTION

IT has been suggested that anti-basement membrane (BM) antibody is responsible for the development of the pulmonary and the renal lesions in Goodpasture's syndrome (Sturgill and Westervelt, 1965; Beirne, et al, 1968; Willoughby and Dixon, 1970). It is a controversial issue whether this anti-BM antibody is formed by the release of glomerular or alveolar BM antigen.

Experimental models resembling Goodpasture's syndrome have been produced in animals by the injections of anti-lung antiserum (Hogadorn et al, 1969; Willoughby and Dixon, 1970). It has also been reported that heterologous non-organ specific antigen (lung homogenate) can initiate an auto-immune type of glomerulonephritis (Stebley and Rudofsky, 1968).

The pathogenic role of the anti-glomerular basement membrane (GBM) antibodies in the production of glomerular lesion in Goodpasture's has also been demonstrated by a successful transfer of acute glomerulonephritis to monkeys by the intravenous injection of eluted γ-globulin from patients with Goodpasture's syndrome (Lerner et al, 1967; Lerner and Dixon, 1968b). Poskitt (1970) demonstrated the presence of γ-globulin in the GBM of mice 24 hours after the injection of a similarly eluted γ-globulin but he failed to produce a progressive glomerular lesion. Nagi et al (1971) induced progressive glomerular and pulmonary lesions in rats which were made proteinuric prior to the intravenous administration of eluted γ-globulin from a case of Goodpasture's syndrome .They concluded that serum sickness plays an important part by allowing the injected γ-globulin to come in contact with some component of the GBM.

The present experiment describes an attempt to induce progressive glomerulonephritis in Wistar rats by intraperitoneal injections of Freund's complete adjuvant and eluted γ-globulin from the kidney of a case of Goodpasture's syndrome .

MATERIALS AND METHODS

The γ-globulin was eluted from the kidney of a case of Goodpasture's syndrome with a method used by Nagi et al (1971).

Thirty-two Wistar rats were divided into the following three groups. Group A – In this group 20 rats were injected by intraperitoneal route with a homogenate of 0.2 ml of the eluted γ-globulin and 0.4 ml of Freund's complete adjuvant per rat for 6 weeks. Group B – In this group 6 rats were injected with 0.2 ml of the eluted γ-globulin in normal saline. Group C – In this group 6 rats were injected with Freund's adjuvant alone. The protein estimations were carried out on the 24 hour specimens of urine at weekly intervals till the termination of the experiment at 18 weeks. Kidney and lung tissues obtained from each rat were examined by histological, immunofluorescent and electronmicroscopical techniques.

Results

During the course of the experiments the proteinuria in the control groups (B and C) of animals was within normal limits, whereas in the test group it showed continuously rising levels (Figure 1).

The results of immunofluorescent studies are summarised in Table I. It is evident that no control animals had the glomerular localisation of human or rat globulins. On the other hand, 16 of the 20 rats in Group A showed linear localisation of autologous γ-globulin and complement along the GBM and alveolar capillary BM.

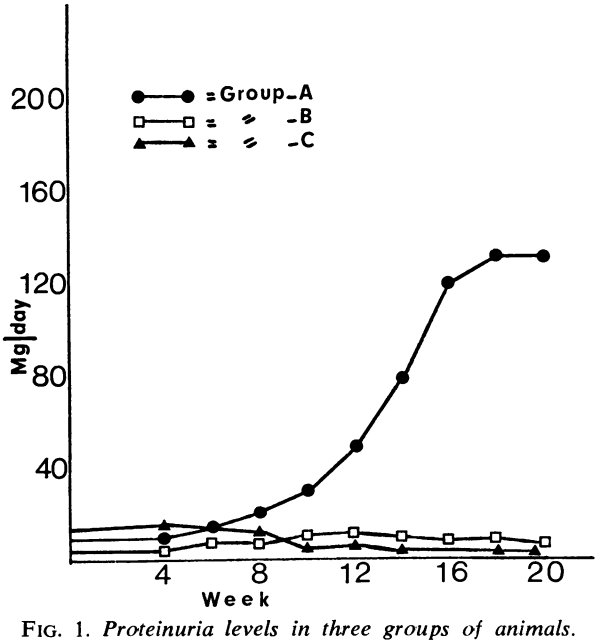

FIG. 1. *Proteinuria levels in three groups of animals.*

In the kidneys of Group A animals moderate to severe histological abnormalities were observed by the various staining techniques. From 50 to 75 per cent. of the glomeruli in this group showed proliferation of endothelial and mesangial cell. Most of the glomeruli showed this change in 2 to 3 lobules whereas the remainder of the lobules appeared normal. The capillary lumens in many glomeruli are filled with PAS

TABLE

Immunofluorescent Studies

Group	*Rat IgG*	*Rat BiC*	*Human IgG*	*Human BiC*	*No. of Animals with lesions*
A +	–ve	4/20			
++	6/20	12/20	None	None	16/20
+++	9/20	—			
++++	1/20				
B +					
++	None	None	None	None	0/6
+++					
++++					
C +					
++	None	None	None	None	0/6
+++					
++++					

positive material which also stained for fibrin. The proximal convulted tubules around the affected glomeruli were dilated and contained proteinaceous casts in their lumens (Figure 2). By electron microscopy the glomerular lesions were recognised by irregularly thickened BM and proliferation of the BM-like material towards the the endothelial aspect. The lumens in some of the capillary loops were obliterated with this BM-like material. Most of the affected loops showed subendothelial electron dense deposits (Figures 3 and 4). The epithelial cell foot processes were fused over the affected loops. The tubles contained numerous protein droplets within their epithelial cells.

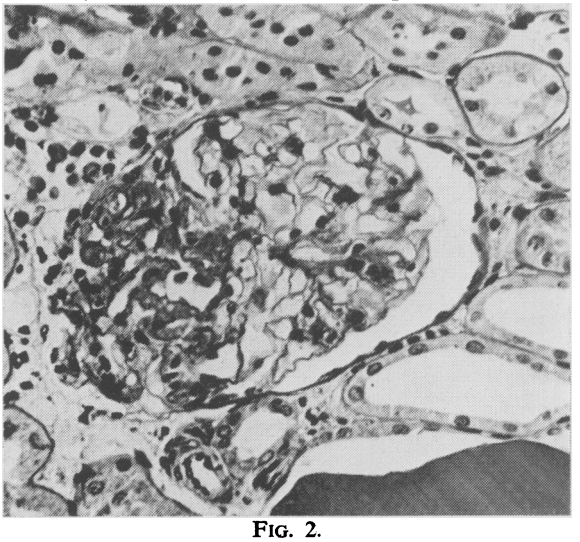

FIG. 2.
Cellular proliferation and other changes described in the text can be seen in part of the glomerulus.

Lungs: All rats in Group–A showed moderate degree of lymphocytic infiltration of the inter-alveolar speta with some thickening of the alveolar walls. A few deposits of haemosiderin were also seen in most of the specimens.

The control groups B and C showed none of the histological, electron or immunofluorescent microscopical abnormalities.

DISCUSSION

The results of the present experiment show that eluted γ-globulin from the kidney of a patient with Goodpasture's syndrome can initiate a disease recognised by progressive lesions of lungs and kidneys. It is also evident that these lesions were consistenly produced in Group–A rats which were injected with a homogenate of the eluted γ-globulin and Freund's adjuvant. Fluorescent antibody test revealed the localisation of autologous γ-globulin and complement in a focal linear distribution along the glomerular and the alveolar capillary BM. On histological and electron microscopical examination these lesions were similar to those described by Nagi et al (1971).

The pathogenesis of Goodpasture's syndrome is still a controversial issue. Various experimental models suggest that the lesions might be initiated by antibodies directed against both lungs and kidneys or anti-GBM antibodies which cross-react with alveolar capillary BM. (Stebley and Rudofsky, 1968, Hagadorn et al, 1969; Willoughby and Dixon, 1970). Lerner and Dixon (1968a) put forward the possibility that certain "undefined associated factors must be present before the GBM-antigen can induce glomerulonephritis". Nagi et al (1971) suggest that these undefined factors could be serum sickness induced changes in the GBM permeability and the presence of an excessive amount of anti-GBM antibody.

The actual mechanism by which the present experimental model is produced is not known. We put forward a hypothesis which may elucidate the events leading to the production of this disease. The vulnerability of the GBM is increased by the administration of Freund's adjuvant (Heymann et al, 1962 and 1963; Watson,

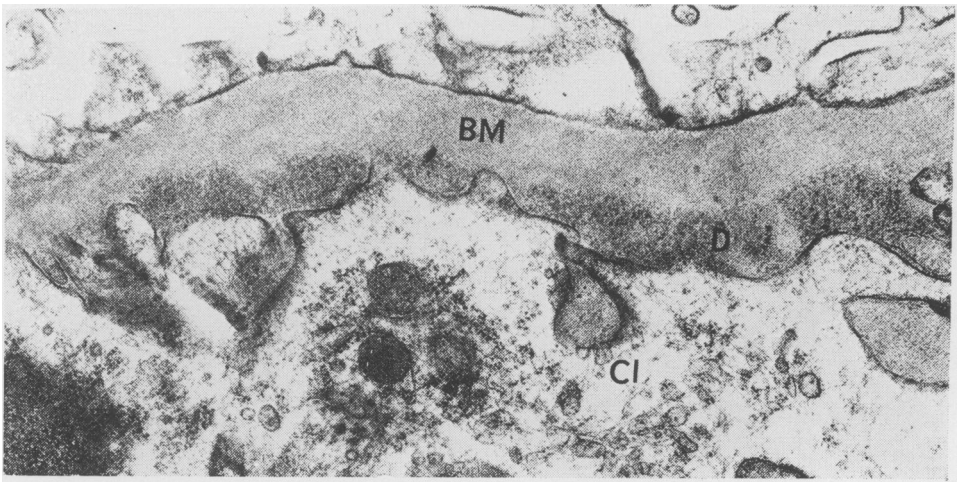

FIG. 3 (*above*)
Osmiophilic deposit along the sub-endothelial aspect of the thickened glomerular basement membrane in a group-A rat. Electron micrograph.

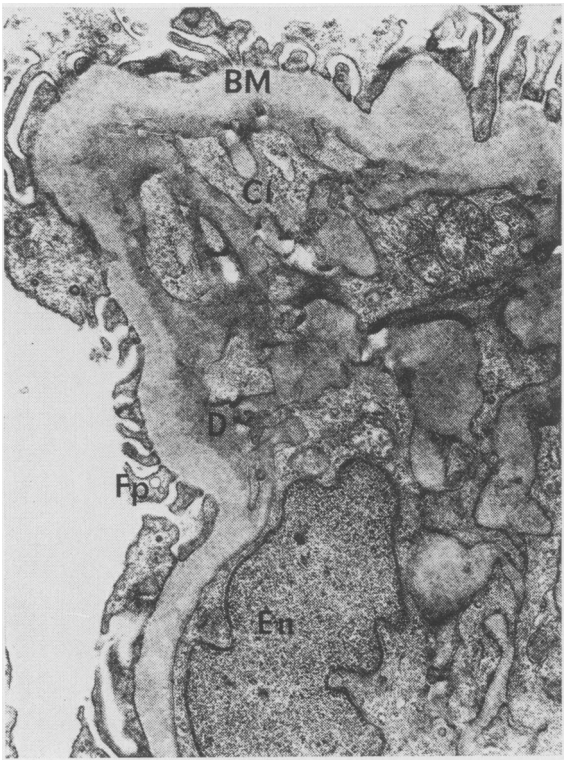

FIG. 4. (*left*).
Another glomerular loop showing marked proliferation of the GBM, resulting in the obliteration of its lumen.
BM = Basement mebrane
En = Endothelial cells
D = deposit
Fp = Foot processes
Cl = Capillary lumen

Dixon and Feldman, 1965; Cuppage, 1965) and by serum sickness (Barabas et al, 1970; Nagi et al, 1971). Freund's adjuvant containing eluted γ-globulin might have come into contact with the nephritogenic material in the GBM leading to its intravascular release and the production of autoantibodies. These autoantibodies might have started a cycle of events which maintained the release of the GBM antigens and the formation of autoantibodies which might result in the type of tissue damage observed in the lungs and kidneys of these rats. Autoantibodies formed against the GBM might have cross-reacted with the alveolar capillary BM resulting in the dual lesions in this disease. In view of this observation it is suggested that in addition to serum sickness certain factors (Freund's adjuvant in this case) which increase the vulnerability of the BM may also help in initiating an event responsible for the production of a progressive disease of this nature.

Summary

A progressive glomerulonephritis and pulmonary lesion have been produced in Wistar rats using a homogenate of eluted γ-globulin from a case of Goodpasture's syndrome, and Freund's complete adjuvant. It is suggested that the Freund's adjuvant—induced changes in the vulnerability of the GBM may contribute to the initiation of an autoimmune cycle which may lead to the production of a progressive disease of the BM.

My thanks are due to Professor Sir J .H. Biggart, C.B.E., for his advice, Messrs. W. Kirkwood, P. Westwood and S. Cameron for their technical help, and Mr. D. Mahaffy for the photography.

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BEHAVIOURAL SHAPING TECHNIQUES IN THE TREATMENT OF CONVERSION HYSTERIA*

by

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IT IS usual nowadays to regard hysteria as a psychiatric disorder, rather than due to movement of the womb or to any specific disturbance of brain function. Despite this it is probable that as many cases of frank conversion hysteria are seen in an average neurological practice as in psychiatric practice; this is the excuse for inflicting a paper on a psychiatric topic upon the Irish Neurological Association.

The treatment of hysteria falls into two broad categories: the removal of the symptoms and the sorting out of the underlying disorders in the patient and his environment which made the symptoms necessary. We should consider whether treatment directed merely at removing the symptoms has any important part to play in the management of hysterical patients. There are four possible reasons why symptom removal is worthwhile in some cases.

First of all, secondary physical disorder may occur if hysterical symptoms continue for many months; I have seen severe flexion contractures in a patient who had an hysterical paralysis of one arm, and marked disuse atrophy of the leg muscles occurred in one of the cases to be described later. Secondly, it may be that the sorting out of the underlying reasons why the hysterical symptoms were necessary would take a long time, but can be appropriately undertaken as an outpatient; symptom-removal may enable the patient to live at home and have outpatient treatment. Thirdly, it may be that some patients can only start to tackle the sorting out of the real troubles when the symptoms behind which they were hiding have been removed. Finally, it sometimes happens that the patient's symptom persists when it is no longer psychologically needed; the patient cannot merely relinquish the symptom, but has to have some "treatment" to give him an adequate reason for abandoning it.

The history of medicine, both orthodox and unorthodox, contains many examples of techniques for "magicking away" hysterical symptoms. We can think of witch doctors, the application of foul smelling substances, faith healers, places or objects of veneration, suggestion with or without drugs and suggestion under hypnosis. One drawback of a technique such as symptom-removal under hypnosis is that it is an "all-or-nothing" effort; if you succeed you have taken the patient's symptoms from him, giving him no time to adjust to the new state; if you fail to remove the symptoms you have shown both the patient and yourself that you are a failure as a therapist. I remember Dr. Denis Brinton, at the National Hospital, Queen Square, warning his pupils against instant cures of hysteria; he knew of a patient

*Based on a paper delivered to the Annual Meeting of the Irish Neurological Association, Belfast, November, 1970.

who was "cured" in the morning and who committed suicide in the evening. This paper describes a technique of symptom-removal which is based on some operant conditioning principles, which seems to be effective in a reasonably short time, but which does not have the "all-or-nothing" quality and therefore the patient can gradually adapt to his improved state.

It is necessary to explain what is meant by "shaping" of behaviour. Let us take a very simple example in which we wish to train a rat in an operant conditioning experiment to press a lever in his cage in order to obtain reinforcement in the form of pellets of food. The difference between operant conditioning and classical conditioning is that in operant conditioning the subject has to emit the behaviour which will lead to the reinforcement, whereas in classical conditioning (such as with Pavlov's dogs) the experimenter both rings the bell and presents the food to make the dog salivate – the dog is the passive recipient of the experimental procedure. One way of training a rat to press the lever in the corner of his cage would be to sit around for hours and days as he explores his cage, waiting for the odd occasion on which he presses the lever by chance and immediately to give the reinforcement. This is extremely time consuming and inefficient. If behavioural shaping is used, at the beginning of the experiment the food reinforcement is delivered whenever the rat moves towards the wall of the cage on which the lever is mounted. This results in a change in behaviour, the rat spending an increasing amount of his time at that end of the cage. The "rules" are then tightened up so that only if the animal rears up on his hind legs at the correct end of the cage does he get the reinforcement. Later the animal must rear up and touch the wall on which the lever is mounted with his fore-paws, and finally he must actually press the lever to obtain the reinforcement. This shaping technique produces consistent bar-pressing behaviour in a much shorter time than if the shaping had been omitted.

Operant techniques including the use of shaping have been used in a number of clinical situations. They have been widely used to improve the social and verbal behaviour of autistic children (Lovaas, 1966; Martin et al., 1968). In some situations the operant technique has involved the establishment of a "token economy". A "token economy" refers to a situation in which the patient is rewarded for increasingly adaptive behaviour by being given "tokens". He can then use or "spend' these tokens to obtain privileges which are individually selected activities which the patient himself would wish to do. The "price" of a given privilege can be varied from patient to patient according to the therapists' judgment of which privileges are most highly valued by the patient. Token economy programmes have been employed in both mental subnormality and chronic schizophrenia (Ayllon and Azrin, 1968). Bachrach, Erwin and Mohr (1965) reported the treatment of a patient with anorexia nervosa by an operant conditioning programme in which privileges were used as positive reinforcers for eating behaviour.

There seem to have been few attempts to use operant conditioning in conversion hysteria. Brady and Lind (1961) reported the investigation, rather than the treatment, of a patient with hysterical blindness by an operant programme. In their book Meyer and Chesser (1970) point out that the procedure of Brady and Lind, and also that of Hilgard and Marquis (1940) in treating a patient with hysterical

anaesthesia and paralysis, were directed mainly at making the patient consciously aware of perception. They do not refer to any behavioural shaping techniques used to remove directly hysterical motor or speech disorders.

For brevity, the two patients will be described without full clinical details; the main emphasis will be on the behavioural shaping techniques used to modify the symptoms.

Case Reports

Case 1. A 43 year old housewife was admitted virtually in stupor: she lay in bed with little or no movement of the body although she did move her neck and eyes; she was mute. She later professed by the use of nodding and head shaking that she also had considerable impairment of vision and hearing. The immobility responded within a few days to support and encouragement from the nurses; at first they got her up into a chair and later helped her to walk. She remained mute, and it was therefore decided to use a shaping technique to treat her speech disorder; ordinary social reinforcement by praise was used as the reward. Initially her personal doctor and all the nurses and occupational therapists were instructed that even if she tried to mouth the shape of words she was to be praised (e.g., "Good; Well done; That's excellent, etc."). As soon as she was mouthing words regularly, conditions were tightened up and the staff instructed to frown in a puzzled way unless she produced a little phonation, even if only a faint whisper. As time went on only a loud whisper was reinforced; conditions were then tightened further so that the staff looked puzzled and irritated if she whispered but gave the reinforcement if she produced a little vocal phonation. Speech and walking were fully normal in about nine days and she was then able to participate in the occupational programme and to talk with normal volume in psychotherapeutic interviews. It is a daunting prospect for a therapist to have to undertake lengthy psychotherapeutic interviews with a patient who only whispers. This still remains a very complicated case; during the follow-up period other difficulties occurred which had to be tackled, but there has been no recurrence of immobility; the only incident of mutism was in acute stress and resolved within a few hours.

Case 2. The patient was a 23 year old garage mechanic who had developed a total paraplegia some 9 months after an operation for removal of a subarachnoid cyst in the lower lumbar region. Despite his past history, it became clear to the neurologists and neurosurgeons who examined him that his paraplegia was hysterical. When he was admitted to the Department of Mental Health he was not only paraplegic but also had strange sensory loss between the knee and the ankle, but not affecting the foot; he complained of severe pain and tenderness from the coccyx to the occiput. He was treated with extensive psychotherapeutic exploration of various problems in his life, coupled with physiotherapy, encouragement from the nurses and occupational therapy programmes. Psychiatric investigation had produced ample evidence of severe personality disturbance, dating back to his mid-teens. After six months treatment, the only progress had been the loss of complaints of pain and tenderness, but he was still paraplegic and living in a wheel chair. It was decided to initiate a token economy in which he would have to earn any of the pleasures of life by motor activity in the lower limbs.

His "base-state", when he had done no exercises during the day, consisted of a single room with no papers to read, no cigarettes to smoke, and no use of the

telephone. He was not permitted visitors, and the ban on visitors included visits by fellow patients; he was not allowed home for week-ends. In order to "buy" any of the pleasures in life he had to earn "points" by performing certain required exercises.

A detailed list of exercises was drawn up for each side, starting with dorsi-flexion and plantar flexion of the toes, moving up to the same movements in the foot and listing 17 exercises on each side. The performance on each exercise was graded from 0 to 4 as follows:

0 – merely a flicker of movement.
1 – movement against gravity.
3 – movement against slight resistance.
4 – movement against strong resistance.

The patient was given three sessions each day in which he could earn "points"; a point was awarded for reaching a new grade in any of the 34 exercises. He could then "spend" points to visit other patients, to have cigarettes, to use the telephone, and so forth.

When the token economy programme was started, he had been completely paraplegic for over six months in our unit, as well as for many weeks prior to transfer to our department. Within a month, he was able to stand unaided and had regained sensation in his feet and legs. After two months he was able to walk 50 yards in one minute unaided. (In the course of the behavioural shaping the element of speed was introduced in order to increase the stringency of the requirements for earning points; speed was also included in the hope of improving his gait, which was very abnormal even though he was able to walk reasonably quickly). At the end of the first two months he was also able to dance at socials in the unit. He was discharged from hospital after four months on the programme able to walk quickly, but still with a very abnormal gait. It was intended that he should attend three times weekly as a day patient to continue the programme, but he lapsed from attendance and refused to return to the unit. He has since been seen by other specialists (both a neurologist and a psychiatrist) but has refused re-admission for further treatment.

Discussion

The question arises whether this technique really has any advantages over the general measures commonly used in medical and neurological wards. Shaping techniques can be readily applied in a general ward, and in no way require psychiatrists or psychologists. In addition to the two patients quoted I have advised on a shaping technique in a general ward for the management of an hysterical hemiparesis and the technique was used with some success. The main difference between the encouragement and suggestion given in the usual management of these cases (often with physiotherapists participating in the treatment) is the systematic reinforcement only of behaviour which is progressing in the right direction. The average kindly physiotherapist encourages and praises the patient when he is doing well, but also feels that it is her duty to encourage him even if he does not seem to be making much headway. One of the factors leading us to reconsider the management of the paraplegic patient was the despair of the physiotherapists who had been treating him for months in order to prevent contractures. With the

shaping technique a systematic plan was made to reinforce small progress to begin with but later only to praise further progress from that position, and not to praise static behaviour.

It must be emphasized that the shaping technique is only a technique for symptom-removal, and is not a treatment for hysterical disorder as a whole. The recurrence of similar or other symptoms in cases of hysteria will only be prevented if the underlying reasons why the symptoms were necessary have been properly dealt with. It is clear that this aspect of the treatment of both patients described was far from fully successful. This poor outcome reflects the poor prognosis in the individual cases because of the underlying disorder of personality, and does not reflect any inefficiency in the shaping technique. A better outcome might well have been expected in the second case had behavioural shaping been instituted much earlier in his treatment.

Summary

1. The value and limitations of symptom-removal in conversion hysteria are discussed.
2. The meaning of "behavioural shaping" is briefly illustrated.
3. The use of shaping techniques in the management of conversion symptoms in two patients is described.

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COXSACKIE A9 VIRUS OUTBREAK IN NORTHERN IRELAND DURING 1970

by

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THE LARGEST outbreak of Coxsackie A9 virus infection yet experienced in Northern Ireland took place during 1970 and a disproportionately greater number of cases occurred in Northern Ireland compared with the rest of the United Kingdom (British Medical Journal, 1970a).

Materials and Methods

Faeces, CSF, throat swabs and acute and convalescent sera were obtained from aseptic meningitis cases, while faeces and/or throat swabs were obtained from other cases.

Primary rhesus monkey kidney cell cultures were used for virus isolation and isolated viruses were typed with Coxsackie A9 neutralizing serum supplied by the Standards Laboratory, Central Public Health Laboratory, London.

Acute and convalescent sera from aseptic meningitis cases from whom virus had not been isolated and who were negative when screened serologically in the complement fixation test against mumps, measles, herpes simplex, lymphocytic choriomeningitis and louping ill viral antigens were tested for Coxsackie A9 virus neutralizing antibody. Sera were inactivated at 56°C. for 30 minutes then serial dilutions were mixed with equal volumes of 200 TCD_{50} of Coxsackie A9 virus which had been isolated from the CSF of a patient in the current outbreak. The virus-serum mixtures were left for 2 hours at 37°C. before inoculation.

Results

In Northern Ireland during 1970, 123 patients had Coxsackie A9 virus infection diagnosed in the laboratory. Coxsackie A9 virus was isolated from 119 patients. This virus was isolated from the CSF of 54 patients (51 per cent) with aseptic meningitis. Coxsackie A9 virus was also isolated from the throat of 60 patients and from the faeces of 93 patients. It was observed that Coxsackie A9 virus was isolated more quickly from the CSF or throat swab than from faeces obtained from the same patient. Serological tests on 40 additional patients with aseptic meningitis showed that four had an eightfold or greater rise in Coxsackie A9 neutralizing antibody which indicated recent infection while 28 had equivalent titres in their acute and convalescent sera which indicated past infection with this virus.

The number of patients with Coxsackie A9 virus infection and the month of onset of their illness are shown in Table I.

TABLE I

Mar.	*Apr.*	*May*	*June*	*July*	*Aug.*	*Sept.*	*Oct.*	*Nov.*
1	2	28	44	23	19	3	2	1

The outbreak began in March and ended in November while 114 cases (93 per cent) occurred between May and August with a peak incidence during June. One hundred and six patients (86 per cent) lived in Belfast or in surrounding suburbs and towns within a ten mile radius, an area which has a population of approximately 500,000. The clinical attack rate in that area was 21 per 100,000 population. There were 17 patients (14 per cent) outside this area. These patients were scattered widely in Co. Antrim (1), Co. Down (1), Co. Armagh (5), Co. Tyrone (4) and Co. Londonderry (6). The clinical attack rate for Northern Ireland as a whole was 8 per 100,000 population.

The illnesses associated with the Coxsackie A9 infections and the age and sex of the patients are shown in Table II.

TABLE II

Illnesses, Age and Sex of Patients with Coxsackie A9 Virus Infections 1970

Illness	*Age in years*						*Sex*		*Total*	
	<1	1-4	5-9	10-14	15-19	20+	*Male*	*Female*	*Number*	*Per cent*
Aseptic Meningitis	1	14	37	22	8	24	73	33	106	86.2
Respiratory	—	3	2	1	—	1	3	4	7	5.7
P.U.O. & Myalgia	2	2	2	—	—	—	4	2	6	4.9
Gastro-intestinal	1	—	1	—	—	—	2	—	2	1.6
Probable excreters	—	2	—	—	—	—	1	1	2	1.6
All clinical categories	4	21	42	23	8	25	83	40	123	100.0

Aseptic meningitis was the predominant illness associated with Coxsackie A9 virus infection in Northern Ireland accounting for 86 per cent of all cases. Eight patients had a rash associated with their illness which included four patients with aseptic meningitis, two patients with P.U.O. and one patient each with vomiting and pharyngitis. In addition one other child had a measles rash in association with his Coxsackie A9 infection. The respiratory cases included five patients with pharyngitis, one patient with pleurodynia and another patient with bronchitis and cough. One 3 year old boy had such severe myalgia in his leg muscles that he had difficulty in walking. The probable excreters included a child with an acute onset haemolytic anaemia and another child who was admitted as a "feeding problem". There were no deaths associated with Coxsackie A9 virus infections during 1970.

The outbreak was confined mainly to children with over half the cases occurring in those under the age of 10 years. The youngest patient was 2 months old and the oldest was 36 years old. In the aseptic meningitis group there were over twice as many males as females affected and the 5–9 year old age group was affected most. The youngest patient with aseptic meningitis was a boy of 9 months and the oldest was a woman of 36 years. Two boys had double viral infections confirmed virologically associated with their aseptic meningitis; one had measles virus infection and the other had mumps virus infection in association with their Coxsackie A9 infections.

There were 8 families where two or more members developed aseptic meningitis associated with Coxsackie A9 virus infection as shown in Table III.

TABLE III

Family Outbreaks of Coxsackie A9 Virus Aseptic Meningitis 1970

Family No.	*Date of onset*	*Age in years*	*Sex*	*Town*
1	1 May	4	F	
	6 May	3	F	Belfast
2	10 May	11	M	
	22 May	7	F	Belfast
	13 June	5	M	
3	13 June	11	M	Hillsborough,
	19 June	21	F	Co. Down
	10 July†	36	F	
4	17 July*	8	F	Belfast
	18 July	8	M	
	19 July	7	F	
5	7 August	12	M	Limavady,
	18 August	6	F	Co. Londonderry
6	12 August	9	M	
	13 August	10	F	Londonderry
7	24 August	11	M	
	29 August	8	M	Belfast
8	29 September	8	M	
	29 September	9	F	Armagh

†Mother of the two affected children.

*Not a member of family 4 but lived in same street and played frequently in their house.

Two children in each of six families developed aseptic meningitis, while in family number 3, three persons developed aseptic meningitis. In family number 4, the mother and a neighbouring child also developed aseptic meningitis. In families number 3 and 8, two children became ill on the same day which may indicate that both children became infected at the same time from an unknown third person, while in family number 6 there was only one day between the onset of their illnesses. The interval between the onset of illness in the two children in the other families was 5–12 days.

DISCUSSION

The most common Coxsackie A type identified in diagnostic virus laboratories in the United Kingdom is Coxsackie A9 and the prevalence was particularly high in 1963–4, 1966–7 and 1969–70 (British Medical Journal, 1970b).

The Coxsackie A9 outbreak in Northern Ireland was typical of enterovirus infections in that it had a peak incidence during the summer months and over half the cases were in children under 10 years old. Although 220 patients with Coxsackie

A9 infections were reported from the United Kingdom during 1969, only 3 patients came from Northern Ireland, the last case being in October, 1969; an interval of 5 months before the onset of the present outbreak. A three month old boy with hyperpyrexia and convulsions died in February 1969 and Coxsackie A9 virus was isolated from his liver and gut at post mortem, but there were no deaths associated with Coxsackie A9 infection during 1970 in Northern Ireland.

There were 259 Coxsackie A9 infections reported in the United Kingdom during 1970 (Private communication, Public Health Laboratory Service) and about half of these came from Northern Ireland. The clinical attack rate for Northern Ireland as a whole was 8 per 100,000 population compared with 0.25 per 100,000 population for the rest of the United Kingdom. This 32 fold difference in clinical attack rates may to some extent be explained by the fact that the rest of the United Kingdom had had an increased prevalence of Coxsackie A9 infections during 1966–67 and 1969 with a subsequent increase in the number of immune persons in the population during 1970. The patients investigated in this outbreak were highly selected in that their illnesses were severe enough to rquire admission to hospital. Undoubtedly there were many more minor illnesses and subclinical infections in the community associated with Coxsackie A9 virus.

Eighty-six per cent of patients with Coxsackie A9 infection in Northern Ireland had aseptic meningitis and this predominance contrasts with 27 per cent of children and 61 per cent of adults reported to have central nervous system involvement associated with Coxsackie A9 infection in the United Kingdom during 1969 (British Medical Journal, 1970b). In most outbreaks of viral meningitis investigated in Northern Ireland including the present outbreak, twice as many males as females were involved.

It was of interest that 8 families had two or more cases of aseptic meningitis associated with Coxsackie A9 infection in each family. Since spread of enteroviruses is by direct person to person oral transfer of human faeces or from the respiratory tract it follows that family infections are common though clinical illness is usually rare. The fact that Coxsackie A9 virus was isolated earlier from CSF and throat swabs rather than faeces probably means that the virus was of higher titre in the CSF or throat so that respiratory rather than faecal spread may have been of importance in this outbreak.

Summary

During the summer months of 1970 there was an outbreak of Coxsackie A9 virus infection in Northern Ireland which was confined largely to Belfast and its environs. The clinical attack rate was 32 times higher in Northern Ireland than the rest of the United Kingdom. One hundred and twenty-three patients were diagnosed. Males were affected twice as often as females and over half the patients were children under the age of 10 years. One hundred and six patients (86 per cent) had aseptic meningitis and there were 8 family outbreaks where two or more people in each family had aseptic meningitis.

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THE RELATION OF MEAT-EATING TO THE INCIDENCE AND SEVERITY OF SPRUE AND THE RELATION OF SPRUE TO DIARRHOEA IN THE UNITED KINGDOM

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SPRUE IS one of the most important diseases in the world. It probably disables thousands of adults each year, and it also occurs in children. With the recession of malaria, bowel diseases bulk bigger in tropical health. If money is available to pay for them, there are effective treatments for bacillary dysentery, amoebic dysentery, cholera, typhoid, and intestinal tuberculosis. Worm infestations were never more easily and safely treated. Sprue, however, remains the same difficult disease. The numbers are so great, and the treatment so tedious and imperfect, that the application of some general preventive principle is very desirable.

This note is written with India sprue in mind, mainly in Indian soldiers. Caribbean sprue apparently is different in some respects, and, like Indian sprue in Europeans, may be more easily treated. The response to folic acid is better. Sprue is said not to occur in Africa, but Trowell (1960) speaks of protein malnutrition in adults causing loose stools, anorexia and oedema. This may be related, as we see later.

We may provisionally define sprue as a disease manifested mainly in the small intestine, with a protracted course, little tendency to natural cure, and a strong tendency to get worse. There is intestinal malabsorption, more or less severe, with anorexia, wasting, loss of strength, anaemia, ankle oedema and glossitis. Apathy and lack of drive are notable. There is usually no fever. The disease is very disabling and may end in death. It is not solely or mainly a disease of Europeans in the tropics. It attacks Indians and Pakistanis too. It is a disease not only of adults, but also of children (Hurst, 1942; Mathew *et al.*, 1964). It is not merely sporadic. It occurs in epidemics. Since it can be epidemic, it must be environmental, and not due to an innate abnormality of the small intestine.

The epidemic of sprue in the 14th Army during its campaigns on the Indo-Burman frontier, particularly in 1944, was specially instructive. Thousands of Indian troops (young men recently healthy, be it remembered) were evacuated to hospitals because of disabling sprue. Europeans were attacked, but in a smaller number. These events established that sprue could be epidemic, and epidemic in Indians.

Further light was thrown on the disease by the observation that the incidence was worse in troops deprived of meat. The difficulties of supply were great. British troops could be supplied with tinned beef and that was done. Indian troops, Musalman and Hindu, had to be supplied with living goats and sheep. These were killed in the unit just before being cooked and eaten. Those for Musalman use are killed by Musalmans by a method prescribed by their religion. Procurement in India, transport to the field and distribution to units of the number of live animals needed was impossible. The extra ration of milk authorised for issue to meat-eaters when the meat ration was deficient, or to non-meat-eaters regularly,

was seldom or never available. Meat protein deficiency therefore was severe and common in those willing to eat meat.

In those Hindus, who never ate meat in any case because of caste rules, the situation was worse. In normal times and in their homeland they relied on large quantities of milk and milk products, especially dahi. These were unobtainable in the field. In these troops sprue was specially severe. The writer observed an epidemic of sprue in the 3/4 Bombay Grenadiers in Imphal in 1944. Almost to a man the Jat company, who never ate meat, went to hospital with sprue. Of the Musalman company only two got sprue. The Musalmans had received and eaten a little meat and they had always been meat eaters. The Jats, because of their caste rules, could not be treated with injections of liver extract nor with the usual high meat protein diet, and had to be evacuated to base.

Walters (1947) reported a very similar experience in another Indian infantry battalion. In this unit too, the Jats being non-meat-eaters and receiving negligible quantities of milk, were seriously affected with sprue. In contrast the Musalmans, who were meat-eaters and who had received a little meat, were minimally affected. Of the 42 worst cases, 39 were Jats and 3 Musalmans.

It will be seen that the class composition of Indian infantry battalions (the companies being recruited from different religions and castes) provided in each battalion reported above, two groups, matched in age, sex, occupation, dress, mode of life, income, and environment, and differing only in food habits. The food habits also were matched, except in meat-eating, because the Jats and Musalmans ate the same unleavened wheaten bread, ghi, vegetables, salt and spices. These troops were not rice-eaters. A further advantage of studying the cases in the 3/4 Bombay Grenadiers was that no food was available to them other than their rations, the composition of which was known. They could obtain no other food item locally.

Similar observations were made by Ayrey (1948). He calculated that an Indian artillery regiment, severely affected with sprue, had been receiving 62 grams of protein per man per day, of which 7 grams was milk protein and the rest vegetable. The unit had had three issues of meat in six months.

The experience of Taylor and Chhuttani (1945) of the contrast in health between non-meat-eating troops and meat-eaters is relevant, though it is stated mainly in terms of anaemia. At the time the troops were in a hot desert country with a low incidence of disease and hospital admission. The meat-eaters received 20 ounces a week of meat without bone. Many of the non-meat-eaters had not received milk in lieu of meat. There was a population of adult Indian males consisting of 17,000 meat-eaters and 1,888 non-meat-eaters. In 4½ months of study there were 50 cases of anaemia. Of the 50 cases 29 were non-meat-eaters and 21 meat-eaters, so the rate of anaemia was relatively enormous in the non-meat-eaters. In 26 of the 29 non-meat-eaters the anaemia was macrocytic, one of the 26 having sprue. In 4 of the 21 meat-eaters the anaemia was macrocytic, and all 4 had sprue. Whether we include or exclude the cases of sprue, the rate for macrocytic anaemia in the non-meat-eaters is very large, compared to the meat-eaters.

Stefanini (1948) was able to contrast the incidence of sprue in Italian prisoners of war, Indian troops and British troops in an Indian hill station, and to relate the varying incidence to the varying protein intakes. The following figures are extracted from Stefanini's paper:

	Daily Rations (*protein in grams*)			
	Animal protein	*Vegetable protein*	*Total protein*	*Calories*
Italian prisoners				
before June 1944	26	51	77	3100
after June 1944	28	71	99	2610
Indian troops	14	81	95	3710
British troops	96	33	129	3690

	Incidence of Sprue per Thousand		
	1942	1943	1944
Italian prisoners of war	13.6	21.5	59.7
Indian troops	12.0	39.3	24.1
British troops	0.0	0.0	6.0

This can be interpreted as showing that the high animal protein intake of the British troops went far to protect them from sprue. The Italians with less protein were less protected and were much affected by sprue. The Indians with the least animal protein of all had the worst experience of sprue in 1943. In 1944, while still bad enough, they were not so bad as the Italians.

Since then epidemics of sprue in south India in Indian civilians have been reported (Baker *et al.*, 1962; Mathan *et al.*, 1966; Mathan and Baker, 1968; Baker and Mathan, 1968). The Indians affected obtain their scanty protein intake almost exclusively from vegetable sources. What milk there is goes mainly to the children. Sprue occurred in women and children as well as men. These southern Indians are rice-eaters, and not eaters of wholemeal wheaten bread as the northern Indians are. If the sprue of the northern Indian soldiers described above is the same sprue as the south Indian sprue described by Baker, then we need not suppose that wheat proteins played any part in producing it.

A distinction should be made between sprue in troops who, however deficient in meat, had had enough calories, and the famine disease seen in troops (e.g., long-range penetration groups) who had also been starved of calories. The Jats of the 3/4 Bombay Grenadiers described above had had sufficient calories, mainly in the shape of whole-meal wheaten bread. They made no improvement when their calorie intake was increased by special cooking of extra issues of atta, ghi and pulses. No extra animal protein was available. Because of the sunlight, anti-rachitic synthesis in the skin was probably sufficient and tetany was not seen. Because the troops were taking 100 mg. of mepacrine daily as a malarial suppressive, it is not likely that giardiasis was responsible for the small intestine disease. No doubt, however, other parasites were commonly present.

In 1944 there was still a reluctance to diagnose sprue in Indians, and a diagnosis of "anaemia", "diarrhoea", "diarrhoea with glossitis", "malnutrition" or the "marasmus syndrome" often concealed the true incidence of sprue. Although the Army commander (Slim, 1956a) recorded malabsorption in his account of the

campaign, neither sprue nor malabsorption are mentioned in the official medical history (Crew, 1966). Presumably such cases are included in "dysentery and diarrhoea."

The part played by the deficiency of meat was sometimes obscured by the reluctance of commanders and staffs to speak openly of malnutrition in the army. The use of the word was considered bad for morale. It is true too that medical officers studying sprue in base hospitals in India could not be acquainted with the environment in which the disease was acquired, and they did not understand the meat deficiency which had occurred. However Marriott (1945), Ayrey (1947) and the army commander (Slim, 1956b) confirm that meat was deficient. Marriott considered that "the anaemia seen in Indian troops appears to be related to inadequacy of meat in the diet". Ayrey considered the sprue was due to deficiency of "B_2 complex vitamins" due to dietary inadequacy. His cases did well on special issues of meat, milk, Marmite and nicotinic acid.

These impressions that normal meat-eating prevents or modifies sprue are supported by the observed value of high meat diets in the treatment of the disease. The meat diet used for many years, and advocated by Fairley (1946) has been accepted generally as significantly helpful, if not totally curative (Leishman, 1945; Woodruff, 1949; Chuttani, 1968).

There are recent observations relating protein-deficiency to small intestine disease. Tandon *et al.* (1968) described abnormality in the small intestine in protein deficient persons and its cure with protein repletion. Mayoral *et al.* (1967a) reported evidence of protein deficiency causing intestinal malabsorption. Klipstein and Baker (1970) have discussed the possible existence of "hypoproteinaemic enteropathy". This is a perhaps unjustified extension of the hypothesis of dietary protein-deficiency enteropathy, or, as the present writer would have it, dietary meat-deficiency enteropathy. Mayoral *et al.* (1967b) drew attention to the close resemblance of tropical sprue (rare in Columbia they say) and chronic protein-malnutrition-induced malabsorption. They attempted to distinguish the two by the response to protein feeding alone, the presence or absence of megaloblastic marrow, and the necessity to administer folic acid, or otherwise. It is doubtful if these really are two separate groups. If one accepts that classical tropical sprue is in main part due to protein malnutrition, then one can discard the distinction.

Althausen *et al.* (1962) observing sprue in Puerto Rico noted the protein deficiency in their cases of sprue, and also in cases of macrocytic anaemia in females. In 86 patients with sprue the average total daily protein intake was 52 grams, of which 29 grams were "complete protein". In females with sprue the average total daily intake of protein was 51 grams, of "complete protein" 29. In females with megaloblastic anaemia of pregnancy the total daily intake of protein was 50 grams, of which 21 grams were "complete'. Althausen *et al.* recognised that these cases were deficient in meat and milk intake but felt unable to blame the meat and milk deficiency alone for the disease, partly because they took a low figure (40 grams in males and 25 grams in females) as an acceptable minimum of "complete" protein, but also because sprue does occur sometimes, if relatively rarely, in people in a normal state of meat nutrition. They invoked a second factor, a hypothetical genetic tendency to sprue, and considered that the coincidence of protein deficiency and the genetic tendency might produce clinical sprue. They were

probably right that besides protein deficiency (the factor that makes the small intestine vulnerable) another factor is necessary. However the epidemic character of sprue, and its occurrence in insanitary areas, and its inability to become or remain endemic in highly sanitary areas, lead the present writer to think that the second factor is a communicable bowel infection.

Florid sprue in the United Kingdom is rare, though it is within the experience of most physicians that cases occur which cannot be distinguished from sprue (Drummond and Montgomery, 1970). That may be because meat is plentiful and cheap enough for all to have it. Sprue is said to have diminished in Hong Kong and Singapore since refrigeration became common. That may be because the retailing of meat and domestic storage are now easier, and meat more available. It is likely that the lower incidence of sprue in Europeans in the 14th Army was due to the adequate ration of acceptable canned beef.

It seems therefore that while some communicable agent, presumably an infection (though not bacillary or amoebic dysentery), initiates the small intestine disease, it is rare for the enteropathy to proceed to florid sprue in people in a normal state of meat nutrition. In such people only infrequent sporadic cases may be expected. We can prevent sprue in meat-eaters by ensuring them a daily supply of meat – say 140 grams of meat with bone. Something can be done for those who do not eat meat by supplying daily milk and milk proteins. One litre of cow's milk a day would supply 33 grams of protein. That would not be enough to give protection. The proteins of at least two litres a day would be necessary.

If we seldom see florid sprue in the United Kingdom, perhaps partly because of our meat eating, do we see the incomplete or abortive disease? Is sprue so modified by our good nutrition that it only shows as a mild subacute diarrhoea? Is our sanitation so good that the communicable agent is limited in its spread, as have been the agents of other intestinal disease? Subacute diarrhoea of unknown origin is the commonest diarrhoeal problem in Ulster and elsewhere in the United Kingdom. Not infrequently it begins when the patient is on holiday, but it can begin at home. After some non-specific treatment the acute phase passes, and the patient continues for some weeks or months to have three or four or five loose stools a day. The stools are more loose and frequent in the morning. They are of small intestine type but they are not steatorrhoeal. There is no colonic disease. Sigmoidoscopy is normal. In the more severe cases there may be a little change in the barium x-rays of the small intestine, but it is usually equivocal. Bacteriological examination is negative. *Giardia lamblia* is practically never found. Cure is usually easily obtained by administering the tablet of sulphasalazine or sulphaguanidine and the tablet of folic acid, each for 8 weeks or so. Probably this jejunoileitis of unknown origin, so familiar to us, would produce sprue if the patient were chronically meat-deficient, like the sepoys described above. All the more, if in addition he had a small intestine impaired with infections and infestations, as so many Indian villagers have.

What is true and important for adult nutrition must be even more important for the growing, developing and maturing child, and young person. If normal meat nutrition is important, not only becaues it supplies amino-acids for anabolism, but also because it maintains a normal small intestine (as this paper proposes), then it

is an important public health duty to see that meat is provided at a price that all can afford.

Lastly, it is difficult for the physician with Indian experience, reading accounts of kwashiorkor in Africa, to see how that disease differs from the protein-deficiency sprue of India. Sprue has traditionally been described in adults who have finished growth and maturation, kwashiorkor in young children in a fast growing phase. The disease mechanisms, nevertheless, seem to be similar.

Summary

It is shown that sprue occurs predominantly in persons with a dietary deficiency of meat. Regular adequate eating of meat protects against sprue or modifies the attack, because it maintains the small intestine in a state in which it resists the communicable, presumably micro-organismal, agent, infection with which is the other factor concerned in producing clinical sprue. Administration of meat is essential in the treatment of sprue for those who will take it, even if other means such as folic acid, hydroxocobalamin, iron and intestinal antibacterial drugs are also employed. Large quantities of milk and milk proteins are helpful, but not so good as meat. What is necessary in adults is even more important in childhood in the time of growing and maturation. Sprue will probably disappear as income per capita rises, more meat is eaten and sanitation improves.

Note on Animal Proteins

Dahi is milk, boiled, and then inoculated with a little old dahi, whereupon it thickens and sets. It has been an article of food among cattle-keeping people, probably since pastoral life began. It was in use among the Irish and appears in the older Anglo-Irish literature as bonnyclabber (Irish: bainne – milk; clabair – something thick like mud). A similar artificial product is sold nowadays as yoghurt. The word is Turkish. Lasi is dahi, diluted with water to make a drink, with sugar or salt added. Dahi and lasi are much used in northern India and West Pakistan.

Buffalo milk contains 4.3 grams of protein per cent (and 8.8 grams of fat and 5.1 grams of carbohydrate). These values are greater than those of cow's milk, but buffalo milk is often diluted before it is drunk.

The only food other than milk which can be derived from animals without killing them is blood obtained by intermittent venesection. This is practised by African tribes. Among the Masai who are a cattle-herding people there is said to be no kwashiorkor. Blood from cattle was a resource of the pastoral Irish. The blood could be mixed with meal and cooked. The modern representative is the black pudding.

Musalmans have no objection to eating beef if it has been ritually killed, but the killing of cattle is so particularly horrifying to Hindus, that for the sake of communal peace, Musalmans confined themselves to the meat of goats and sheep. Equally pork is forbidden to Musalmans, and it was never used in the Indian army by any group. Had whale meat been available, it would have raised a difficult question for Musalmans. If a whale is an animal, it would have to be killed ritually (which is impossible). If it is a fish, that would not be necessary.

Fish, although allowed for Hindu meat-eaters and for Musalmans, is so small in quantity and so local in availability as not to count as a source of protein. Fowl

are allowed to Hindu meat-eaters, and if killed ritually to Musalmans, but for the same reasons fowl made no contribution to supply. Nor did eggs.

The word mutton is commonly used in Indian English for both sheep and goat meat. Usually only male goats and sheep are killed for meat, so as to preserve the breeding stock. It is evident that great attention should be paid to the health and nutrition of sheep, goats and cattle, if the world supply of meat and milk is to be increased.

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HEART BLOCK IN MYOCARDIAL INFARCTION: A HISTOPATHOLOGICAL STUDY

by

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THE CASE with apoplectic attacks described by Robert Adams (1827) and also one of the cases reported by Stokes (1846), had yellow discolouration and friability of the left ventricular wall, almost certainly had organising myocardial infarcts. Recently Davies *et al* (1967) studied the histology of the conducting tissue in fourteen cases with complete heart block complicating myocardial infarction, but in view of the therapeutics advantages of endocardial pacing (Scott *et al* 1967), a further histopathological study of nine cases was undertaken.

METHODS

The hearts were examined in the routine manner at the time of necropsy. Later the formalin-fixed specimens were prepared for examination of the conducting tissue using the method described by Hudson (1965). Partial serial sections at 0.5 mm were made through a block of the upper interventricular septum to display the main AV bundle, the AV node and bundle branches. The histological sections were routinely stained with haematoxylin and eosin, and also with Hart's modification of Weigert's elastic tissue stain, counterstaining with van Gieson's picro-fuschin. Sections displaying the AV node, the main bundle and the bundle branches were examined in order to assess the relative amounts of conducting fibres, fibro-elastic tissue and the presence of fatty tissue (Sims, 1968). The examination was made without a knowledge of the clinical details in nine cases with heart block and also in sixteen cases with normal conduction. The results of this were compared statistically using an exact probability computer programme.

RESULTS

The details of the nine cases with complete heart block are presented in Table I. The ages ranged from 38 to 76 years (average 65 years. The duration of the heart block, as estimated from the first electrocardiograph showing heart block until the time of death, was less than six hours in five cases. The heart rate was between 40–50 beats per minute, but one case had AV dissociation with a rate of 100/minute. Heart failure was present in seven of the nine cases.

In five cases a recent coronary occlusion was demonstrable, while in seven cases recent infarcts were present, of which four were anterior and three posterior in position. Recent infarction involving the conducting tissue was present histologically in four cases, and in these the duration of the block was longer than six hours. In three instances, with one anterior and two posterior recent infarcts, there was infarction of the bundle branches, and in case No. 3 (Figure) only the right bundle branch showed evidence of infarction. Case No. 6, a 38-year-old male, showed recent infarction of the AV node and main bundle associated with the unusual

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TABLE I

Cases of Complete Heart Block with Myocardial Infarction

Case	*Age & Sex*	*Duration of Block*	*Heart Failure*	*Occlusion Site & Age*	*Infarct Site*	*Infarct Age*	*Histological findings in conducting Tissue*
1	60 M	Several hours	+	LAD & CA Recent	Antero-Septal	Recent	Bundle branches: slight fibrosis
2	76 M	2 days	+	RCA Recent	Posterior	Recent	Bundle branches: Fibrosis and recent infarction
3	75 M	24 hours	+	LAD Old & Recent	Antero-Septal	Healed Recent	Bundle branches: Fibrosis and Recent infarction R. branch
4	74 F	1 week	+	RCA Old	Posterior Antero-Lat.	Healed Recent	Bundle branches: Recent infarction
5	71 F	3–6 hours	+	LAD Recent			Bundle branches: Fibrosis
6	38 M	1 week	+	RCA Recent	Antero-Laternal	Recent	AV node & AV bundle: Recent infarction
7	74 M	Several hours	+	LAD Old	Antero-Septal Posterior	Healed Recent	AV bundle and Bundle branches: Fibrosis
8	65 M	Several hours	—	RCA Old	Posterior	Recent	
9	57 M	2 hours	—	LAD & RCA Old			

LAD=Left anterior descending artery CA=Circumflex artery (left)
RCA=Right coronary artery

TABLE II

A comparison of the histological features of conducting tissues in cases with complete heart block complicating myocardial infarction and cases with normal conduction.
(*Age comparison of groups* P>0.25)

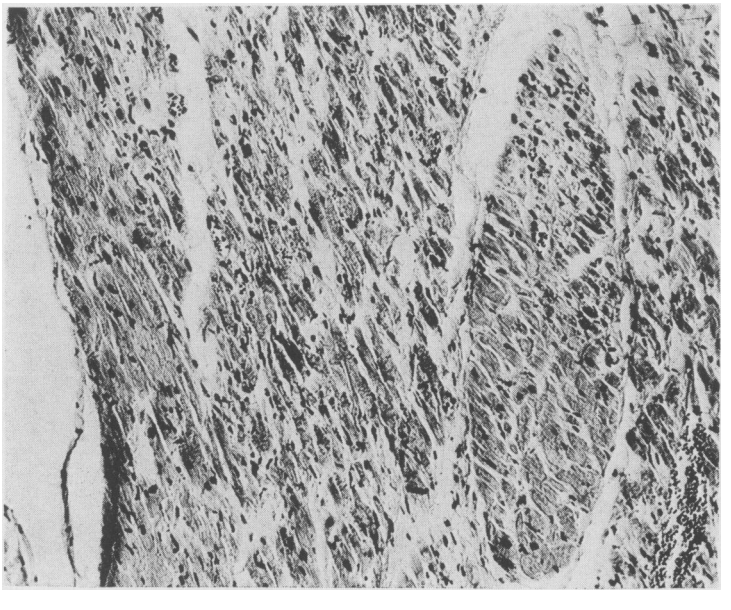
Conducting Tissue	*Number of Cases*	*Histological Assessments*													
		Amount of Conducting Tissue			*Fibrosis*				*Elastosis*				*Fat*	
		—	+	-|-|-	—	+	++	+++	—	+	++	+++	—	+
AV Node	Heart Block 9	0	3	6	4	3	2	0	0	6	3	0	4	5
	Normal 16	0	10	6	2	10	4	0	0	3	9	4	4	12
		P=0.23			P=0.29				P=0.034				P=0.39	
AV Bundle	Heart Block 9	0	4	5	2	4	3	0	0	6	3	0	4	5
	Normal 16	0	7	9	0	9	4	3	0	5	6	5	8	8
		P=1.0			P=0.16				P=0.11				P=1.0	
Bundle Branches	Heart Block 9	2	4	3	0	4	1	4	0	6	3	0	6	3
	Normal 16	0	9	7	1	10	3	2	0	3	8	5	10	6
		P=0.23			P=0.38				P=0.032				P=0.99	

finding of a recent occlusion of the right coronary artery and an anterior infarct. In several cases there was fibrosis of the bundle branches. When the histological features were compared statistically with assessments from a similar age group with normal conduction (Table II), no significant difference is detected, where P is never <0.02.

Discussion

Since myocardial infarction cannot be recognised using haematoxylin and eosin staining technique for about 10–12 hours after the onset of ischaemia, changes could not be expected in three cases where heart block was present for less than six hours. In these cases histological examination was therefore not helpful.

From a knowledge of the conducting tissue of the human heart and its blood supply (James and Burch, 1958) it could be predicted that occlusion of the right coronary artery would produce posterior infarction with ischaemia of the AV node in 90 per cent of cases, producing perhaps heart block for a short time until a satisfactory collateral blood supply developed. Occlusion of the anterior descending branch of the left coronary artery will lead to anterior infarction, which may produce heart block by extension into the septum to involve the bundle branches. The heart block so produced is more likely to be associated with a large infarct and to be permanent. This association of small posterior infarcts with transient heart block and of large anterior infarcts with permanent block is much modified by the individual variation in blood supply, the distribution of atherosclerosis, and the development of the collateral circulation in the heart. This is demonstrated by case No. 2, in whom there was extension of the posterior infarct forwards into the septum to involve both bundle branches, while in case No. 6 the recent infarction of the AV node and bundle was associated with a recent anterior infarct.



Case No. 3

The right bundle branch is involved with the surrounding septal myocardium by recent infarction. H. & E. x 100

Another factor which would appear to be important is previous damage of the conducting tissue, so that the remaining tissue is more likely to be vulnerable to further ischaemia. This would seem to be the case in four of the present cases where scarring was present in the bundle branches. In two of these cases the bundle branches were infarcted but in the other cases no infarction was discernable. However, when the histology of cases of similar age with normal conduction was compared, no significant increase of fibrosis in the bundle branches was found. This apparent fibrosis may therefore represent an ageing change, and in this state ischaemia could easily produce either a transient or persistent failure of conduction. In case No. 3 there was infarction of the septal myocardium and right bundle branch. After 24 hours normal AV conduction returned, but with residual right bundle branch block. Davies *et al* (1967) in a study of fourteen cases with transitory complete heart block complicating acute myocardial infarction, showed that focal necrosis of the AV node was present in three instances, four had partial and one had total necrosis of the left fascicles, and two had bilateral branch necrosis when chronic heart block was more likely to ensue.

Seven of the nine cases studied were also included in a clinical investigation of the treatment of complete heart block complicating myocardial infarction with either endocardial pacing or drugs (Scott *et al*, 1967). Of the fifty patients in that study, twenty-nine had inferior (pathologically referred to as posterior) infarcts and eighteen had anterior infarcts, while two had both. The mortality was found to be 59 per cent in patients with anterior but only 39 per cent in those with inferior infarcts. The reason for the greater mortality with anterior infarcts complicated by complete heart block, is that infarcts must be large to involve both bundle branches, and are therefore more likely to be accompanied by shock and heart failure. It is in this group that endocardial pacing was found to be of value, since by raising the heart rate to 85–90 beats per minute, the cardiac output could be maintained and the circulatory failure corrected.

Complete heart block occurs in 5–8 per cent of cases with myocardial infarction but only in about 10 per cent of these does it become chronic (Lancet, 1968). In the study of Scott *et al* (1967), the heart block lasted an average of three days and in only one case did Grade I heart block persist. Clinically they found that the prognosis depended upon the age of the patient, the site of infarction and the time of onset of complete heart block.

Summary

The conducting tissue of the heart has been examined in nine cases with complete heart block complicating myocardial infarction. There was evidence of recent infarction in the bundle branches in three cases and of the AV node and main bundle in another case, but the distribution of necrosis has little relationship to the site of arterial occlusion or the location of the infarct. This is accounted for by the widespread nature of the arterial disease and the extensive collateral circulation. Previous damage with subsequent fibrosis, or the degeneration due to ageing of the conducting tissue, may also make the development of heart block more likely with any further episodes of ischaemia. These findings are discussed in the light of a contemporary clinical investigation.

Acknowledgements

I should like to thank Professor Sir John Biggart, C.B.E., under whose direction this work was done, and the consultants of the Royal Victoria and Belfast City Hospital for access to their patients' records. I am also indebted to Mr. W. Kirkwood for the preparation of the histological sections, to Dr. D. Merrett for his advice on the statistical methods, and to Mr. D. Mehaffey for the photography.

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CHRONIC SUBDURAL HAEMATOMA
(A Review of 66 Cases)

by

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IF THE presence of a chronic subdural haematoma is suspected, the neurosurgical management of the case can be simple, and may consist of no more than an exploratory burr-hole, which at the same time confirms the diagnosis and cures the condition. Often, however, alternative diagnoses on clinical grounds are likely, and special methods of investigation are preferable. The majority of the cases are admitted to general medical wards, sometimes with rather vague history and complaints, and it falls to the physician to suspect the condition and select suitable cases for special opinion and investigation. This is often not an easy selection to make, and it is in the hope that this selection might be facilitated that we record our more recent experiences in the diagnosis and treatment of patients with chronic subdural haematoma.

Material and Method

The case records of all adult patients with chronic subdural haematoma treated in the Department of Neurological Surgery, Royal Victoria Hospital, Belfast, from January 1962 to June 1969 were reviewed. The haematoma was regarded as chronic if there was an interval of over three weeks from injury, or when there was no history of head injury (Ecklin *et al*, 1956; McKissock *et al*, 1960; Rosenbluth *et al*, 1962; Gilmartin, 1964).

Clinical Features

There were 66 patients in this series. It is interesting to note that 52 cases (79 per cent) were first admitted to a medical ward (Fig. 1). Only eight patients were admitted directly to the neurosurgical ward. The age and sex distribution of these patients is given in Table I. There were three times as many males as females.

TABLE I
Age and sex distribution of 66 patients

Age in Years	11-20	21-30	31-40	41-50	51-60	61-70	71-80	*Total*
Males	1	3	2	6	8	22	7	49
Females	0	0	1	1	4	8	3	17
Total	1	3	3	7	12	30	10	66

Aetiology

Forty-two patients (61 per cent) had a history of head injury from three weeks to six years previously, though this history was not always available at the time of admission. Two patients were known hypertensives. A history of chronic alcoholism was present in 12 cases. Two patients had idiopathic thrombocytopaenic purpura and one had factor X deficiency. Two cases were known epileptics.

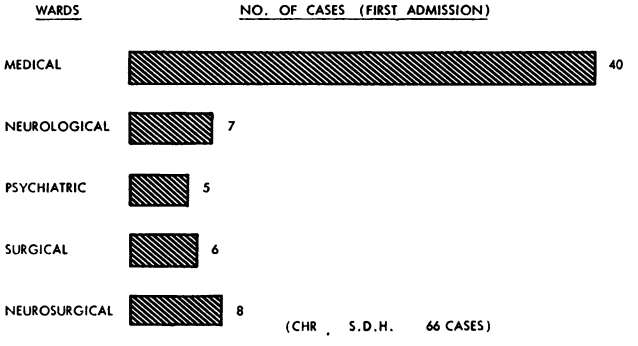


FIG. 1
Wards to which these patients were initially admitted.

Symptoms

The overall frequency of symptoms and their occurrence as the first symptom are shown in Fig. 2. Over half the cases (56 per cent) presented with headache. It was unilateral in seven cases and was lateralised towards the side of the lesion. One of the characteristic features observed in 12 cases was fluctuation in the level of consciousness. This was helpful in suspecting the diagnosis.

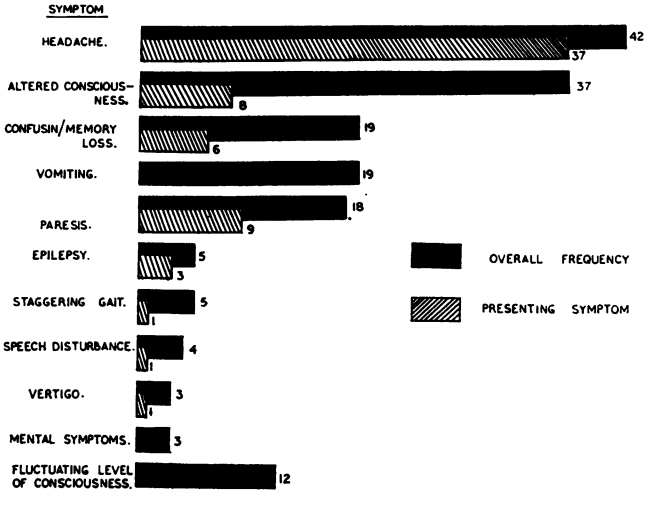
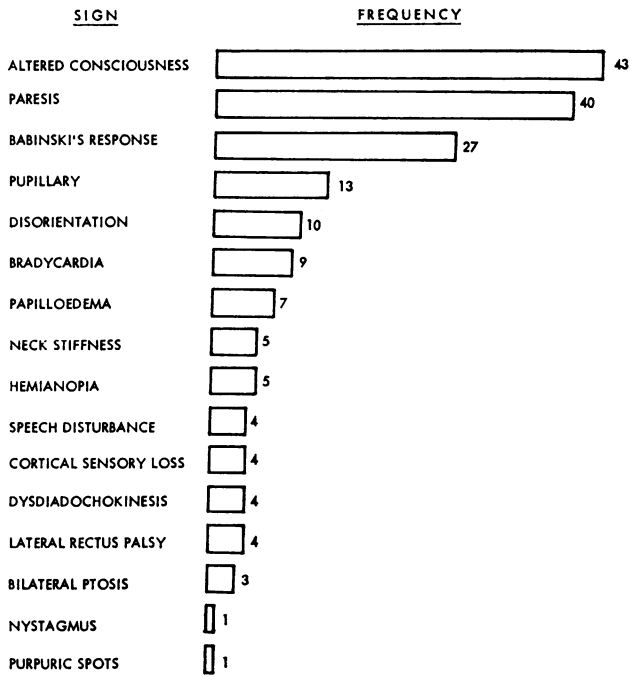


FIG. 2
Frequency of the symptoms and their occurence as the first symptom.

Signs

The frequency of various clinical signs is shown in Fig. 3. Of the 43 cases with altered consciousness, 24 were drowsy, 11 were semi-conscious and eight were comatose. Motor weakness was seen in 40 patients, 24 had hemiparesis, 12 monoparesis and four quadriparesis.

FIG. 3
Frequency of clinical signs.



Lateralising Value of Signs

(a) *Paresis*: Hemiplegia or monoplegia was contralateral to the side of the lesion in 26 cases (73 per cent) and ipsilateral in eight cases (22 per cent); two patients had bilateral haematomata. Of the four patients who had quadriparesis, one had bilateral haematomata, while the other three had a severe degree of brain stem compression.

(b) *Pupil*: The larger pupil was on the side of the lesion in ten cases (77 per cent), on the contralateral side in two (15 per cent), and on the side of the more bulky haematoma in the one case which had haematomata on both sides.

(c) *Speech Disturbance*: All four patients with aphasia or dysphasia had the lesion on the dominant side.

(d) *Hemianopia*: Visual field defects had accurate localising value in all five cases in which they could be elicited.

INVESTIGATIONS

Plain X-rays of the skull were done in 49 patients. These revealed a fracture of the skull in three cases (6 per cent), and a displaced pineal in 16 cases (32 per cent). The diagnostic value of other ancillary investigations is shown in Table II.

Lumbar Puncture

Cerebrospinal fluid examination was done in eight patients prior to their transfer to the neurosurgical unit. The protein content was slightly elevated in four cases.

However, the procedure is not without its risks, and five patients had definite deterioration in their neurological status following lumbar puncture, necessitating urgent transfer to the neurosurgical unit.

TABLE II
Diagnostic accuracy of special investigations

Investigation	*Total Number*	*Correct Localisation*	*Percentage Correct*
Electroencephalogram	34	18	53
Angiogram	24	24	100
Echoencephalogram	24	20	83
Brain Scan (Tc^{99m})	2	2	—
Air Encephalogram	1	1	—

TREATMENT AND RESULTS

Treatment

The initial operative procedure in each case was bilateral burr-holes and evacuation of the haematoma. The cavity was reaspirated, if required, more than once. A drain was left in the subdural space in 15 cases. There were eight cases (12 per cent) with bilateral subdural haematomata. A haematoma capsule was present in 34 cases (51 per cent), and in six of these it was described as "thick" or "leathery". Three cases required delayed craniotomy and excision of the membranes when the haematoma failed to resolve after repeated aspirations. Failure of the compressed hemisphere to expand after evacuation was encountered on 20 occasions. In four it was dealt with by intraventricular injection of saline; the remaining 16 had saline injected by the lumbar route. The latter was undoubtedly a more satisfactory procedure. The intracranial hypotension persisted post-operatively in six patients, and lumbar saline injections were repeated in these cases. The expansion of the hemisphere was studied by the simple procedure of applying clips on the surface of the brain and the dura, and following their approximation with serial radiographs.

TABLE III
Results – As a function of the initial level of consciousness

Level of Consciousness	*Total No.*	*Fully Recovered*		*Slightly Disabled*		*Severely Disabled*		*Died*	
		No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Alert	23	23	100	0	—	0	—	0	—
Drowsy	24	23	96	1	4	0	—	0	—
Semicomatose	11	8	73	0	—	1	9	2	18
Comatose	8	2	25	1	12.5	1	12.5	4	50

TABLE IV

Results – As a function of the age of the patient

Age	*Total No.*	*Fully Recovered* No.	%	*Slightly Disabled* No.	%	*Severely Disabled* No.	%	*Died* No.	%
Up to 50 years	14	14	100	0	—	0	—	0	—
51–60 years	12	9	75	1	8.3	1	8.3	1	8.3
61–70 years	30	28	93	0	—	0	—	2	7
71–80 years	10	5	50	1	10	1	10	3	30

Results

Of the 66 cases, 56 (84 per cent) made a full recovery. Four patients were left with some disability, which was slight in two and severe in the other two. Six patients died, giving an operative mortaliy of 9 per cent. Thus, of the 60 survivors, 58 made good recoveries. An analysis of the results against the initial level of consciousness shows that patients in semicoma and coma carried a worse prognosis (Table III). A similar analysis against age of the patients showed that patients in the older age group fared worse (Table IV).

DISCUSSION

Some of the difficulties as well as pointers to the diagnosis of chronic subdural haematoma have been revealed in this study. The main difficulty in diagnosis or even suspicion of the lesion is the absence of any history of head injury, or failure to assess the significance of a trivial head injury. It should be remembered that headache and mental symptoms, with relatively few signs in the limbs, are prominent in patients with chronic subdural haematoma. These features are commonly attributed to cerebral infarction or arterio-sclerosis, especially when the traumatic incident has been forgotten. One must be vigilant while making these diagnoses, especially in elderly patients, as in a case of "stroke" further investigations are not usually pursued. In fact, an untreatable lesion ("stroke") should seldom be diagnosed on clinical grounds alone. In a review of the pathological diagnosis of 276 cases diagnosed as "stroke", Ranskind and Weiss (1969) found 16 cases of chronic subdural haematoma.

The source of most chronic subdural haematomata is the bleeding from the communicating veins and the pacchionian granulations near the superior sagittal sinus. The haemorrhage tends to gravitate downwards and the clot is contained by the formation of a membranous sac (Gardner, 1932). Further expansion of the haematoma has been thought to be due to osmotic processes. However, some doubt has been cast on this hypothesis of the pathogenesis of chronic subdural haematoma by the experimental work of Goodell and Mealey (1963).

Skull X-rays may or may not reveal a fracture line. If the pineal gland is calcified it may be displaced from the midline, but with bilateral haematomata it may be central. Occasionally a chronic subdural haematoma may become calcified (Jackson and Clare, 1965). Lumbar puncture not only does not provide any help in the diagnosis, but may be hazardous, and should not be done if a subdural haematoma is suspected, but the case should be referred for neurosurgical opinion.

If available, echoencephalography would demonstrate cerebral hemisphere displacements and the haematoma echoes in the majority of, but not all, cases, and should be over-ruled if findings are contrary to clinical expectation. The final proof of the diagnosis comes from angiography or burr-holes. In the elderly or acutely ill patients, suspected of harbouring chronic subdural haematomata, the diagnosis is best confirmed (or excluded) by exploratory burr-holes. This simple procedure simultaneously diagnoses and, in the majority of cases, cures the condition. In view of the reported 10 to 20 per cent incidence of bilateral lesions the burr-holes must always be made on both sides. Our experience with brain scanning was too limited in this series, but further experience since has shown it to be a very valuable diagnostic procedure in chronic subdural haematomata, but again not absolutely reliable, false negative findings occurring occasionally, even in chronic haematomata with thick membranes.

Isolated reports have appeared documenting spontaneous resolution of chronic subdural haematomata without surgery (Bender, 1960; Gannon, 1962; Gannon *et al*, 1962), and even of medical treatment by prolonged dehydration (Suzuki and Takaku, 1970). The preferred treatment of chronic subdural haematoma, however, remains surgical in the vast majority of cases.

The results of the treatment depend upon the age and the level of consciousness of the patient (McKissock *et al*, 1960; Walker *et al*, 1968). Five of the six patients who died in this series were above the age of 60 years, and all were in coma or semicoma at the time of surgery. The prognosis would, therefore, seem to depend upon the alterations produced locally in the underlying cerebral tissues by the haematoma and the general changes in intracranial dynamics. Because the haematoma is extracerebral and slow in evolution, the latter effects predominate in the form of alterations in the level of consciousness and headaches. The older patients have a worse prognosis because the ageing cerebrovascular state does not allow of these gross disturbances.

Summary

A review of the clinical features, diagnosis and management of 66 cases of chronic subdural haematoma is presented. Difficulties in the initial suspicion of the diagnosis are described. In general, cases of chronic subdural haematoma have a good prognosis, except in elderly persons or those with severely depressed level of consciousness.

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CERVICAL CYTOLOGY IN NORTHERN IRELAND: AN OPERATIONAL REVIEW

by

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INTRODUCTION

CERVICAL CYTOLOGY was introduced into Northern Ireland in 1962 when a programme for the taking of smears from patients attending Gynaecological and Obstetrical clinics in teaching hospitals in the Belfast area was initiated (Willis and Woods, 1969). In 1965 the screening programme was extended throughout Northern Ireland, using a variety of medical settings (Robertson and Crozier, 1968). The comprehensive service has therefore been in operation for some five years.

This paper concentrates on the problems of organisation and acceptance of cervical cytology in Northern Ireland. The authors are aware that consideration of these operational aspects might be considered premature as the evidence in favour of cervical cytology as a preventive measure is, as yet, uncertain. Knox (1966) in a scrutiny of the available evidence remarks, 'There is an informed scepticism of the claims made of it'. Evidence from British Columbia is far from conclusive, even after considering the recent decline in incidence as indicated by Fidler *et al* (1968). On the other hand, a recent report from Aberdeen indicates a significant fall in the incidence of cervical cancer and an increase in the five year survival rate (Macgregor, Fraser and Mann 1971), after a very high rate of screening had been achieved. Figures from the Office of Health Economics (1969) suggest that cervical cytology cannot be justified on grounds of economic investment alone, as can, for example, health education on cigarette smoking or a programme of influenza vaccination. While acknowledging this uncertainty there does appear to be a case for examining a service which has been so widely adopted as that of cervical cytology in the United Kingdom. Also, the conclusions might be of general applicability in helping to formulate policy for the introduction of other preventive measures.

Three aspects of the service are examined:

(i) The source of the smears;
(ii) The social and demographic characteristics of the population screened, and
(iii) The interaction of (i) and (ii).

An analysis of these particular features should provide some clues as to the appropriate direction of the service in the future, and, in particular, the development of health education.

MATERIALS AND METHODS

Other papers in this field (Wakefield and Sansem 1965, Robertson and Crozier, 1968, Willis and Wood, 1969) have relied upon data taken from the laboratory reports which of practical necessity do not include a large number of social and demographic variables. Even where specific attempts have been made at recording the social class of the client, difficulties have arisen with unclassified records, e.g., in Manchester (Sansem, Wakefield and Yule, 1970) social class was missing from

11.5 per cent of all smears received from general practitioners. However, the material analysed in this paper is part of a much larger community survey to determine the relationship between knowledge of cancer, and attitude towards the disease.

A random sample of approximately one in 1,000 women aged 20 and over from the whole of Northern Ireland was drawn from the population register of the General Health Services Board, using a random number technique. In January 1970, the participants were interviewed, mainly by graduate Psychology students. A response rate of 84.1 per cent was achieved with a total sample of 531.

The following variables were recorded at interview:

1. Tested/not tested.
2. Where test was taken, i.e. hospital, maternity hospital, local authority health clinic, general practitioner or family planning association.
3. Age.
4. Marital status.
5. Occupation, later assigned to a social class using the Classification of the Registrar-General 1966.
6. Age on completion of full-time education.
7. Geographical location; which was consequently analysed in terms of urban, i.e. the greater Belfast area, semi-urban, i.e. towns of more than a population of 5,000 and rural, constituting the rest.
8. Knowledge of cervical cytology as a preventive measure.
9. Knowledge of symptoms of cancer of the cervix.

As Wakefield (1969) has shown that some women are not aware of having been tested, this method of self-classification, at interview, might be slightly deficient in differentiating between the screened and unscreened participants.

Results

The overall rate of testing amongst women aged twenty and over, from all sources, was 23.2 per cent. However, as previously mentioned, Wakefield (1969) found in a sample of women in the Manchester area that 9 per cent of tested females were not aware of having been tested. If a similar pattern exists in Northern Ireland this would mean an upward adjustment of 2.3 per cent making an overall rate of 25.6 per cent. This figure is compared with those of England and Wales in Table I. However, these figures are crude rates; i.e. they do not take into account the differing age and social class structures and the percentage of married women in the different regions; and must be regarded with care. The Department's figures might also conceal a number of second tests as they are calculated from laboratory returns. It is interesting to note that the South Western Region has the second lowest figure. It is a region not dissimilar to Northern Ireland, being dominated by one major city (Bristol) with the remainder of the population living in towns and isolated farmsteads.

During the preliminary analysis it was found that 1 per cent of single women had been tested compared with 29.1 per cent of married/once married respondants. Marital status is therefore a very important factor but unfortunately, due to the small number of tested single women in the sample, the subsequent investigation was based solely on the data for married/once married women.

TABLE I

Percentage of women screened in each hospital region

Region		1964–1969 *% of women aged 20 or over tested*
Northern Ireland		23.3
Northern Ireland adjusted		25.6
East Anglia	R.H.B.	44.5
Birmingham		44.5
Leeds		41.2
Liverpool		28
Manchester		47
Metropolitan	N.E.	44
	N.W.	55
	S.E.	46.5
	S.W.	47.5
Newcastle		44.5
Oxford		53
Sheffield		54
South Western		37
Welsh		40
Wessex		41

Source: Department of Health and Social Security, London.

TABLE II

Percentage of married women screened by social class

Registrar's General Social Class	*% of women aged 20 or over tested*
I	59
II	35
III	33
IV	26
V	22

TABLE III

Percentage of married women screened by age

Age	*% of women tested*
20–29 years	45
30–39	43
40–49	28
50–59	12
60 plus	2
40 plus, and in social class IV & V,	14

The social class gradient is illustrated in Table II. The rates vary from 58.8 per cent for social class I to 21.6 per cent for class V. Similarly the age rates (Table III) decline from 44.6 per cent for the 20 to 30 age group to 2.2 per cent for the over sixties.

The population tested can be logically divided into two parts:

(a) Clients tested because of their particular health circumstances, e.g. pregnancy, desire for family planning advice, referral to gynaecological clinic or presentation at a venereal disease clinic.

(b) Clients motivated by their general practitioners or themselves.

Obviously there is some interaction between the two groups; for example, if a woman has been tested in hospital it eliminates the need for her to go to a clinic or general practitioner for a test. Also, the categories are not mutually exclusive, because some of the participants have been tested in more than one medical setting. (In the sample of women tested) 75 per cent belong to the first category and 25 per cent to the second.

The univariate analysis was therefore divided into two parts (a) all sources of smears and (b) smears from general practitioners and local health authority clinics. With all sources of smears (Table IV) social class and education were significant, age and knowledge of cervical cytology as a preventive measure highly significant. In the self or doctor motivated group (Table V) social class had a larger effect, while knowledge of cervical cytology was again significant. The rate for small towns was significantly higher than for greater Belfast or the rural areas. Age was no longer significant, as the majority of younger women are tested at the maternity clinics.

Discussion

Epidemiological studies indicate that in the development of cancer of the cervix, women of low socio-economic class and aged over forty are most at risk. These facts were emphasised in the report of the Registrar-General for England and

Table IV

Univariate analysis – all sources of smears

Variable	*Degrees of Freedom*	χ^2	P
Age	4	29.30**	<0.005
Social class	4	9.93*	0.05–0.25
Education	2	7.90*	0.025–0.01
Urbanity	2	4.17	0.25–0.1
Towns v. rural	1	2.79	0.1–0.05
Urban v. rural	1	3.66	0.1–0.05
Urban v. towns	1	0.004	>0.9
Knowledge of cervical cytology as a preventive measure	1	30.69**	<0.005
Knowledge of symptoms of cancer of the cervix	1	0.77	0.5–0.25

**Significant at the 1% level

*Significant at the 5% level

TABLE V

Univariate analysis – Self or doctor motivated (General Practitioner/or Local Health Authority cervical cytology clinic)

Variable	*Degrees of Freedom*	χ^2	P
Age	4	7.005	0.25–0.1
Social class	4	15.62**	<0.005
Education	2	5.22	0.1–0.05
Urbanity	2	9.55*	0.01–0.005
Towns v. rural	1	7.68*	0.01–0.005
Urban v. rural	1	1.23	0.5–0.25
Urban v. towns	1	9.20**	<0.005
Knowledge of cervical cytology as a preventive measure	1	14.81**	<0.005
Knowledge of symptoms of cancer of the cervix	1	0.60	0.5–0.25

**Significant at the 1% level. *Significant at the 5% level.

Wales (Kennaway 1948). Unfortunately, the present survey shows that these are the groups with the lowest rates of testing here in Northern Ireland. Again, the incidence of cervical carcinoma has been shown to be highest in urban as contrasted to rural communities (Coppleson, 1969). The findings indicate that amongst the self motivated group, i.e. those tested by the general practitioner and at the local authority cervical cytology clinic, the percentage screened is highest in the towns, and this suggests that if the programme is to be really effective, then the rates must be increased, not alone in the rural parts of the province, but particularly in the greater Belfast region.

It might reasonably be asked if attention should be directed towards single women whose rate of testing depresses the overall total. Studies have revealed that virgin and celibate women are almost without risk (Martin 1967). Even with the influence of the permissive society, there certainly would not appear to be a very strong case for diverting effort and resources to this particular group of the community.

The survey shows that only 23 per cent of women aged twenty and over were tested. Even this figure, low as it is, can be very misleading, when we consider that almost 75 per cent of the women screened were tested because of their particular health circumstances, e.g., attendance at maternity, family planning, gynaecological or venereal disease clinics, i.e., 'captive clients'. Obviously most of these women are in the younger age groups and whilst it is important that they should be tested, one might reasonably suggest that future growth of the service must come from the local authority and general practitioner sector; especially in testing the high-risk low-responding groups. How is this to be achieved? Robertson and Crozier (1968) have argued that further development of the service is now as much dependent upon effective publicity, as upon the provision of laboratory facilities. In this study there is no direct evidence that health education increases the rate of testing, but it is interesting to note that a significant group of untested women were unaware of

cervical cytology as a preventive measure. It seems extremely unlikely that without such knowledge this particular group will come forward for testing.

There would, therefore, appear to be an important information gap which can only be filled by health education through the mass media or communication on the personal level; but, which is the more effective? Katz (1957) with support from many others has postulated that communication flows in two steps from the mass media to the public via "opinion leaders" and has stressed the importance of personal communication in helping to bring about change. The acceptance of a cervical smear test has been examined in the light of this approach (Rolfe 1961). He found that in the American setting the local primary access physician was in fact a very powerful "opinion leader" and through face-to-face conversation could influence a very great number of patients to come forward for the test. In the National Health Service, therefore, it would appear that the general practitioner could very well have an important or, indeed a key role to play, not alone in the carrying out of the tests but in educating the public. Apart, however, from the general practitioner it is obvious too, that the local authority clinics have an important contribution to make. Wakefield and Baric (1965) for example have suggested that older women prefer the anonymity of a clinic, and particularly so, when the test is carried out by a female doctor.

This brief report is part of a larger survey on public attitudes to cancer in Northern Ireland (Brown and Lynch, to be published). Some of the other findings of the survey might explain, in part, the reluctance of women to come forward for the test. Whatever the explanation is, the authors suggest that the role of health education in persuading women to be screened might usefully be further considered. It is only by raising the percentage of women tested that the laboratory facilities can be exploited to their full potential, and that a drop in the incidence and mortality of cancer of the cervix can be anticipated in the future.

Summary

In a sample of women aged twenty or over, interviewed in Northern Ireland:

(i) 23.2 per cent had been screened for cancer of the cervix.

(ii) 1 per cent of the single women had been tested.

(iii) 25 per cent of the smears were taken by general practitioners or local authority cervical cytology clinics.

(iv) The rates were lowest in the over forties and social class IV and V groups.

(v) A woman was more likely to have been tested by her general practitioner if she had lived in a town rather than in a rural area or Greater Belfast.

The impications for the programme of screening are discussed.

Acknowledgements

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We are grateful to Dr. R. P. Maybin for the use of the population register of the General Health Services Board. Thanks are also due to Professor J. Pemberton (of the Department of Social and Preventive Medicine, The Queen's University of Belfast) for his encouragement and advice, and Mrs. I. Hay for her help in data processing.

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TRIAMCINOLINE ACETONIDE (KENALOG) IN TREATMENT OF CASES OF HAY FEVER AND ITS EFFECT ON PITUITARY-ADRENAL AXIS

by

W. P. McMILLIN, M.B., M.R.C.G.P.

INTRODUCTION

Since 1949, when Hench and Kendall introduced the use of cortisone and hydrocortisone, a voluminous literature has accumulated concerning the employment of these agents in allergic diseases. Steroid therapy requires careful thought in view of the evidence that long term treatment can react adversely on the pituitary-adrenal axis. Nevertheless, an injectable steroid may be used in the treatment of chronic hay fever, and I have undertaken a trial in my own practice. In this small series of cases a study of the clinical effects, and a laboratory investigation of adrenal function are compared.

Hay fever is a hypersensitive state characterised by hyperactivity of the body to contact with a foreign substance in an amount which would not disturb a non-allergic individual. Allergy is a chronic and progressive disease, but hyposensitisation in some way alters the basic hypersensitivity and thereby helps to prevent the allergic symptoms from being multiple. Total avoidance of exposure to the incriminating allergen is usually impossible as it is airborne and inhaled, so hyposensitisation is the more practicable treatment. However, not every chronic case responds to this desensitisation programme, and in addition, it is time consuming for the patient and doctor alike, and new methods have to be explored which may be more efficacious.

Hay fever has been treated with a variety of adrenocorticosteroids. Much research has been performed to develop new adrenocorticosteroids which would possess greater biological activity and maintain an adequate ratio of glucocorticoid to mineralocorticoid activity. Alterations in the chemical configurations of the steroid nucleus and its chains produced triamcinolone. 16 – α – hydroxlyation markedly decreased mineralocorticoid activity and abolished the sodium retaining properties of 9 – α – fluorosteroids without destroying their glucocorticoid activity. It is one and a half to two times more potent than prednisolone. The greater therapeutic activity of triamcinolone is not accompanied by a corresponding increase in production of adverse effects. Triamcinolone Acetonide (the corticosteroid used in this trial) suppresses the clinical manifestation of hay fever and affords symptomatic relief. The mechanism of action is not entirely clear. In allergic conditions the union of antigen and antibody acts as a stress and either alone, or with the stress stimuli, e.g., psychogenic influences or infection, causes a liberation of histamine, acetyl choline, serotonin, and unknown substances which trigger allergic tissue responses. The exact mode of action is not clearly understood. It is suggested that they act locally upon the sensitised tissue cells, decrease the reactivity of the shock organs to specific antigens and stress and suppress local tissue responses rendering the cells less subject to injury. They increase permeability

and prevent formation of oedema and granulation tissue. Triamcinolone decreases oedema and increases the permeability of basement membrane. This is interpreted as being caused by the arrest of muco-protein breakdown and restoration of the normal state.

Design

The aim of this study was to test the efficacy and note any adverse reactions of triamcinolone acetonide following one 80 mg I-M injection in the gluteus maximus in patients with hay fever.

Patients included in the trial were between the ages of 25 and 45 and were of both sexes. They had symptoms of seasonal hay fever this year and also a history of severe seasonal hay fever over a minimum of three years which had responded poorly to other medicaments, e.g., antihistamines. Patients with a history of tuberculosis, hypertension, diabetes and duodenal ulcer were excluded. All patients were otherwise in good health and apyrexic.

The use of any exogenous steroid will affect the pituitary-adrenal axis. It was therefore decided to estimate cortisol levels at specific times following the injection. Each patient had cortisol estimated by withdrawal of blood from a vein in the antecubital fossa before the injection of 80 mg. triamcinolone acetonide was given intramuscularly in the gluteus maximus. Cortisol estimations were carried out at 24 hours, 48 hours, 7 days, 14 days, and 21 days after the injection. Response to injection was noted at each interview and patients were given a diary card to record symptoms of hay fever and/or side effects. Only patients who were willing and able to attend for repeated investigations at a fixed time – viz. 10 a.m., were studied. The time factor was considered very important as it is well known that cortisol levels vary along a 24 hours phasing scale under conditions standardised to eliminate stimulation other than daily routine.

The range of normal values of any body constituent, a time honoured standard, does not take into account the specific time at which the level was determined but rather allows for the existing variability of body function throughout the 24 hours. Circadian rhythms contribute largely to normal variations and this physiological regulation must be understood if disturbances of health are to be recognised and interpreted, and thus prevented or corrected. Depending on their time dimension, circadian rhythms either hinder or further studies in adaptive responses, and must to some extent dictate the experimental approach. Possibly the simplest approach is to attempt to eliminate their effect by fixed sampling times as is done in this series. The adrenal cycle is modulated by superimposed or juxtaposed endocrines and the interactions involved await further study. This field of study has a long history and has been reviewed repeatedly (Conroy and Mills, 1970).

The method of cortisol estimation used was that described by Mattingly (1962). The preparation under study was an aqueous suspension of triamcinolone acetonide, each ml. containing 40 mg of the corticosteroid with sodium chloride for isotonicity, 0.9 per cent benzyl alcohol as a preservative, and 0.75 per cent sodium caboxymethyl cellulose, and 0.04 per cent polysorbate 80 as excipients. 2 ml. of this preparation was administered intramuscularly to each patient of the series, injections being given into the gluteal muscle in every case.

Results

Clinical results were regarded as excellent in 12 of the 18 cases whose symptoms subsided rapidly, usually within 12 to 48 hours after injection, and did not reappear thereafter, so that no further supportive therapy was required. The clinical results were considered as good in five additional patients. In these patients there was marked symptomatic relief following the injection of triamcincolone acetonide although very mild symptoms recurred which required no further supportive treatment. One patient had a recurrence of his hay fever four weeks after the initial injection which necessitated further therapy. Response of three patients who in previous years had received desensitisation injections was dramatic and much appreciated, in that only one injection was required. The remaining 15 patients, who in previous years had antihistamines, were relieved since they were no longer drowsy at work. Side effects were negligible in the series. Three patients remarked on flushing of the face within 36 hours of injection.

The clinical benefits from this series compare favourably with the smaller series treated with two 80 mg. doses of methyl prednisolone acetate given at an interval of two weeks (Ganderton and James, 1970).

The cortisol estimations show depression of the pituitary adrenal axis within 24 hours. This lasted for varying times, but within three weeks the cortisol level returned to its initial level. Individual variations can be seen and response to emotional factors may explain the elevated levels in one case at two weeks in comparison with three weeks after the injection. The table shows the actual cortisol

Table

Cortisol Level mgs. per cent

Sex	*Age*	*Before injection*	*24 hrs. after*	*48 hrs. after*	*1/52 after*	*2/52 after*	*3/52 after*
M	39	10	4	3	3.5	4	9
M	28	21	9.5	4	2.5	3.5	17
M	31	10.5	8	5	2	3	10
M	28	21	9	5	2	21	31
M	34	13	9	5	5.5	9	27
M	40	9.5	2	2	7	9.5	10
M	32	15	6.5	5	9.5	9.3	17
M	32	17.5	5.5	4	9.5	9	14.5
M	45	11.5	4.5	2	3.5	9.5	13.5
F	26	7	3.5	2	5.5	8	9.5
F	28	19	14	9.5	8.5	12	13.5
M	35	7	5	2	7.5	12	16
M	35	13	9	5	7	9	11
F	26	9	2	2	5	9	10
M	27	15	8.5	3	7.5	11	13.5
M	25	11	4	2	4.5	9	10
F	26	14.5	7	4	9.5	9	13.5
F	28	17.5	6	2	7	9.5	16

levels obtained. This part of the study was thought to be of particular importance as any long-term influence on the pituitary-adrenal axis would be a definite drawback to the widespread use of triamcinolone acetonide injections for hay fever. It would appear from this study that recovery from adrenal suppression occurred in every case.

The small incidence of side effects may be attributed to the strict criteria used for selecting the personnel in the trial, and I feel that in the future, more use may be made of triamcinolone acetonide in this type of susceptible hay fever victim.

Summary

The use of a synthetic corticosteroid in the treatment of hay fever is described and the effects of the treatment on adrenal function are considered. It is sugested that treatment with triamcinolone can be of value to the susceptible hay fever sufferer.

The author is indebted to Mr. D. W. Neill, Chief Biochemist, Royal Victoria Hospital, in whose department the cortisol estimations were carried out, and who assisted in the production of this paper.

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BOOK REVIEWS

INFECTION IN HOSPITAL – A CODE OF PRACTICE. Edited by G. L. Gibson. (Pp. x+101. £1.25). Edinburgh and London: Churchill Livingstone, 1971.

This excellent little book should be read by all doctors, nurses and administrators who work in hospital and I am only sorry that it is presented in rather too lavish a format with stiff covers so that it is both too large and too heavy to carry in the pocket. It contains all those details which need to be known about preventing and dealing with infection in hospital which cannot be found easily in a textbook. It is the sort of information which should appear in the 'Hospital Handbook' which is issued to new house officers and which usually only contains normal biochemical values and details of how to do complex investigations. This book tells you how to deal with a case of typhoid, what antiseptics are to be used for cleaning lavatory seats and how to test a new autoclave. It contains an outline policy for the use of antiseptics and antibiotics in the hospital environment without building up any risk of virulent 'hospital' infections. What is most important is that the information is up to date and is applicable to current hospital conditions and not to some Utopia where all patients are in single isolable wards with their own toilet facilities and there is an ambulance of bacteriologically pure nurses.

The contents are brief, concise and dogmatic; most readers will disagree with a sentence or two here and there but few will disagree with the whole. It will need revision as hospital facilities improve and new antiseptics, antibiotics and methods of sterilisation become available. But the revision will only affect the detail: the principles behind this book have been tried and proved effective in hospital practice over the last 10-15 years. It is a fitting memorial for Dr. George Gibson's period of work as bacteriologist to the Belfast Group of Hospitals.

P.C.E.

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L.J.H.

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THE MENTAL HOSPITAL IN THE PSYCHIATRIC SERVICE – A CASE REGISTERED STUDY. By J. A. Baldwin, M.A., M.D. (Pp. xvi+340; graphs. £5.00). London: Oxford University Press for Nuffield Foundation, 1971.

This is an important book and should be compulsory, if somewhat heavy, reading for all those involved in planning psychiatric services. The word "heavy" refers to the detailed content with many statistical tables, and not to the writing style which is clear and readable. Although the studies described refer to the psychiatric services in North East Scotland this book is extremely relevant to N. Ireland today, while the Health Services are being re-organised.. After a brief review of the development of the Mental Health Services, with particular emphasis on Scotland, Dr. Baldwin goes on to describe the psychiatric case register method, which identifies persons and develops accumulative record in contrast to older statistics which give an index of events such as admissions or discharges, but do not identify persons. The methods of collecting the data for the register and the checks on the reliability of the data are described in detail.

In a review of this sort it would be impossible to attempt any sort of precis of the results of the studies described. Later chapters of the book report findings about the mental hospital population, the demand for hospitalisation and some detailed studies of what has been called 'the revolving door.' There are also chapters about the overall psychiatric service in the community, and the place of the mental hospital within that overall service.

Psychiatrists in N. Ireland will find much of interest in this book, particularly when it is considered in relation to the N. Ireland Mental Health Records Scheme.

CALLING THE LABORATORY. Edited by W. A. R. Thomson. Third Edition. (Pp. vii+147; figs. 5. £1.50). Edinburgh and London: Churchill Livingstone, 1971.

This is the third edition of a book whose value is reflected by the excellent reception given to the first two editions. It provides a guide to general practitioners in the laboratory investigation of disease. The indications for various laboratory tests are described, as well as the method of obtaining a suitable specimen and a discussion on the interpretation of the result. Despite being quite a small book, it has thirty contributors. It originated from a series of articles which appeared in the Practitioner and which have been brought up to date in this new edition.

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