VOLUME XXXV, 1966

THE ULSTER MEDICAL JOURNAL

PUBLISHED BY
THE ULSTER MEDICAL SOCIETY

The Ulster Medical Journal

VOL. XXXV

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1966

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Fellows and Members of the Ulster Medical Society receive the Journal Free. Details as to subscriptions on back page.

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THE ULSTER MEDICAL JOURNAL

NOTICE TO CONTRIBUTORS

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

2. Manuscripts should be typewritten with double spacing and with wide margins. They should be fully corrected, and contributors will be responsible for the payment of any sum charged for alteration in printer's proof in excess of ten shillings per sheet (16 pages).

3. References should be given as in the publications of the British Medical Association and in most other modern British journals. If contributors are uncertain of the correct abbreviation for the title of any journal, as given in the World List of Scientific Periodicals, they should give the full title.

4. Tables and illustrations should be reduced to a minimum, and should be sent detached from the typescript of the paper. Line drawings should be used whenever possible. All illustrations must be in a finished form ready for reproduction. Authors will be charged for all blocks at cost price. Half-tone blocks require to be printed on special art paper. Authors will be responsible for the cost.

5. Order for reprints must be given when the author returns the printer's proof. The cost of these may be obtained from the printers in advance.

6. Editorial communications should be sent direct to the Editor, Dr. J. E. Morison, D.SC., M.D., The Laboratory, Belfast City Hospital. The Editor will be glad to advise authors on the preparation of their manuscripts.

ADVERTISEMENTS

First advertising forms go to press thirty days in advance of the date of issue. In forwarding copy, time must be allowed for setting up and submitting proof. All communications for space must be sent to:

H. A. CRICK, 6a Victoria Avenue, Whitehead, Co. Antrim.
Telephone: Belfast 24807.

Mr. Crick has been appointed the sole representative of The Ulster Medical Journal.

DATES OF PUBLICATION

It is hoped to issue a Winter and Summer number each year in February and September.

REPRODUCTIVE PHYSIOLOGY OF THE POST-PARTUM PERIOD. By Albert Sharman, M.D., D.Sc., Ph.D., F.R.C.O.G., F.R.C.S. (Glasg.). (Pp vii + 127; figs. 87. 25s.). Edinburgh and London: E. & S. Livingstone, 1966.

THE title of this monograph is too broad for its limited though valuable approach. The approach is that of morbid anatomy and clinical observation and the term "physiology" is justified only since it is an attempt to define the normal changes of the post-partum period. Essentially these studies are limited to a histological study of the endometrium from soon after delivery till nine months thereafter; the re-establishment of ovulation as indicated by the appearance of the secretory phase of the endometrial cycle and the date of re-appearance of menstruation and its subsequent rhythm.

The basis of the work is 626 endometrial biopsies from 285 women from the fifth day to nine months after parturition and all free from puerperal infection. Of these women 230 had one biopsy only. The remainder had from 2 to 24 biopsies and in these the normality of the later biopsies might be questioned. However the author has an extensive series of biopsies, which he seems to have carefully correlated, and which enables him to describe regeneration of the endometrium following childbirth. Cellular reaction as indicated by the presence of plasma cells is described as late as the third and fourth months in over one-third of the cases. The earliest appearance of secretory endometrial patterns, taken as presumptive evidence of ovulation, was noted during the seventh week.

This is a useful study presented in detail and with an over abundance of microphotographs, many of which show little distinctive change. It would seem that the work in a shortened but unimpaired form might have graced a learned journal. Elegantly presented and beautifully printed and bound as, indeed, one expects from the publishers, it is moderately priced and should be included in the library of all interested departments. J.E.M.

NOTICE

This is volume 35 of the *Ulster Medical Journal*, and the usual two parts are combined. It is the only issue dated for the year 1966. In future, for reasons relating to the dates of delivery of Presidential and other addresses and to difficulties in printing before Christmas, the Journal will be published in late February and late September and the two numbers will be dated as Winter and Summer.

It is regretted that the last issue dated December 1965 was incorrectly described as volume XXXV, No. 2. It was volume XXXIV, No. 2. The volume number on the contents page of the last volume was also incorrectly given.

THE ULSTER MEDICAL SOCIETY

P.O. Box 222.
Belfast City Hospital,
Belfast, 9.

Dear Sir (or Madam),

If you are not a member of the Ulster Medical Society, we would appeal to you to give the question of joining your consideration. The Society has been in existence since 1862, and has always been active in keeping its members interested in the advances in medical science. Meetings are held at intervals of a fortnight during the winter months, and papers are contributed by members and distinguished guests. Facilities are provided for doctors to meet informally afterwards and have a cup of tea. *The Ulster Medical Journal, the official organ of the Society, is issued to all Fellows and Members free of charge.*

May we, therefore, appeal to you to join the Ulster Medical Society, and so enable us to widen its influence and sphere of usefulness still further? A proposal form is appended: your proposer and seconded must be Fellows of the Society.

If you do not wish to become a member of the Society, will you consider entering your name as a subscriber to THE ULSTER MEDICAL JOURNAL? The subscription is fifteen shillings per annum, payable in advance to the Honorary Treasurer.

We remain,

Yours faithfully,

J. S. LOUGHRIDGE, *President.*
D. MILLAR BELL, *Hon. Secretary.*
W. BINGHAM, *Hon. Treasurer.*

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FELLOWS.—1 (a) Annual subscription of Fellows resident, practising or holding an appointment within ten miles of Belfast, **£3. 3s.**; (b) husbands and wives in the above category who are both Fellows will be entitled to pay a combined subscription of **£4. 4s.**; 2 (a) annual subscription of Fellows resident, practising or holding an appointment outside the above area, **£2. 2s.**; (b) husbands and wives in the above category who are both Fellows will be entitled to pay a combined subscription of **£3. 3s.**; 3, annual subscription of retired Fellows, provided that any Fellow who, by reason of retirement either through age or illness, is no longer engaged either in private practice or in salaried employment, shall be entitled, *on application,* to pay an annual subscription of **£1. 1s.** only, and provided always that such Fellow has previously paid to the Society a subscription at the current rate for an uninterrupted period of at least ten years, or during such time has been in practice or service abroad.

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To DR. W. BINGHAM,
7 STRANGFORD AVENUE,
BELFAST, 9.

.....19.....

Dear Sir,

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Name of Candidate .....

Postal Address .....

.....

Year of Qualification and Degrees .....

.....

Signature of Proposer .....

Signature of Seconder .....

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NORTHERN IRELAND.

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THE
ULSTER MEDICAL JOURNAL

PUBLISHED ON BEHALF OF THE ULSTER MEDICAL SOCIETY

Vol. XXXV 1966 Nos. 1 and 2

FIFTY YEARS—A RADIOGRAPHIC RETROSPECT

By RALPH M. LEMAN, M.B.E., F.S.R.

ONE DAY Dr. R. S. Allison, who is the Archivist to the Royal Victoria Hospital, suggested that I put down on paper some memories of my experiences as a Radiographer and my recollections of the history of the X-Ray Department in the Royal Victoria Hospital, Belfast. The 'Royal' is so known to the entire population of Belfast and so I will continue to call it in this paper. The X-ray department and I grew up together, and I belong to that group of pioneers who, like the pioneers in any new technique, learnt my job by experience, for in those days there was not and could not have been any official training or any recognised qualification. It was perhaps the absence of any official qualification that encouraged me in my later days to go for so much competition work, but I feel that to leave out these aspects of my life would lessen the interest of what I have to say.

I am an East Anglian and my home town is Norwich, where in the exciting period of 1914-15 I was studying for the Minor Pharmaceutical examination. I was also a local member of the Red Cross Society and used to assist with the admission of war casualties at the Norfolk and Norwich Hospital where I spent some time in the X-ray department.

Early in 1915 I volunteered to serve abroad as a Red Cross worker and soon found myself in France at a hospital in Wimereux near Boulogne. This was a converted hotel and was known as Lady Hadfield's Anglo-American Red Cross Hospital. Having an elementary knowledge of X-ray and pharmacy I was appointed to take X-rays and dispense stock medicines. My main interest was undoubtedly X-ray work, although the apparatus available was primitive, consisting of a 24 inch induction coil with a hand cranked mercury interruptor, operated like an egg beater, while power was obtained from a bank of accumulators. Speaking of mercury interruptors, I still remember when I ruined a gold signet ring given me by my parents when leaving for France. Mercury in the interruptors got very dirty with use and required to be frequently cleaned by squeezing it with the hands through a cloth or rag. Once when carrying out this procedure I forgot to remove my gold ring, but the mercury did not and formed an amalgam with it. All my efforts failed to remove the mercury, resulting in the ring breaking into six pieces a few days later. We used gas X-ray tubes and great care was necessary

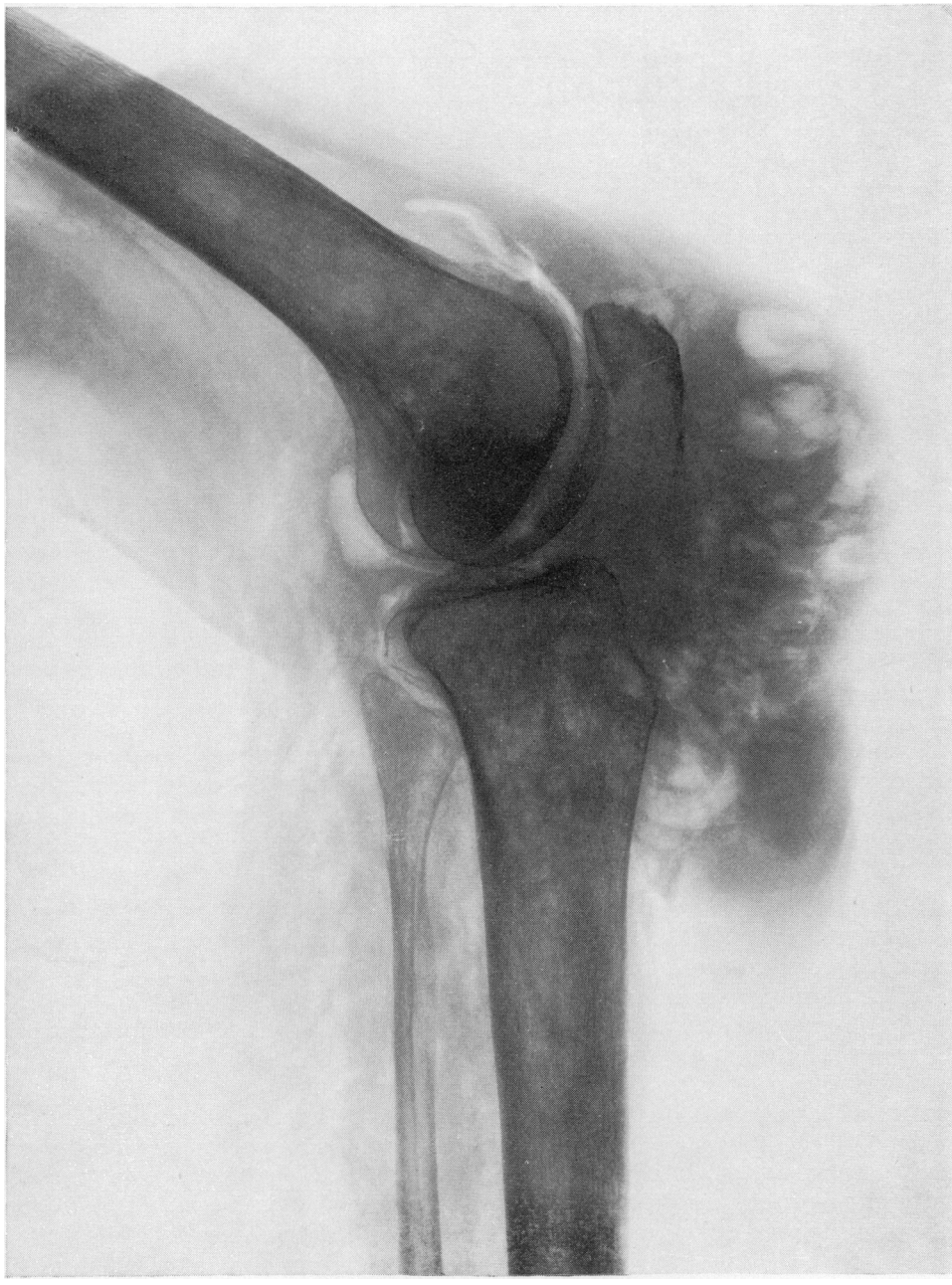

FIG. 1. *Gas Gangrene of Knee and Leg. First World War*

to avoid overloading them, otherwise they would be useless due to the vacuum being reduced. Films had not been thought of for X-ray work and we used glass plates which had to be developed in dishes in a bathroom. Pyro-Hydroquinone developer was used for processing the negatives and so skiagraphers, as we were known in those days, bore the trade mark of brown stained finger nails due to handling the plates in this solution. The plates were propped up on pieces of wood in the bath for washing. Figure 1 shows gas gangrene in the leg and was taken in 1916.

Exposure times even for extremities were long, and the odd barium meal was attempted but with little success, for the patients had difficulty holding their breath for the exposure time required, about 30 seconds, and we had difficulty hand cranking the interruptor at a constant speed. Later barium-platino-cyanide intensifying screens became available which certainly did reduce exposure times, but the screens at that time had one big fault, namely 'lag' and it was necessary to run outside to expose the screen to daylight when changing plates in the wood cassette, otherwise the latent image of the first exposure would be superimposed on the next one taken. Little was known of the radiation hazard in those days and we occasionally screened our hands to test the output of the X-ray unit and tube. Our greatest concern was to avoid electrical shock from the bare high tension wires suspended from the ceiling and connecting the induction coil with the X-ray tube.

I was fortunate in working with many eminent surgeons and physicians at the Anglo-American Hospital, including Lord Dawson of Penn and Major Valadier, Consultant Oral and Facial-maxillary Surgeon to the B.E.F. It was here that I met Colonel Andrew Fullerton, Consultant Surgeon, A.M.S., and John Campbell, F.R.C.S., who was attached to the British Red Cross Society, both of whom were from Belfast.

Before leaving the Anglo-American Hospital for home at the end of the War Colonel A. Fullerton and Mr. John Campbell suggested that I might like to do X-ray work in Belfast, to which I readily agreed. Arriving home in Norwich and hearing nothing from Belfast I reluctantly took up my pharmaceutical studies again, but in April 1919 a telegram arrived instructing me to report to Dr. J. C. Rankin, electrical department, Royal Victoria Hospital. On receipt of this welcome news I threw my pharmaceutical books away and departed for Belfast, arriving Easter Tuesday 1919. I found the X-ray room situated on the first floor of the King Edward Building of the hospital and under the charge of Dr. Rankin and Sister Miller. The X-ray equipment costing less than £500 was most impressive in appearance and the noise generated, but had a low output. So called flash exposures were made by the dropping of a weight down a tube, the exposure being controlled by the distance of the weight dropped. Glass X-ray plates and gas X-ray tubes were still used, but the patients requiring X-ray only numbered 6 to 8 per day.

By 1920 X-ray equipment with increased power and output became available. (Figure 2). A special alternating current mains was supplied to the X-ray room as most of the hospital electrical supply was direct current. This A.C. mains permitted the installation of the latest type of wax insulated high tension transformer which greatly improved radiographic results. It was, in fact, the first X-ray

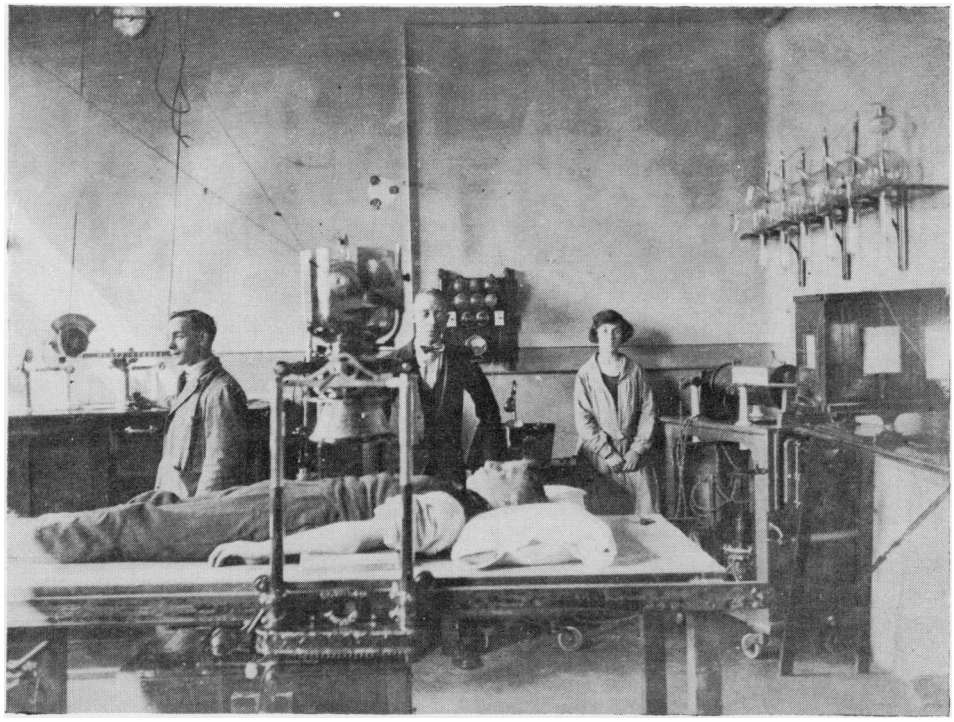

FIG. 2. *Royal Victoria Hospital. Only X-ray Room in* 1920

transformer to be used in the Royal. A mobile induction coil X-ray unit was also purchased for radiography of patients in bed when limbs were suspended in a Thomas splint and gantry. All X-ray apparatus was still non-shockproof with bare wires conveying voltages of up to 70,000 from the transformer or coil to the X-ray tube, so that to do a ward portable in those days was often quite an adventure, sometimes painful, for both radiographer and patient. The radiation hazard was by now being realised and lead rubber protective aprons and gloves were available, but radiographers had to be physically strong to work with those heavy lead aprons slung around their necks or to use their hands after struggling to don the very stiff gloves. The amount of work and demands on the department gradually increased and in 1921 Dr. Maitland Beath was appointed assistant radiologist to Dr. J. C. Rankin.

In 1921-22 Belfast was troubled by political riots and for a time a strict curfew was imposed from 11.30 p.m. to 5 a.m. and being frequently called to the Royal during the night to X-ray riot casualties it was necessary for me to obtain a night pass from the police and a permit to use a motor cycle, my only means of transport at night. These journeys were far from pleasant, particularly on the Grosvenor Road, with occasional bullets coming from side streets and from the park opposite the Royal. If on reaching the gates of the hospital I found them closed it was safer to keep riding round the road in circles until the night porter would dash out to open them.

Until 1921 no official body was recognised for the training and examination of radiographers, who were mostly men who had a technical knowledge of X-rays. It was from this group that the Society of Radiographers, London, was established and in 1922 I was successful in gaining the Diploma of that Society.

The year 1922 saw three developments which revolutionised radiography: the old gas X-ray tube with all its problems of maintaining the correct vacuum was replaced by the Coolidge hot cathode tube, and double coated X-ray films replaced the single coated X-ray plates, while later the Potter-Bucky diaphragn was invented, a device to reduce the scattered radiation reaching the film. The Royal was the first hospital in Northern Ireland to instal this piece of equipment, although Dr. Beath did instal a Bucky diaphragm at "Elmwood" just before this. Its arrival created great interest. Dr. Beath and I spent a whole evening taking X-rays of our spines and pelves. I well remember the thrill and satisfaction of seeing for the very first time clear radiographs of the heavier parts of the body without the usual blurring caused by scattered or secondary radiation.

During 1923 the number of patients X-rayed was in the region of 4,800 and the steadily increasing demands for X-ray examinations necessitated moving the X-ray department to the main floor of the hospital, so providing accommodation for a second X-ray unit.

In 1926 it became very apparent that more people were required for the taking of X-rays in the Royal and that they should be qualified according to the Society of Radiographers standards. During 1926 I accepted and trained a male student, my first student to qualify. Now many of the radiographers whom I trained hold senior posts in the Royal, throughout Northern Ireland, and abroad.

In 1927 Dr. F. P. Montgomery, M.C., was appointed assistant radiologist.

It was some time in 1927 that Dr. J. C. Rankin arranged for a mobile X-ray unit, a coil unit, to be transported and set up in the yard of Mr. Ewing Johnston, a veterinary surgeon of May Street, Belfast (Figure 3). Our mission was to X-ray the leg of a well known racehorse, 'Jerpoint', a most unhelpful patient, for every time we attempted to take the X-ray he backed away, due to the electrical discharge from the bare high tension wires. Eventually, with his back end against the wall, the examination was successfully carried out and a fractured shin bone diagnosed.

On another occasion Dr. Rankin, always interested in the unusual X-ray, arranged for a horse with a slight leg injury to be walked to the Royal for an X-ray. It was a Saturday evening in summer and instructions were given for the horse to be brought in by the lower gate and round to the old workshops at the back of the hospital, where a portable X-ray machine was set up. After waiting half an hour with no sign of the animal, I went round to the front of the hospital, when to my horror I saw a man trying to take the horse into the hospital through the extern door, much to the amusement of spectators gathered at the gate lodge. The man in charge of the horse insisted that he was acting on instructions, but the sister in charge of extern was equally determined that no horse was coming into her extern. My timely arrival saved the situation and the horse was led away to the appointed place.

It was in the early thirties that I first considered entry to the various radiographic essays, competitions and exhibitions, realising that work of this nature would

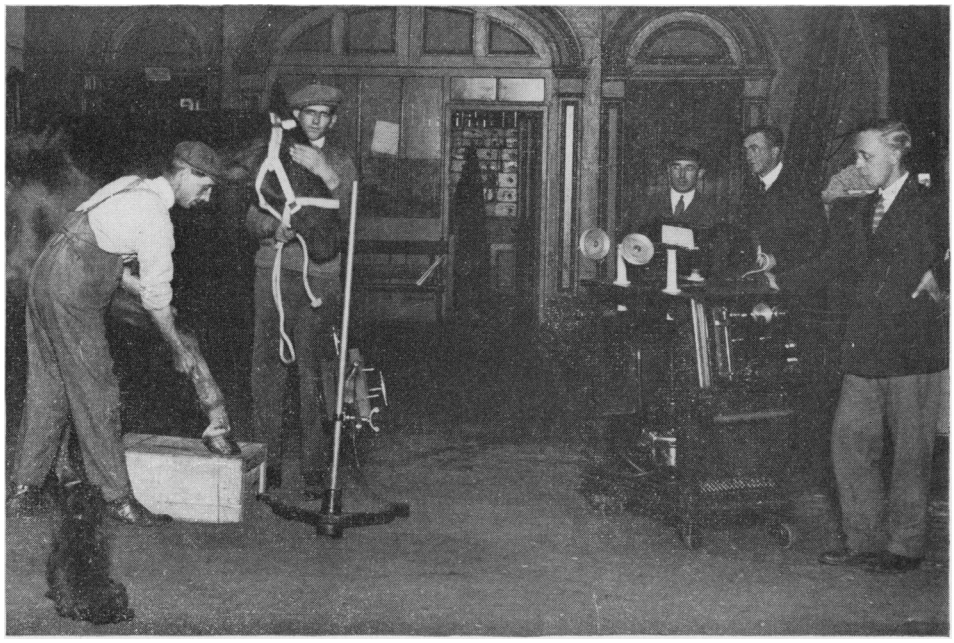

FIG. 3. *Veterinary radiography by Dr. Rankin and R.M.L.*

increase my professional knowledge. In 1931 and 1933 I gained the Archibald Reid Memorial Medal of the Society of Radiographers for theses on radiographic techniques, and am the only radiographer to have two such medals, while from 1934 to 1941 I exhibited, by invitation, medical and technical radiographs in Stockholm, Czechoslovakia, Rochester and other cities in the U.S.A. In 1935 the Western International Salon invited me to contribute to an album of photographic art for presentation to King George V and Queen Mary. But the two distinctions which gave me the greatest pleasure were my election in 1936 to the Foundation Fellowship of the Society of Radiographers and the recognition of my exhibits in 1941 by the award of the Rodman Medal of the Royal Photographic Society of Great Britain.

During the years of the 1939-45 war it was somewhat difficult coping with all the radiographic demands. In addition to being called to the Royal when air raid alarms were sounded it was, because of the reduced medical staffs of the Royal, necessary for the radiographers to work longer daylight hours and postpone the X-ray work associated with Smith Peterson pins, ventriculograms and other surgical procedures until night time. Although we frequently worked twelve hours or more during the war, life was not without amusing incidents. One of these concerns an emergency telephone call to my house one Sunday morning from a nursing home, at which a Belfast surgeon, Mr. J. S. Loughridge, was attempting to remove kidney stones. Being unable to locate them he requested my help. Following a hurried drive to the Royal to collect a portable X-ray machine,

developing dishes, chemicals and small films, I made for the nursing home and set up the X-ray machine on a theatre trolley and two X-rays were taken. Then I had difficulty finding a dark room or cupboard in which to develop the films, but eventually it was suggested that the matron's bedroom had the best blackout in the building. Even this was not dark enough, so bedclothes were draped around the bed to the floor. I then crept under the bed with my dishes and chemicals, instructing a nurse to wait outside the door and knock when five minutes had elapsed because I was working in complete darkness. From the films taken the stones were located and successfully removed. The matron was at church.

During half a century of radiography one would expect and indeed welcome unusual and difficult cases, for not only do they provide a break from routine work but extend one's knowledge of radiographic technique. The following are some of the interesting investigations I have undertaken. On leaving a breakfast table a mental patient informed the hospital nurse that she had swallowed a spoon. Examination of the patient's throat and patient's clothing revealed no spoon. So the nurse duly reported the matter to sister and so on to the doctor and medical superintendent who, although no clinical signs were present, thought an X-ray advisable and requested me to carry out the investigation, with apologies for troubling me as the story appeared so ridiculous. However X-ray examination did reveal a dessertspoon in the stomach. The patient was admitted to the Royal for operation and the spoon successfully removed.

A man with every appearance of being ill arrived at the X-ray department with a history of having swallowed his false teeth during the night. Although the distressed condition of the patient gained him much sympathy from other patients, X-ray of the whole gastro-intestinal tract failed to show any sign of the teeth. His wife then arrived in the department with the missing denture which she had found in his bed. I never saw a patient recover so quickly.

During the first World War I noticed a Frenchman wearing abnormally large shoes. After being tactfully questioned he agreed to submit to an X-ray of his feet. The radiograph revealed that he had six metatarsals and seven toes on each foot.

Some years ago the late Mr. Cecil Calvert walked into the X-ray room at the Royal with a chest X-ray showing a screw in the main bronchus. Then producing six iron screws he asked me to pick out the one of corresponding size to that shown on the radiograph, as the size of the screw would determine the bronchoscope necessary for its removal. The unknown degree of magnification on the film made a snap decision impossible. However, I endeavoured to assist by using the following procedure. Placing the patient supine on the X-ray table so that the posterior chest wall was in close contact with a double wrapped X-ray film, the distance from the film to the anterior chest wall and the tube film distance was noted. Stereo exposures were then made on one film from which the exact distance of the head and point of the screw from the posterior wall was calculated; a lateral film confirmed the degree of obliquity of the screw. Using a long loaf of bread as a phantom it was placed on its end and cut the same height as the distance from the posterior chest wall to the screw and four of the most likely screws were placed on it. From X-rays taken of the loaf with four screws on it and further calculations the identical size of screw with that in the bronchus was determined. This was confirmed after operation.

Walking along a beach I was impressed by the pattern and beauty of the seaweed lying around and wondered how it would look if X-rayed, so I collected some and made the experiment. This success prompted the thought of X-raying plants and flowers to show the delicate leaf structure and bud formation. To produce radiographs of the correct density, contrast and artistic appearance, it was necessary to experiment with many technical factors as there was virtually no latitude; these required to be varied with almost every exposure. Selection of

FIG. 4. *X-ray of Lilium longiflorum*

FIG. 5. *X-ray of Magnolia*

suitable subject matter and improved techniques resulted in radiographs suitable for exhibition purposes (Figures 4 and 5).

The Medical Officer of a local circus asked me to X-ray one of his company, a giraffe neck woman who was a native of the Shan State, Burma, to demonstrate the bone structure and contour of the neck. This lady's unusual appearance created quite an interest when passing through the extern of the Royal. It was not possible to show the cervical vertebrae because of the radiopacity of the twelve or so solid brass coils around her neck. However, a form of collar of the same material extended down and out to rest on the shoulders, this could be raised so making it possible to X-ray the shoulders and upper chest. It was found that the giraffe neck effect was really produced by the continued weight of the heavy collar depressing the shoulder girdle with compression and elongation of the upper thorax.

But now I must return to the hospital department when the number of patients requiring X-ray had now reached such proportions that extension was very necessary. Plans were prepared for a completely new department at an estimated

cost of £30,000. However, because of the war, this project was deferred and additional X-ray rooms had to be sited in any available rooms, including the King Edward Building.

In November 1940 the Royal Victoria Hospital and Radiology suffered a great loss by the untimely death of Dr. Maitland Beath. I treasure the memory of twenty years' happy and close association with Dr. Beath. His kindly friendship and sincere consideration for all hospital personnel whatever their rank or station inspired loyalty and affection. Dr. F. P. Montgomery was appointed radiologist-in-charge after the death of Dr. Maitland Beath.

By 1949 my time was fully occupied for, in addition to being superintendent radiographer at the Royal, I was organising secretary and lecturer to the now officially recognised Radiography Training School. Also as adviser in radiography to the Northern Ireland Hospitals Authority I made a survey of all X-ray departments in the Province and put forward proposals for short and long term improvements. One very interesting project was that for the Belfast City Hospital whose Management Committee asked me to plan the conversion of a large ward to two X-ray rooms and ancillary rooms.

By now I was one of the oldest members of the department and it was a very great honour when in 1951 I was awarded the M.B.E. I feel that this reflects on the prestige radiography had by that time attained, not only in the Royal Victoria Hospital but all over the North of Ireland where so many of our old students were in post.

In 1954 the many friends of Dr. J. C. Rankin were grieved to hear of his death some ten years after his retirement. Dr. Rankin had a thirst for knowledge, to further which he spent some months in Copenhagen to study the treatment of skin diseases and later to Vienna to acquire a knowledge of X-rays. Johnny Rankin, as he was affectionately known by his colleagues, possessed the gift of friendship, had outstanding versatility and technical ability and sought neither office nor preferment. His name will long be remembered by those who were privileged to know him.

By 1956 the number of radiographers in Northern Ireland was sufficiently great to justify the formation of a Branch of the Society of Radiographers. As father of the flock I was the first Chairman and also became the Branch Nominee on the London Council.

The work of the department had now increased to the point when an entirely new department became necessary and inevitably I was closely associated with the planning. Nine X-ray rooms were to be provided in the first stage.

I officially retired from the Royal in June 1959 after forty years' service, but was invited by the Belfast Hospital Management Committee to act in an honorary advisory capacity and so was able to see my old X-ray department into its new home. Retirement does not necessarily mean that a man stops working and I assisted and advised radiologists, architects and contractors with the planning of X-ray departments at Altnagelvin Hospital, Londonderry, the Ulster Hospital, Dundonald, and the Dental Hospital, Royal Victoria Hospital. Perhaps it is little wonder that in 1960 I had to give an account of myself to the B.B.C. and explain how X-ray work had grown with the years and how it had been applied to the service of mankind.

Maybe my friends thought that the old man was then finished and that it was time to close down, and in that same year I was awarded the Honorary Fellowship of the Society of Radiographers of which I was a Foundation Fellow. But there was still work to be done. In July 1962 the British Medical Association meeting was held in Belfast, at which I was invited to exhibit in the Scientific Section by the President, Sir Ian Fraser, who was my sponsor, and in 1963 I was invited by the Council of the Society of Radiographers to deliver the Stanley Melville Memorial Lecture at the British Institute of Radiology, London. I chose as my subject "Thoughts on Function and Design of Diagnostic Departments".

In 1965 the Society of Radiographers held their Annual Conference at Portrush, Northern Ireland. I was particularly pleased that the Council thought fit to honour us thus and during my term of office as Chairman. This conference coincided with my fifty years of active association with radiography and was in fact my "Swan Song", for having been Chairman of the Northern Ireland Branch on two occasions and Branch Nominee for nine years, I felt that for the good of the Branch and in fairness to younger members I should resign my position of office. Following this one of the greatest tributes to my fifty years of X-ray work was paid me by the members of the Northern Ireland Branch, for on September 8th 1965 I was their guest of honour at a dinner in the Woodbourne House Hotel, and was presented with a tape recorder and cheque. For me it was a memorial evening, over eighty radiographers from all parts of Ulster were present and colleagues of earlier days came over from Surrey. The occasion was graced by the President of the Society of Radiographers. The centre piece on the top table was a large iced cake on which was inscribed "Fifty Years and still Radiating". It was indeed a wonderful climax to a long and happy career and I was deeply touched by the friendship and affection of my fellow radiographers.

I cannot close this account of my career in radiography without expressing my gratitude for my happy and encouraging associations over the years with the medical staff of the Royal Victoria Hospital and indeed with many medical friends throughout Northern Ireland.

> May the younger generation profit by the experience
> of their predecessors who had to learn the hard way.

My thanks are due to Dr. R. W. M. Strain for his interest and help.

THE DEVELOPMENT OF GASTRIC SURGERY

By PROFESSOR JAN NUBOER
University of Utrecht

The Sir Thomas and Lady Edith Dixon Lecture delivered in the Royal Victoria Hospital, Belfast, on 24th February, 1966

CONTEMPORARY gastric surgery developed within a relatively short period during the latter half of the previous century, after the work of Pasteur and Lister had eliminated the grave risks entailed by a laparotomy. They paved the way to the development of gastrointestinal surgery. It was the Viennese professor Theodor Billroth and his pupils who accomplished the enormous advances on which today's gastro-intestinal surgery is based. Billroth was the great pioneer of visceral surgery, and teacher of a large number of prominent surgeons, many of whom became professors of surgery in various countries of the continent.

When in 1890 the Medical Faculty in Utrecht decided to pump new life into surgical teaching at our University, the board approached Billroth with a request that he send one of his pupils to occupy the chair. In 1890 F. A. Salzer—probably the most gifted of Billroth's pupils—was appointed professor of surgery in the University of Utrecht. Unfortunately he died as early as 1893, whereupon the Faculty again approached the Viennese master. From 1893 to 1896, the chair in Utrecht was occupied by Anton, Freiherr von Eiselsberg; he was called to Königsberg in 1896, and from there went to Vienna. After Von Eiselsberg's departure, Billroth's last pupil A. Narath was appointed professor of surgery. He remained in Utrecht from 1896 to 1906, when he accepted an appointment at Heidelberg University. In 1906 H. J. Lameris was appointed his successor; he was a pupil of Von Eiselsberg and Narath, and my teacher. Until 1943 he occupied the chair to which I succeeded him. In view of these historical notes it is not surprising that abdominal surgery, and especially surgery of the stomach and duodenum, has always received considerable attention in the Utrecht University Surgical department. Nor is it surprising that in this department the name of Billroth—who may be considered the spiritual great-grandfather of the present incumbent—is held in very high esteem.

While the technique of gastro-intestinal operations attained a definitive level during the early years of this century, the surgical treatment of gastric and duodenal ulcers has shown a gradual evolution. New procedures have been introduced in the past 15 years, and if we are to understand the value and significance of all these procedures, we must consider the historical perspective.

Although with the introduction of antisepsis, and later of asepsis, a laparotomy was not longer something like an attempt on the patient's life, it is more or less obvious that the first gastric operations were performed in the treatment of fatal affections. After Billroth performed the first successful partial gastrectomy for a pyloric carcinoma in January 1881, his pupil and assistant Wölfler was the first, in September of that year, to be confronted at the table with an inoperable carcinoma of the pylorus. His assistant during the operation—Nicoladoni—advised him to establish a communication between the distended stomach and the small intestine: thus the first gastro-jejunostomy was performed. This gastro-jejunostomy was established antecolically and anisoperistaltically; in spite of its technical imperfec-

tions, it yielded an excellent result. In subsequent years, therefore, this operation was performed for all types of pyloric stenosis. But soon it became clear that this procedure can have serious disadvantages and that the food can be propelled, not into the efferent, but into the afferent loop, giving rise to what we call a vicious circle. For this reason, Lauenstein in 1891 suggested that an anastomosis be made between the afferent and the efferent loop, and in the course of the following year Jaboulay was first to perform this operation. The type of entero-anastomosis to gain widest acclaim, however, was that introduced by Braun in 1892. Innumerable modifications of gastro-enterostomy have been described, but the anterior gastro-enterostomy caused so many complications that establishment of a posterior retrocolic gastro-jejunostomy came to prevail. This was first carried out by Czerny in 1883 but, surprisingly, this technique has become known by the name of Von Hacker—the author of the first publication to describe this procedure, in 1885.

In view of the successful use of gastro-enterostomy in pyloric stenosis, and in view of the difficulty of differentiating between benign and malignant processes, gastro-jejunostomy was soon used also in the treatment of benign pyloric stenosis. The results were impressive, even in stenoses caused by peptic ulceration. As the understanding of peptic ulceration began to improve, these successes led to the erroneous conception that gastro-enterostomy could have a curative effect on ulceration of the stomach and duodenum. At the same time, advancing studies of the chemistry of gastric juice disclosed that in these conditions, particularly in duodenal ulcer, the gastric juice showed much higher acidity than in normal subjects. It was assumed, for sound reasons, that the development and persistence of these ulcers must be ascribed to the influence of the peptic factors in gastric juice on the wall of the stomach and duodenum.

When the results obtained in benign pyloric stenosis continued to be excellent, and when later postmortems invariably disclosed only ulcer scars, and no active ulcers, it was believed that the open communication between stomach and intestine ensured free entry of intestinal juice into the stomach, where it could neutralize the gastric juice. It was said that the "internal pharmacy" was put to work in this way. On the basis of this view, the indications for gastro-enterostomy in duodenal ulcer were extended more and more. In cases of gastric ulcer it was soon found that the results were not so favourable.

Instead of confining gastro-enterostomy to old, extinguished, healing ulcers which had given rise to cicatricial stenosis, and in which the acidity of the gastric juice had been reduced by atrophy of the mucosa, the same operation was also carried out in the treatment of more active ulcers in an earlier stage. Admittedly, this operation had a favourable effect in many of these cases, and it was maintained as a standard procedure until shortly before the last World War. Moynihan and William Mayo were past masters of gastro-enterostomy, and they would not be convinced that other modes of treatment might be better. In those days, however, an indication for operation was less readily accepted than it would be later, and the majority of the patients had a long history of ulceration; certainly their ulcer was no longer in the active stage when they reached the operating room.

As gastro-enterostomy became a procedure used on a large scale in the treatment of active processes, in which the peptic properties of gastric juice were still fully active, it became increasingly clear that it was not only possible for intestinal

juice to flow into the stomach, but also for gastric juice to flow into the intestine. The intestinal wall proved to show poor tolerance to the effects of gastric juice, and jejunal ulcers were observed more and more frequently. Although as a rule these jejunal ulcers were not observed until years after the gastro-enterostomy, their frequency was found to be so high (Tanner: 50 per cent.) as to throw doubt upon the correctness of performing this operation in cases of active ulceration. It is nevertheless an established fact that, in benign pyloric stenosis in an advanced stage of ulceration, the operation can yield excellent results.

The first partial gastrectomy for ulceration was probably performed by Van Kleef—a Dutch surgeon from Nijmegen. But it was not until the time of the first World War that this operation for peptic ulcer attracted wider attention under the influence of men like Von Haberer in Germany, and Strasberg and Lewisohn in the U.S.A. It took many years, however, before the operation was generally accepted. As late as 1927, a prominent American surgeon told Lewisohn after a paper read before a meeting of the American Medical Association: "If anybody wanted to cut out half of my good stomach in order to cure a little ulcer in my duodenum, I would run faster than he"—a remark which drew applause. Nevertheless the operation has gained more and more ground, and until recently it has been regarded as the operation of choice for duodenal and gastric ulcers.

What was the actual principle underlying resection therapy for peptic ulcer? As experience increased, it became increasingly clear that gastric juice is of preponderant importance in the aetiology of these ulcers. All pathological data indicate that "ulcus ventriculi et duodeni" is truly a peptic ulcer, formed as a result of the action of gastric juice upon the mucosa of the stomach and intestine. The ulcer is found exclusively at sites where gastric juice can exert its influence, that is: in the stomach, duodenum, in the oesophagus in the case of reflux, in the jejunum after gastro-jejunostomy and in the ileum if a Meckel's diverticulum contains gastric mucosa. As early as 1910, Schwarz expressed the general opinion in the aphorism: "no acid, no ulcer", and the very latest of modern investigations have failed to disprove this view.

Why then should one perform a gastric resection which removes the pyloric part and leaves intact the fundus, in which the acid-forming cells are localized? During the years after World War I, the physiology of gastric digestion was only superficialy understood. While preparing this paper, I read once again the chapter on gastric digestion in my old textbook of physiology, written by my teacher, Zwaardemaker. I was surprised to see just how limited the knowledge in this field still was in about 1918. It was known that, apart from digestion, the stomach secretes virtually no gastric juice. It was also known, from the investigations of the Russian physiologist Pavlov and his co-workers, that digestive secretion begins before food reaches the stomach and that a conditioned reflex starts digestive secretion when food is seen and smelled. It had been established with certainty that this reflex is produced *via* the vagus nerves. This had been demonstrated by Pavlov as early as 1889, in experiments with sham feeding in dogs with an oesophageal fistula. In addition it was known that, as soon as the food bolus passes into the stomach, digestion proper begins when gastric juice is secreted in response to physical and chemical stimuli chiefly arising from the pyloric antrum. It had been demonstrated that secretion of gastric juice is caused by a hormone which

was given the name of gastric secretin. At that time it was already clear that the pyloric part of the stomach exerts a regulating influence on secretion of gastric juice, that this secretion is inhibited when the acidity in the pyloric part increases, and promoted when the gastric contents become alkaline. Since it was also clear that the presence of a duodenal ulcer was associated with an increased secretion of gastric acid, resection of the pyloric antrum was resorted to with the exclusive object of reducing the acidity of the gastric juice. It was soon found advisable to resect at least two-thirds of the distal part of the stomach if sufficient reduction of gastric acidity was to be achieved. This, then, was the basis of resection therapy for peptic ulcer. And new therapies introduced later continued to aim at reduction of gastric acidity. In gastric resection, the ulcer too was removed if possible, although this was not considered a necessity. Relapse of ulceration was relatively uncommon, and this therapy was for a long time considered the treatment of choice of ulcers of the stomach and duodenum.

The operation can be performed in two ways. The gastrectomy that Billroth performed in 1881 involved a resection followed by a gastro-duodenostomy; this procedure is known as the Billroth I gastrectomy. In 1885, Billroth found it necessary to introduce a modification. Because a gastro-duodenostomy was not feasible, he closed the stomach and established a gastro-jejunostomy of the antecolic type. This procedure, with gastro-jejunostomy, became known as the Billroth II gastrectomy. Numerous modifications of this procedure are still in use. Today we preferably establish a retrocolic termino-lateral gastro-jejunostomy with a short loop. The discussion between the advocates of the first and those of the second Billroth procedure is not yet closed. The great advocates of the Billroth I were Schoemaker in The Hague and Von Haberer in Cologne. But gradually it was found that the first Billroth procedure had a much larger percentage of relapses in duodenal ulcer than the second. Perhaps we must ascribe this to the fact that, in cases of duodenal ulcer, the duodenal mucosa is diseased and therefore less suitable for renewed contact with gastric juice. The results in gastric ulcer are much more satisfactory, and this explains why many surgeons in the Netherlands today confine the first Billroth procedure to gastric ulcers, and use the second Billroth exclusively in the treatment of duodenal ulcers. Especially the second Billroth procedure has been carried out in numerous modifications, and for a long time the Polya modification was employed extensively; in this procedure, the entire transverse section of the stomach was anastomosed end-to-side with a loop of jejunum.

Thus, while resection therapy in general gave very good results and had relatively few relapses, it nevertheless had serious disadvantages. Foremost among these was the so-called dumping syndrome, which greatly inconvenienced the patient in a varying number of surgical cases. A detailed discussion of this syndrome would require too much time, but I must point out that symptoms of this type are seen not only after a gastrectomy but also following gastro-enterostomy, pyloroplasty and vagotomy (although their incidence in these cases is less high). It seems probable that the size of the anastomosis between stomach and jejunum influences the development of this syndrome. Serious forms of this syndrome are less frequently seen after establishment of a smaller anastomosis, which still ensures some reservoir function of the stomach.

It need not be stressed that a partial gastrectomy which removes two-thirds to three-quarters of the stomach, entails a serious mutilation. This mutilation is the more serious because we know that it is inflicted in order to control a symptom of a disease. After all, the ulcer is not the primary seat of the disease; this primary seat must be sought elsewhere, the ulcer being merely a consequence of the secretion of large amounts of gastric juice with highly peptic properties. Essentially, therefore, gastrectomy for peptic ulcer is a very questionable operation. It gives rise to severe changes in the physiology of digestion; fat absorption is disturbed to varying degrees, and other changes in the pattern of digestion also occur. It is consequently not surprising that many investigators have sought ways to avoid this mutilation. Even Billroth himself seems to have resorted to segmental gastric resection in some cases. Von Mikulicz described the technique of this procedure in 1889. In subsequent years, it was Wangensteen and Werner in particular who continued to accept this operation. But it was never generally accepted because the risk of persistence of the ulcer is very high (not surprising, since the physiology of gastric secretion remains uninfluenced).

Since it was difficult to excise the ulcer in some cases, operations were accepted which leave the ulcer in situ. In the case of duodenal ulcer this led to exclusion of the pyloric antrum—an operation first performed by Doyen in 1893, and recommended by Von Eiselsberg in 1895. It was soon found, however, that when part of the antral mucosa was left in situ, the gastric phase of secretion was enhanced because the alkaline duodenal juice touched upon the antral mucosa, and the fundus region was stimulated to pronounced secretion via hormonal mechanisms. Relapses of ulceration were seen in nearly 100 per cent of cases, and the operation is consequently no longer performed today.

Not surprisingly, every surgeon active in the field of gastric surgery accepted gastrectomy for ulcer only with reluctance, although the results obtained were certainly satisfactory. The quest for less mutilating procedures continued, and an improved understanding of the physiology of digestion caused Dragstedt to re-emphasize the value of vagotomy, previously already carried out by Exner in 1911, Bircher in 1920 and Laterjet in 1922. Lester Dragstedt was originally a physiologist and pupil of the Utrecht professor Zwaardemaker. Physiological thinking continued to determine his actions throughout his career as a surgeon.

It may be useful at this point to present a very brief review of our present knowledge of the physiology of gastric digestion. Chiefly as a result of the work done in the experimental laboratories of surgical departments, a great many facts have been collected which have enriched our understanding, Meanwhile, many questions have remained unanswered.

The mucosa of the stomach is lined by a layer of high cylindrical cells which secrete mucin; the internal surface of the stomach is completely covered by a layer of mucus. The gastric mucosa begins abruptly at the junction of oesophagus and cardia and at the level of the pylorus changes to intestinal mucosa. The internal surface of the stomach has numerous depressions—the foveolae—which greatly enlarge the actual surface area of the mucosa. There are three types of gastric glands, namely: cardiac glands, chief glands and pyloric glands. These glands contain at least five types of cells. The zone of cardiac glands is narrow and of variable size; it contains mucin-secreting cells. The chief glands (also known as

proper gastric glands, fundic glands or principal glands) are localized from the area of cardiac glands to that of pyloric glands. They contain cells of all types except the type of the pyloric glands. The chief glands secrete hydrochloric acid—a unique biological process about which we know very little. In the same glands, the principal enzyme—pepsin—is produced in the zymogenic cells. The function of pepsin is to hydrolyse proteins to proteases and peptones preparatory to complete digestion in the small intestine. The degree of peptic hydrolysis varies with the pH in the bolus, and attains a maximum at pH 1.8. Other proteolytic enzymes have been identified in gastric juice, but little is known about them.

The principal cell type found in the pyloric glands secrete mucin and the hormone called gastrin. The hypothesis of hormonal control of gastric juice secretion was originally advanced by Edkins (1906). He called the hormone gastrin. Today we know that gastrin can be secreted as a result of local stimuli and stimulation of the vagus nerve.

Local stimuli governing the production and release of gastrin act by two mechanisms :

(a) mechanical distention, and
(b) chemical stimulation.

Dragstedt and co-workers demonstrated the importance of local stimuli in the release of gastrin in normal daily acid secretion. After resection of the antrum, they observed a decrease in the 24-hour secretion of acid by 65-95 per cent. Mechanical stimulation of the gastric mechanism is effected by the food bolus as it passes the area of pyloric glands. Chemical stimulation is provided by proteins and their degradation products. It is probable that all food products activate this mechanism in one way or another.

No other topic in the physiology of gastric secretion is surrounded by more controversies than the process of the vagal release of gastrin. Surveying the various data, we may accept the following tentative conclusion.

During the cephalic phase of acid secretion, gastrin is released in response to direct stimulation of the antrum by the vagus nerve. However, in the absence of the antrum, vagus stimulation is likewise followed by acid secretion—apparently as a result of direct action of the vagus stimuli on the chief cells. While the exact mechanism of vagal release of gastrin is still obscure, it seems probable that the vagus nerve possesses fibres extending to the antral mucosa either directly or via the submucosal plexus; when stimulated, these fibres probably cause the release of gastrin. In any case we can accept as certain that gastrin release from stimulation of the vagus nerve must be completely separated from gastrin release caused by local antral stimuli.

It is of importance in this context to recall that, in 1959, DeVito demonstrated that complete antral mucosal denervation reduces the 24-hour secretion of gastric acid by 20-80 per cent. Since neither antral mucosal denervation nor extrinsic antral vagal denervation alters the release of gastrin caused by mechanical and chemical stimuli, the conclusion seems justifiable that the percentage of total daily gastric acid secretion based on vagally induced gastrin release is considerably larger than has been assumed. However this may be, both gastrin release due to vagal stimuli and that caused by local antral stimuli are influenced by the same antral acid-inhibiting mechanism. The only positively known inhibitory influence

on the mechanism of gastrin release is the pH of the antral mucosa. An acid pH prevents gastrin release in response to all types of stimuli. It is doubtful whether, in addition, an antral inhibitory hormone is secreted.

The period and phases of gastric secretion can be summarized as follows:

Period I: Interdigestive secretion
Period II: Digestive secretion
1. Cephalic phase
A. Direct vagal phase
B. Vagal-antral phase
2. Gastric phase
A. Local antral phase
(a) chemical stimuli (b) mechanical stimuli
3. Intestinal phase

In normal individuals, the interdigestive secretion is virtually zero. All investigators agree that in patients with duodenal ulcer this interdigestive secretion is increased—even in patients with a healed asymptomatic ulcer. Unlike these individuals, patients with gastric ulcer and gastric carcinoma produce a normal amount, or less.

Digestive secretion begins with the cephalic phase. Seeing, smelling and tasting food produce conditioned reflexes which, via the vagus nerve, cause secretion of gastric acid within five minutes, partly by gastrin release and partly by direct stimulation of the chief cells.

The gastric phase is introduced by the hormone gastrin, as a result of chemical and physical stimulation caused by the food bolus. As early an investigator as Pavlov demonstrated that all sorts of food introduced directly into the small intestine, induce secretion of gastric acid. It was also demonstrated that this intestinal phase of gastric digestion is subject to hormonal control. We know very little about the intestinal phase of gastric digestion in man; while it is believed to exist, it is considered to be of little importance. We cannot dwell on the endocrine glands; suffice it to mention that products from the pancreas, parathyroid and thyroid glands, and gonadal hormones, are known to exert an influence.

The peptic qualities of normal gastric juice are so pronounced that the normal gastric wall would be affected were it not for the fact that the organism has certain means of defence. It is generally agreed that the mucus which covers the entire gastric wall, and which is secreted in large amounts upon all forms of stimulation, lends some protection to the gastric wall—together with superficial gastric mucosal cells which show a particularly swift reaction to lesions or degeneration. But the best protection from an individual's gastric juice is afforded by the food he ingests, which binds the acid.

The secretory pattern in gastric ulcer differs widely from that in duodenal ulcer. In the former case, there is no abnormal interdigestive secretion, and often only inconsiderable activity during digestion. Although the digestive activity of gastric juice must be a common causative factor in both gastric and duodenal ulcers, we must assume that in the case of gastric ulcer there are additional factors which reduce the resistance of the gastric mucosa. As regards the treatment of gastric ulcers, there is hardly any doubt that gastrectomy with excision of the ulcer is the therapy of choice, though there are others who recommend vagotomy with pyloroplasty.

The situation concerning duodenal ulcers is quite different. Nearly always, there is greatly increased secretion, particularly during the interdigestive period (that is: at night, on an empty stomach). This hypersecretion is obviously induced by the vagus nerve, and vagotomy of the trunk is therefore bound to cause a favourable change in the pattern of secretion. It eliminates the dangerous "fasting" secretion, during which the gastric juice is not bound by food.

Vagotomy for the treatment of duodenal ulcer was advocated with conviction by Dragstedt, and was accepted on a large scale in Anglo-American countries. And indeed this operation—which Dragstedt described as a physiological operation—seemed to influence only the pathological physiology of the stomach. Soon, however, it became apparent that the procedure also affected gastric motility, and that additional gastric drainage operations were therefore required. The gastro-jejunostomy which Dragstedt advocated in this context, has a number of disadvantages. It stimulates the gastric mechanism; pyloroplasty as later recommended by Weinberg ensured more adequate drainage of the atonic stomach and was therefore more readily accepted.

In spite of its affect on motility, vagotomy continued to be attractive because it did not entail mutilation of an important organ in order to control a symptom of disease. The stomach as a reservoir was spared. Especially in Anglo-American countries, vagotomy is being increasingly used in the treatment of duodenal ulcer. In other countries, however, it has hardly been accepted, if at all, and numerous surgeons all over the world have remained advocates of gastrectomy for duodenal ulcer. It would therefore seem useful to present a critical review of the principal procedures in current use.

Many investigators, particularly in the U.S.A., maintain that a gastrectomy (I mean a Billroth II procedure) performed for duodenal ulcer, should be a resection which removes some 75 per cent of the stomach. Only in this way, it is contended, can one reasonably hope to prevent development of a stomal ulcer. On the other hand, we know that more than 25 per cent of the surface area of the stomach must be left intact if digestion is not to be seriously disturbed. This means that one must sail between Scylla and Charybdis, and that the resection must be very mutilating. Even then, the rate of recurrence is believed to be about 3 per cent. I cannot agree with this point of view. In my department no more than two-thirds of the stomach is resected, and recurrent ulceration is nevertheless relatively seldom. A few years ago I studied a continuous series of 600 non-emergency cases in which I found three recurrences, that is 0.5 per cent. However, the operation is performed in our department only for strictly defined indications and not until medical therapy has been given ample opportunity to heal the ulcer. In no case do we operate on ulcers in the active stage. It is my conviction that a technically faultless gastrectomy, performed on a correctly determined indication, gives the patient an excellent chance of a further life without ulcer and with no or hardly any disturbance of digestion. If in addition one ensures that the stoma between gastric stump and jejunum is narrow, the cases developing a dumping syndrome are few, and serious forms of the syndrome do not occur.

Perhaps dumping symptoms have been reduced in general by vagotomy with drainage procedure, but certainly these symptoms have not disappeared. While it must be admitted that the mortality of this procedure is lower than that of

gastrectomy, it has hardly reduced the percentage of recurrent and stomal ulcers. There has been a tendency to ascribe this to incomplete vagotomy (Burge and Pick indicate a percentage between 3 and 30). It is hoped that Burge's method of electrical verification of the completeness of vagotomy will improve the results. Nevertheless, some fervent advocates of vagotomy, such as Harkins, have found it necessary to combine the so-called physiological operation of vagotomy with an antrum resection. This combination of vagotomy with antrum resection seems to have virtually solved the problem of stomal ulcers. Instead of 75 per cent, only some 50 per cent of the stomach need be resected so that a large reservoir remains intact. Meanwhile we do not know to which extent dumping symptoms occur, nor whether and to which extent digestion is disturbed.

Let me for a moment consider the question of the extent to which vagotomy can be regarded as a physiological operation. Dragstedt emphatically maintains that vagotomy is a physiological procedure, and admittedly the operation does not mutilate anatomical relationships. The stomach remains intact, the pathological digestion is corrected and appears to be the only factor influenced because the excessive vagus-induced secretion of gastric juice is arrested. If this were all, one could describe the effect as a restoration of physiology. Now I wish to ignore the influence of vagotomy on the functions of pancreas and liver. We know that the secretion of these glands is largely subject to humoral control, but we know nothing about the long- term effects of vagotomy on these glandular functions.

The effect of vagotomy on gastro-intestinal motility is more important. We know that severance of the vagus nerves leads to atonia of the stomach. Besides and in addition, however, vagotomy affects the motility of the intestine. The consequences are unpredictable: they may be absent, they may be mild, they may be serious. However this may be, severe diarrhoea with paroxysms has been observed in at least 3 per cent of postvagotomy patients, and less severe symptoms of this type occur in a much larger percentage of cases. In a series of about 80 cases in which I myself used this procedure, there were a number of postvagotomy patients who, although the ulcer symptoms had disappeared, were worse off than before, as a result of diarrhoea and abdominal symptoms.

It is remarkable to note that the advocates of vagotomy would seem to wish to keep their eyes closed to this therapeutic complication, which in my opinion is a very serious one. Only a few, such as Burge, frankly point out these serious and unpredictable ill-effects of vagotomy. I am personally of the opinion that a frequency of 3 per cent of symptoms of such severity is too high a price to pay for the benefit of seeing fewer dumping symptoms than after gastrectomy; the more so because marginal ulcers as complication following vagotomy are only slightly less frequent than after gastrectomy.

Thus we can again raise the question whether vagotomy may be considered a physiological operation; I must answer this question firmly in the negative. Vagotomy is as unphysiological an operation as gastrectomy. While the latter procedure mutilates the anatomy of the stomach so as to influence the pathophysiology, vagotomy mutilates the physiology of the stomach and intestine. It is therefore understandable that some investigators such as Burge seek to perform selective vagotomy, severing only such fibres of the vagus nerve as govern the stomach. The history of these selective operations is still very short, and the results

are in part still unsurveyable and in part contradictory.

In October 1965 I attended the congress of the American College of Surgeons in Atlantic City. During a panel discussion I found that the vast majority of the younger surgeons present were advocates of vagotomy, while the older surgeons—such as Ochsner and myself—continued to regard gastrectomy as the treatment of choice in duodenal ulcer. Future findings will show who is right; even so, "vagotomists" are meanwhile well advised not to regard their procedure as physiological, as I frankly told Dragstedt during the discussion on that occasion. In Great Britain, too, vagotomy seems to be becoming popular among the younger generation of surgeons. In continental countries this trend is not seen. In the Netherlands, there is only a single large surgical department of a municipal hospital where the operation has been regularly performed for the past few years. According to information which I received from Professor Linder, the procedure has not been accepted in Germany; in France, the department of Professor Weisz in Strasbourg is virtually the only department where vagotomy plus drainage procedures is being performed as standard operation.

Undeniably, the question of the treatment of choice in duodenal ulcer still lacks a definitive answer. We are in a field of many controversies. To me, personally, the fact that a surgeon such as Harkins has come to combine vagotomy with antrum resection is very meaningful.

Finally, let me be allowed to say a few words about recurrent ulcers. On 20th October 1960 it was my privilege to deliver a Moynihan Lecture before the Royal College of Surgeons of England, in which I discussed 200 consecutive cases of recurrent ulcer treated in my department. The series included three patients who had been previously treated in my department. My chief interest lay in the cases of recurrence after a Billroth II procedure. There were one hundred such cases. Analysis showed that nearly all these instances entailed faulty technique or imperfect determination of indications. The majority were technical errors, in that a pyloric antrum had been left in situ or a much too small resection performed. One might expect that a correction of the technical faults and resection of the recurrent ulcer would be sufficient to prevent further relapses. Not true! In no fewer than 15 per cent of cases, a subsequent marginal ulcer developed in a manner as unexpected as it was incomprehensible. This experience throws a harsh light indeed upon the inadequacy of our current knowledge of the patho-physiology of the secretion of gastric juice. Since in all these cases we found ourselves "backed up against the wall", if you'll permit me to use the metaphor, we resorted to a vagotomy in treating the second recurrence. Experience has further taught us that a recurrence can be prevented only by adding a vagotomy to the corrective operation. It is a remarkable fact that no, or hardly any, diarrhoea has been observed in these cases.

Ladies, and gentlemen: I have taken much of your time in this attempt to confront you with the many unsolved problems which we must face in the surgical treatment of gastric and duodenal ulcers. Much (too much) is still obscure in the normal and pathological physiology of gastric digestion, and consequently the surgical treatment of these ulcers as yet lacks a firm foundation. It is up to younger generations of surgeons to attempt to clarify these problems, and to devise a therapy which is truly physiological.

A FOLLOW-UP OF A CASE OF DOCTOR HARVEY'S

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HONEUR SANS REPOSE

WILLIAM HARVEY recorded in De Generatione Animalium his famous observation of a young man whose chest wall was deficient, so that his heart could almost directly be palpated. Much information about the young man's life and death is available in the work of William Montgomery who was his cousin and brother-in-law. This note is to provide a collation of the two accounts, that of Hill (1869) being available to few.

Even Harveian orators have not known that the young man was Hugh Montgomery, of Newtownards, in the County of Down. He was the older son of Hugh, the second viscount Montgomery of the Great Ardes. His mother was Jean Alexander, daughter of Sir William Alexander, Secretary for Scotland. His grandfather, the first lord, originally sixth laird of Braidstane in the county of Ayr, was the celebrated planter of the Scottish colony in North Down in the neighbourhood of Newtownards, Donaghadee and Comber. This youth, says Harvey, "when a child had a severe fall attended with fracture of the ribs of the left side. The consequence of this was a suppurating abscess, which went on discharging abundantly for a long time, from an immense gap in his side". This Harvey learned "from himself and other credible persons who were witnesses". One of these credible persons may have been Doctor Patrick Maxwell, for it was he, so William Montgomery tells us, "who had made the orifice in his side when a boy at school and prescribed the lotion for it". This Doctor Patrick Maxwell had long lived in North Down, presumably at Newtownards, as "pensionary phisician" to the first and second lords. In 1641–42, William Montgomery tells us, Maxwell was attending King Charles I as physician. About the end of 1641 young Hugh, then eighteen, who had been travelling in Europe, was called home because of the outbreak in October of the great Irish War. It is safe to assume that he would not pass through England without seeing the doctor who had attended him so long. William Montgomery says Maxwell "was glad to meet with Mr. Montgomery, of the Ardes, his quondam patient (as it is lately said), now in good plight of strength and health". It seems likely that Maxwell told the King of Hugh's remarkable disorder. Harvey says that the King, having learned of the circumstances as "something miraculous", sent Harvey himself "to wait on the young man and ascertain the true state of the case".

"And what did I find? A young man, well grown, of good complexion, and apparently possessed of an excellent constitution, so that I thought the whole story must be a fable. Having saluted him according to custom, however, and informed him of the King's express desire that I should wait upon him, he immediately showed me everything, and laid open his left side for my inspection, by removing a plate which he wore there by way of defence against accidental blows and other external injuries. I found a large open space in the chest, into which I could readily introduce three of my fingers and my thumb; which done, I straightway perceived a certain protuberant fleshy part, affected with an alternating extrusive

and intrusive movement; this part I touched gently. Amazed with the novelty of such a state, I examined everything again and again, and when I had satisfied myself, I saw that it was a case of old and extensive ulcer, beyond the reach of art, but brought by a miracle to a kind of cure, the interior being invested with a membrane, and the edges protected with a tough skin. But the fleshy part (which I at first sight took for a mass of granulations, and others had always regarded as a portion of the lung), from its pulsating motions and the rhythm they observed with the pulse—when the fingers of one of my hands were applied to it, those of the other to the artery at the wrist—as well as from their discordance with the respiratory movements, I saw was no portion of the lung that I was handling, but the apex of the heart! covered over with a layer of fungous flesh by way of external defence, as commonly happens in old foul ulcers. The servant of this young man was in the habit daily of cleansing the cavity from its accumulated sordes by means of injections of tepid water; after which the plate was applied, and, with this in its place, the young man felt adequate to any exercise or expedition, and, in short, he led a pleasant life in perfect safety. Instead of a verbal answer, therefore, I carried the young man himself to the King, that his majesty might with his own eyes behold this wonderful case: that, in a man alive and well, he might, without detriment to the individual, observe the movement of the heart, and, with his proper hand even touch the ventricles as they contracted. And his most excellent majesty, as well as myself, acknowledged that the heart was without the sense of touch; for the youth never knew when we touched his heart, except by the sight or the sensation he had through the external integument. We also particularly observed the movements of the heart, viz.: that in the diastole it was retracted and withdrawn; whilst in the systole it emerged and protruded; and the systole of the heart took place at the moment the diastole or pulse in the wrist was perceived; to conclude the heart struck the walls of the chest, and became prominent at the time it bounded upwards and underwent contraction on itself."

William Montgomery says the King "had the curiosity to look at the palpitation of his heart which was plainly discernable at the incision which was made in his side; 'Sir,' said the King, 'I wish I could perceive the thoughts of some of my nobilities hearts as I have seen your heart,' to which Mr. Montgomery readily replied, 'I assure your majesty, before God here present and this company, it shall never entertain any thought against your concerns, but be always full of dutiful affection and steadfast resolution to serve your majesty.' He stayed a few days at court, and the King had him in particular favour and here (I believe) was laid that unshaken foundation of loyalty whereon all his succeeding actions were built".

Hugh hastened home to county Down. The strength of the closely settled Scottish colony in North Down made it one of the few semi-tranquil places in Ireland for the next ten years. The Irish army never penetrated there. But the boy was rarely to know tranquillity again. The second lord, who had raised a regiment of a thousand foot and five troops of horse, died in 1642, so that at nineteen Hugh became the third viscount and head of his people. Thereafter he was busy for eight years in the arms and counsels of the North, until in 1650 he perforce surrendered to the English parliament; Cromwell's victories having put an end to

the long and confused war. In June, 1646, he had commanded the horse at the second battle of Benburb, where a combined Scottish and English army was heavily defeated by Owen Roe O'Neill. Montgomery "warmly charging" and "coldly seconded" was captured, and imprisoned for some twenty months in the castle of Cloughoughter in a lake in county Cavan. "It impaired his health tho' he wanted not wholesome vivers". In February, 1648, he was released by being exchanged for the Earl of Westmeath and passing through the Anglo-Scottish lines reached Carrickfergus. In 1649 the Royalists and Presbyterians of the North combined against the English Commonwealth, and Montgomery received the King's commission to be general of all the forces in Ulster. He was energetic and had considerable success but, though he seized Carrickfergus and Coleraine, he failed, as might have been expected, to take Derry. His troops and people were not so royalist as himself. The great Owen Roe was formidable in south-west Ulster. Coote held Derry for the English Commonwealth. All came to an end with Cromwell's short sharp campaign.

The Commonwealth exiled Montgomery to Holland where he spent some lonely years. In 1651 he visited Leyden, seeing "an Atomy chamber". In Hunsterdyke he saw "a copper pan and a brass one in which a countess of Holland's birth were baptised, the males and the females separately, but at one time; the infants (in all) were three hundred and sixty-five". Perhaps the poor countess was delivered of a hydatidiform mole. In 1652 or 1653 the Commonwealth allowed him to return to Ireland. He was no more than thirty. He was joyfully united with his family and although he suffered inconvenience and indignity at the hands of the Commonwealth government in Dublin, and even was imprisoned in Kilkenny for a short time, he was no longer in serious political trouble. However, for the most part he had to remain in Dublin. His fortune was seriously diminished, and his debts increased. His health was not so good. He grew corpulent. When Henry Cromwell became ruler of Ireland in 1657, Montgomery's relations with the government became comparatively easy. It was at this time, when he came to Dublin to salute Henry Cromwell, that he "was taken with sickness, which did cast him into a deep palsy that seized all one side of him; and being lodged next house to Doctor Ffennell after many weeks his lordship recovered", though "melancholy".

The restoration of the King in 1660 improved Montgomery's fortunes. He was made a commissioner for putting into execution the King's declaration for the settlement of Ireland. He was made earl (Mount-Alexander) and a privy councillor. He became Master of the Ordinance and Military Stores in Ireland, and seemed to be in a fair way to improve his fortune and to pay his debts. He "lived in grandeur" in Dublin, "highly esteemed and respected by all, and for his ripe judgement appearing when he spoke in the House of Lords or at the Council Board". Yet his health was worse. "His lordship had fallen into a discentery, which lay sore upon him, changing its complexion twice or thrice. It was very dangerous, his body having grown unwieldy and bulksome; but by God's blessing (on Doctor Fennell's endeavours) he recovered and was but weakly well mended, for that flux had brought him low too suddenly, by evacuating a great abundance of humours and fatt by which he was become formerly uneasy to himself."

He had not long to live. Blood's plot took him to County Down, where he was able to do much for the government in settling the unrest, and for his people in

keeping them out of trouble. There "his drowsy distemper grew fast upon him, that in a fortnight he was much indisposed to write." At this time he got little business done, because of "the daily increase of his distemper which was plethorick; his liver was large and strong, and sent more blood to the heart than it could vent fast enough (for his heart was wissened and shrivelled to less than it should be (occasioned by defect of the plurae) to preserve which from corruption the lotion aforesaid was used every morning and at bedtime, by injection at the said orifice with seringe); and this surcharge of blood upon the heart caused the swimming and obfuscation in his brain (which in itself had no fault the abundance thereof) and made him drowsy every third and fourth hour. The first remedy was to let his veins often breath out part of that superfluous mass of rarified blood ; but Primrose, the Belfast apothecary (who practised physic) understood not the matter, and was timorous to tamper in that case. Wherefore his lordship hastened back to Dublin and (by the way) died in his bed at Dromore, the fifteenth night of September, 1663 : The next morning Dr. Gray (who had been sent for) averred that if his lordship had often been bled in several veins, and his blood sweetened and thickened, it had not gushed out (as it did divers times) at his nose, nor so oppressed his brain making it giddy and his eyes to be bemisted. This Dr. disembowelled him and embalmed him". He had died at forty years of age.

William Montgomery says he was "among the properest of middle-sized men, well shaped, of a rudy sanguine complexion ; his hair had been reddish and curled . . . his eye grey and quick, and his countenance smiling and complacent, his arms and thighs sinewy and brawny". He was a kind good person, "the most regarded Scottish man in Ireland".

It is difficult to know if the original disease really was traumatic. If it was, it seems there was no open injury. Perhaps a haematoma became infected. If the history of the fall is only a rationalisation, the abscess may have been due to tuberculous or staphylococcal osteitis of a rib. It may more likely in that case have been staphylococcal. The transient hemiparesis may have been embolic, but the atria can hardly have been fibrillating when Harvey examined him, for he would surely have recorded irregularity of the ventricles. The only congestive failure was terminal. We know of no record of Dr. Gray's observations. His disembowelling gave him the opportunity to examine the heart, and perhaps William Montgomery's note that the heart was "wissened and shrivelled" was information obtained from Dr. Gray. It is hard to understand, unless there was a constrictive pericardial thickening. Perhaps we may hazard a diagnosis of staphylococcal osteitis of rib, with pericarditis, bone necrosis, surgical drainage and chronic open pericarditis, eventually constrictive.

Montgomery's social and political situation is interesting. His race was Scottish, his domicile was Irish. He "had no hatred or love solely for country sake; English, Scotts and Irish were welcome to him, yet he liked and esteemed the English most (both his Ladys being such)". In religion he was inclined to his mother's Presbyterianism in early life. In later life he adhered to the Episcopal Church. In early life he belonged to the patriarchal farming community in North Down. In later life he joined the governing circle in the field and in the capital. William Montgomery is probably right when he says that the early attachment to the King ruled his life-course. If so it was disastrous for Montgomery and his family. It

separated him in religion and politics from his tenants and dependents in North Down, and ensured him the enmity of the English Commonwealth. The estate could not but be maladministered during the troubled time. His debts grew, and after the Restoration the patronage of the King and his Irish government were a poor substitute for the revenues of the colony his grandfather had founded. By the end of the century most of the Montgomery estate was in other hands. It has to be remembered, however, that Montgomery's royalist sympathies were rooted in more than his kindly reception by King Charles I when he was eighteen. It was the King's Scottish father, James VI of Scotland and I of England, who had granted the territory in North Down to the first lord, and it was only because James VI of Scotland had become James I of England that it was possible for the Scots easily and legally to settle in Ireland. There until the union of the crowns they would have been the subjects of a foreign and sometimes hostile king. The Montgomery kinship's fortunes had grown with the fortunes of the Stuarts, and derived from their favour, and it is not to be supposed that a man like Montgomery would desert them when they were in trouble.

References and Notes

Harvey, William. De Generatione Animalium, translated by Robert Willis, and printed in The Works of William Harvey. The Sydenham Society, 1847.

The Montgomery Manuscripts, compiled by William Montgomery of Rosemount, and edited by George Hill. Belfast. 1869. Archer and Sons.

Every Ulsterman must feel respectful gratitude to William Montgomery for his remarkable history of his time. The Manuscripts are a sufficient biography of the man and the scholar. Hill's edition is very important.

For a biographical note on George Hill, see McCleery, J., The Nonsubscribing Presbyterian, March, 1961, Number 652.

In some contemporary accounts of the Irish War of 1641 the third viscount is referred to as the Lord of Ardes.

Hill notes that the third viscount Montgomery was married firstly to Mary, daughter of Charles, second viscount Moore of Drogheda, and secondly to Catherine Jones, daughter of Arthur Jones, second viscount Ranelagh.

In the last sentence of Harvey's account the word "diastole" is used twice. On the first occasion it plainly means ventricular dilatation with relaxation. On the second occasion Harvey speaks of the "diastole in the wrist", and we may suppose he means the dilatation of the radial artery with ventricular systole. In the Hague edition of 1680 the passage reads . . . "fierique in corde systolen, quo tempore diastole in carpo percipiebatur". The words "or pulse" are a gloss, presumably of the translator's.

Harvey says that Hugh Montgomery "came to London". William Montgomery says that Hugh "kissed King Charles his hand at Oxford". The Irish war broke out on the 23rd of October, 1641, and then Hugh was called home from Europe. The King left London on the 10th of January, 1642, not to return until the time of his trial and execution in 1649 (though for some time he was not far away at Windsor). He did not settle in Oxford till the 29th of October, 1642, after Edgehill. However, William Montgomery says "Mr. Montgomery came home before Ao 1642 (as I think)". So London may be right. We have no precise date of Hugh's visit to the King.

TEN YEARS OF GENERAL PRACTICE MIDWIFERY 1954/1963

By NOEL SANDERSON, M.B.
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MY partner and I conduct a mixed rural and urban general practice in a market town in Northern Ireland with a population of about five thousand. There are somewhat over three thousand on our National Service list, most of them living within six miles of the surgery premises, and providing a yearly average of eighty maternity cases. There is a consultant obstetric unit in our local general hospital, and a fully equipped maternity unit in the cottage hospital.

During the ten years 1954 to 1963, there were 813 maternity cases, 110 of which were delivered at home, 379 in the cottage hospital, and 324 in the general hospital. There were 41 miscarriages included in the general hospital figure, and as 400 women accounted for the 813 cases, a pregnancy occurred in each family about every five years, the first child being born when the mother was 27 years old on the average. If infertility is a problem here, it has not often been brought to our notice.

It has been our custom for many years not to conduct a first confinement at home, and if the primipara does not want to have her baby in hospital she is asked to engage some other doctor. This attitude has now become acceptable to the patients, our reasons being that in the past we had to deal with difficult forceps extractions and manual removals of placentae in wholly unsuitable houses. The grand multipara also, is encouraged to have her delivery in hospital, as the intermediate children, especially the second, are least likely to be complicated.

Three primigravidae were delivered in the cottage hospital for every two delivered in the general hospital, an equal number of multiparae were delivered in each and several primigravidae were confined elsewhere. Although domiciliary nursing services were greatly improved during this decade, the number of home confinements fell from 25 per cent to 3 per cent per annum.

As midwifery is such a very personal service, it may be an advantage to have known the patient, her family circle, religion and background, and attendances for ante-natal examinations were as a general rule satisfactory. No special or separate ante-natal sessions were conducted, inquiry having shown that there was no particular demand for them. Unfortunately, in this area it is necessary to send three samples of blood for grouping and Rh factor, haemoglobin estimation and Wasserman reaction, each packaged and accompanied by a form to three separate laboratories. This should be avoidable; it is irritating, time consuming and inefficient.

In the last few years every patient has been given iron and folic acid in the third trimester and I feel sure that the introduction of folic acid has been a great advance, for the patient coming to term with a satisfactory haemoglobin level makes a favourable difference to the confinement and the resulting improved outlook of the patient is important. The administration of folic acid, which is not expensive, is said to reduce the incidence of ante partum and post partum haemorrhage, and the experience in this practice supports that view.

Classification of Maternity Cases

As there are two case records of each patient there were some 1,500 to consider, and after classification they were described as primiparae, intermediates and grand multiparae. This last category implies that the patient has had five or more viable pregnancies, and a primipara is one who is pregnant but has not had a baby. The others, making up the largest group were called intermediates.

The 813 cases were composed of 200 primiparae, 500 intermediates and 113 grand multiparae. Excluding miscarriages the figures were 187, 480, and 105 respectively.

Ante-Natal Care and Special Vigilance

The amount of ante-natal care given was noteworthy. All cases have vigilance, but a large number required what I call "special vigilance".

This means that they required an increased number of ante-natal examinations by reason of poor previous obstetric or medical history, toxaemia, ante partum haemorrhage, multiple pregnancy, negative Rhesus factor, unstable lie, anaemia, heart disease and so on. Special vigilance was kept on thirty-five per cent of primiparae, thirty per cent of intermediates and thirty per cent of grand multiparae; in all, amounting to 236 cases out of 772, which was 31 per cent overall. Less than half the midwifery cases were completely normal, for no account was taken of those with varicose veins, urinary infections, vomiting and other minor but potentially serious conditions which often did require considerable vigilance.

Miscarriages

The miscarriage rate was 5 per cent, that is, 41 cases out of 813. Miscarriages as a rule were seen only at the time of the event, and all were admitted to hospital for curettage. Thirteen primiparae (6.5%), 20 intermediates (4%), and 8 grand multiparae (7%) miscarried. It has not been common for these patients to have repeated miscarriages, but probably many cases have been unrecorded.

Pre-Eclamptic Toxaemia

A considerable number, 133 (18%) required special vigilance on account of pre-eclamptic toxaemia. Of these, 47 (33%) were primiparae, 74 (55%) were intermediates and 17 (12%) were grand multiparae, but in the groups, 25 per cent of primiparae and 16 per cent of all multiparae were toxic. For our purposes, the toxaemic state was characterised by a blood pressure rising, with or without albuminuria and/or oedema. Not everyone is agreed on the precise criteria. A blood pressure of 130/90 to 140/90 was taken to be indicative of toxaemia, and to avoid complication there has been no division into pre-eclamptic toxaemia, essential hypertension and chronic nephritis, all being called toxaemia, for in this small number the difference would not be significant. Of the total number of cases delivered 18 per cent had toxaemia to some extent, and of those who required special vigilance 47 (40%) of the primiparae, 74 (40%) of the intermediates, and 17 (30%) of the grand multiparae required their special vigilance because of toxaemia. About one out of every six expectant mothers (138 out of 772) had some degree of toxaemia, and one out of every three (138 out of 360) requiring special vigilance had toxaemia. The figures show, that in our surgeries we see twice as many cases of toxaemia among those who are not primiparae, having dealt with 585 multiparae and 187 primiparae.

Recurrence of toxaemia in pregnancies subsequent to the first was found in 34 per cent. Two patients did not have toxaemia in their first pregnancy, but had in their second and subsequent pregnancies, and two grand multiparae developed toxaemia for the first time, as far as could be ascertained, in tenth and twelfth pregnancies respectively.

Treatment was conservative and if necessary artificial rupture of the membranes was performed, diuretics being very seldom used for they can be dangerous as they may mask a persistent underlying morbid condition of the placenta in particular. Also diuretics can cause an electrolyte imbalance if pushed too far; induction should have been performed before that point was reached otherwise the foetus may die as a result of unsuspected placental deficiency.

Ante Partum Haemorrhage

There were 26 cases (3%) of ante-partum haemorrhage and they were all admitted to the general hospital obstetric unit, there being no maternal deaths. As Table I shows, there were almost as many for which no cause was discovered, as there were of those with accidental haemorrhage and placenta praevia. That is, 45 per cent were unexplained, a figure similar to that found in the Royal Maternity Hospital, Belfast, and elsewhere.

Table I

Toxaemia	5 cases	1% Accidental
Intra-partum	1 case	
Postmaturity	2 cases	
Anaemia and W.R. +	1 case	
Placenta praevia	5 cases	½%
Unexplained	12 cases	1.3%

The perinatal mortality rate in mothers with antepartum haemorrhage was 20 per cent, while in general it was 0.6 per cent. Its distribution is shown in Table II.

Table II

	Incidence of APH	*Perinatal mortality*
Primiparae	5 cases (2.7%)	2 cases (1%)
Intermediates	14 cases (3.3%)	1 case (0.2%)
Gr. multiparae	7 cases (7%	2 cases (2.0%)

Of those infants who died the primiparous mothers had both placenta praevia, the intermediate mother had a stillborn twin. One of the grand multiparae had anaemia and a positive Wassermann reaction and the other had an accidental haemorrhage.

Prolonged Labour

Prolonged labour is by definition one lasting more than 24 hours, and a common cause of concern to the medical attendant. We had 59 cases (7%) of prolonged labour, chracterised by inertia, disproportion, and breech presentation. It is difficult to say when the patient has inertia because she has disproportion, or if she has

inertia because of incoordinate uterine action. At any rate, it was remarkable how simple some forceps extractions for what was thought to be disproportion turned out to be when the strength of the uterus at term is considered. As was expected a much greater percentage of primiparae were involved with prolonged labour than multiparae. The comparison was 13 per cent of primiparae to approximately 5 per cent of multiparae, and less than half (40%) of the cases of prolonged labour were in primiparae.

Delivery by Forceps

Delivery by forceps was carried out in 46 (5.5%) of all cases. Thirty (16%) of all primiparae, 15 (3%) of the intermediates, and less than 1 per cent (1 case) of the grand multiparae were delivered by forceps. Thus two out of every three forceps extractions were performed on primiparae, one being on the after-coming head of a breech presentation which was easily carried out.

Caesarean Section

If one assumes that every baby delivered by means of forceps or Caesarean section would otherwise have been still-born, then the figures show that the perinatal mortality rate would have been 7 per cent instead of 3 per cent. The Caesarean section rate was 1 per cent which equals the national average. There were eight cases, distributed as follows :

Primiparae (2 per cent of total)
1 elderly, 2 disproportion, 1 twins (first not advancing).

Intermediates
1 aged 43, free head, 13 years since last child; 1 disproportion, 1 toxaemia (still-born).

Grand multiparae
I antepartum haemorrhage (placenta praevia).

Postmaturity

It is uncertain what causes postmaturity; perhaps it occurs less often in the debilitated and the under-privileged. In this series the occurrence of postmaturity was 3 per cent, which is about the national average. Diagnosis too, is often uncertain, but it is no doubt an important condition for at 42 weeks the perinatal mortality rate is doubled. This is probably due to increased demand on the placenta, and for the same reason, postmaturity becomes doubly important when it is associated with toxaemia, where additionally there is decreasing placental efficiency.

Serious Foetal Incidents

There were 43 incidents of major importance to foetuses excluding miscarriages, of which there were 41, so that it appears that at least 10 per cent of conceptions are not fully effectual. Of the 43, 27 died (see also Table III) and 16 survived. An analysis of the latter follows:

To primiparae, 6 cases :
atelectasis, delayed collapse*, cardiac abnormality, hydrocephalus, prematurity*, prematurity in twins.

To intermediates, 8 cases :

atelectasis, eclamptic mother* cardiac abnormality, hydrocephalus, 3 exchange transfusions, jaundiced twin.

To grand multiparae, 2 cases :

cerebral haemorrhage, mental deficiency with cleft palate.

(*denotes toxic mother).

Foetal Abnormalities

Twenty cases of foetal abnormality occurred out of 772 deliveries, this being a rate of 2.6 per cent, composed of the infants of 7 primiparae, of 10 intermediates, and of 3 grand multiparae. Eight of these abnormal foetuses died, giving a perinatal mortality rate among abnormal foetuses of 40 per cent, which compares with a rate of 37.5 per cent at the Royal Maternity Hospital, Belfast, but contrasts with a recent very large series in Birmingham, in which the rate was 21 per cent. Of the forty-three serious foetal incidents, 27 of the foetuses concerned died, and of these, 8 (30%) were abnormal, and of the 16 survivors twelve (75%) were abnormal.

In 5 per cent of maternity cases (primiparae 7.5%, intermediates 4.5%, grand multiparae 4%) there was death and/or abnormality of the foetus, not taking into account those infants who, in a year or two were found to be defective, but for whom I have no data.

Table III suggests that the baby of a primipara may be more subject to disaster but perhaps survives it better.

Table III

	Primiparae	*Intermediates*	*Grand multiparae*
Stillborn or died	4.5% (9) 1 in 20	3% (15) 1 in 30	2.8% (3) 1 in 40
Survivors	3.0% (6) 1 in 33	1.8% (8) 1 in 50	1.8% (2) 1 in 50

Causes of Perinatal Mortality

The perinatal mortality rate from all causes was 3.3 per cent, but if there had been no toxaemia it could have been about 2.6 per cent. It is analysed in Table IV. This means that about 0.03 infants per 1,000 births per annum would be saved, amounting to about 10,000 in the United Kingdom, and the maternal mortality rate would be about halved, if toxaemia were eliminated. In this connection, the following facts came to light that out of the 20 abnormal foetuses :

4 of the mothers had toxaemia

2 of the mothers had negative Rhesus factor (but not antibodies)

1 of the mothers was an alcoholic

1 of the mothers had syphilis

1 foetus had toxoplasmosis and was stillborn

1 foetus had haemorrhagic disease of the new-born

TABLE IV

Causes of perinatal mortality

Toxaemia	6 cases
Foetal abnormality	8 cases (2 mothers had toxaemia)
Other toxic conditions	4 cases
Placenta praevia	3 cases
Breech presentation	2 cases
Face presentation and postmaturity	1 case (mother had toxaemia)
Unexplained	4 cases
Total	27 (1 paired)

Maternal Causes		*Foetal Causes*	
in primiparae		*in primiparae*	
Toxaemia	3 cases*	Grossly malformed	1 case
Placenta praevia	2 cases	Hydrocephalus	1 case*
Breech	1 case	Toxoplasmosis	1 case
in intermediates		*in intermediates*	
Toxaemia	2 cases*	Haemorrhagic disease of new born	1 case
Unexplained	3 cases	Cardiac abnormality	1 case
Twins	2 cases	Oesophageal atresia	1 case
Prematurity	2 cases	Defective diaphragm	1 case*
Breech	1 case		
Alcoholic	1 case		
in grand multiparae		*in grand multiparae*	
Toxaemia	1 case*	Syphilitic	1 case
Unexplained	1 case		
Total 19		Total 8	

*denotes toxaemia

It may be reasonable to conclude that some sort of toxic factor accounts for the majority of foetal abnormalities, and is associated with about half of the overall perinatal mortality rate, and one need only consider the danger of using thalidomide to see how true this could be. The remainder are mostly caused by genetic factors including about 1 in 40 from consanguinity. There is no record of the incidence of rubella or other virus infections, and I understand that bacterial infections play no part in the aetiology of congenital abnormalities.

INTERVENTION

A number, 83 (35%) of those requiring special vigilance, were expected to need intervention, the group in which intervention was "unforeseen" until after labour had commenced amounted to 111, and the ratio of "expected" to "unforeseen" intervention was 42 per cent to 58 per cent, no account being taken of external versions, episiotomies and repairs of the perineum. It was disappointing to find that we foresaw intervention in only 2 out of every 5 cases, and although this may be explained to some extent by the Tables V, VI and VII, the ratio would depend largely on the ability to assess a case, especially about 36 weeks. For instance, all forceps extractions have been put under the heading of "Unforeseen" but I have no figures to prove otherwise, and, not surprisingly, the figures are somewhat better in multiparity where there was an equal number of expected and unforeseen

instances of intervention; because there is usually a past history, examination is easier and therefore so is prediction.

TABLE V

PRIMIPARAE

Expected Intervention		*Unforeseen Intervention*	
Artificial rupture of membranes	10	Forceps extraction	30
Medical induction	4	Ante-partum haemorrhage (including 2 cases of placenta praevia)	5
Breech delivery	2	Post-partum haemorrhage	12
Caesarian section	2	Caesarian section	2
Missed abortion	1	Manual removal of placenta	0
Total	19 (30%)	Total	49 (70%)

INTERMEDIATES

Expected Intervention		*Unforeseen Intervention*	
Artificial rupture of membranes	26	Forceps extraction	15
Medical induction	1	Ante-partum haemorrhage (including 1 case of placenta praevia)	16
Breech delivery	17	Post-partum haemorrhage	5
Caesarian section	1	Caesarian section	2
Shirodkar suture of cervix	2	Manual removal of placenta	5
		Ectopic pregnancy	1
		Appendectomy followed by miscarriage	1
		Pneumonia followed by eclampsia	1
		Suicide (10 days post-partum)	1
		Puerperal sepsis	1
Total	47 (50%)	Total	48 (50%)

GRAND MULTIPARAE

Expected Intervention		*Unforeseen Intervention*	
Artificial rupture of membranes	9	Forceps extraction	1
Breech deliveries	6	Antepartum haemorrhage	3
Missed abortions	2	Postpartum haemorrhage	6
		Caesarean section (placenta praevia)	1
		Manual removal of placenta	1
		Maternal death (pulmonary embolus)	1
		Puerperal sepsis	1
Total	17 (55%)	Total	14 (45%)

TABLE VI

Expected and unforeseen intervention

	Primiparae	*Intermed.*	*Gr. multip.*	*Totals*
Normal at all stages	75 (40%)	293 (60%)	53 (50%)	421
Unforeseen intervention	49 (25%)	48 (10%)	14 (14%)	111
Expected intervention	19 (10%)	47 (10%)	17 (16%)	83
Special vigilance including above expected intervention	63 (35%)	139 (30%)	38 (33%)	240
Totals	187	480	105	772

TABLE VII

Intervention before, during and after labour

	Primiparae	*Intermed.*	*Gr. multip.*	*Totals*
Antepartum	20 (25%)	48 (57%)	15 (18%)	83
Intra partum	36 (45%)	35 (45%)	8 (10%)	79
Post partum	12 (37%)	12 (37%)	8 (26%)	32
Totals	68 (35%)	95 (20%)	31 (30%)	194 (25%)

ASSOCIATED CONDITIONS

Association of various conditions, noteworthy especially in relation to toxaemia became apparent even in this small series. For example, out of 16 primiparae with toxaemia, 12 behaved as follows:

4 had inertia
2 had prematurity
3 had an intra-uterine death
3 had post-partum haemorrhage.

There were many similar examples of associated conditions among the intermediates and the grand multiparae. As regards the occurrence of association,

10 per cent of primiparae had two or more conditions,
4 per cent of intermediates had two or more conditions, and
12 per cent of grand multiparae were similarly involved.

SUMMARY

This survey has explored some of the more important aspects of midwifery in an average sized and mainly rural general practice during the decade 1954-1964. Some unexpected observations have been made, especially as regards the high proportion of abnormal midwifery, and still unexplained element in many cases of antepartum haemorrhage. The importance of toxaemia, its relationship to other conditions and in particular foetal abnormalities is noted. It is concluded that toxaemia is still the obstetrician's greatest enemy.

I am indebted to Professor J. H. Pinkerton, Mr. H. I. McClure, F.R.C.S., F.R.C.O.G., and Mr. C. G. Irwin, F.R.C.O.G., for their help and encouragement in the preparation of this paper. Also, I am grateful to the hospital staffs in assembling records, and to the County Antrim Health Committee for extracting our ante-natal records.

WHAT'S NEW IN MIDDLE EAR SURGERY?

By G. D. L. SMYTH, F.R.C.S.
Royal Victoria Hospital, Belfast

Based on a lecture given to the Ulster Medical Society, 29th March, 1966

DURING the past ten years there have been major advances in E.N.T. practice, particularly in the treatment of ear disease. Regretably, we have often failed to communicate these advances to our colleagues, particularly those in general practice. It is hoped to remedy this omission, at least in part, and so describe treatment now available to hard of hearing patients—available not just in London and New York, but in Belfast, at the Royal Victoria Hospital.

The term "chronic suppurative otitis media" (C.S.O.M.) is used to include all forms of chronic middle ear disease including cholesteatoma where there is perforation of the drum, hardness of hearing and permanent or recurrent discharge. This disease demands treatment by surgical means and surgical means alone. Just as the standard treatment of appendicitis is by surgery—though until the era of Charles Mayo this was not so—so the treatment of chronic middle ear disease must now also be surgical. Operation should be carried out early, because so frequently the destructive process advances without hindrance, often with recurrent discomfort and pain, and may eventually render the patient very hard of hearing in the affected ear. This is the more serious if both ears, as is often the case, are affected. The prevalence of the condition is not always fully realised, but it can be gauged by the fact that 7 per cent of candidates for the British Armed Forces in World War II were rejected on the grounds of this disease. Again, the importance of early surgical treatment is stressed by the fact that at least two patients died in Belfast during 1965 from complications of chronic suppurative otitis media. Recurrent outpatient therapy by toilet, drops or powders is really only procrastination, and the results of persistence with such well-tried and unsatisfactory treatment is a sheer waste of time, and can never rival the results obtained by skilled surgery.

The treatment of this condition is primarily preventive. Chronic suppurative otitis media almost always has its origin nowadays in upper respiratory infection when this causes chronic Eustachian obstruction. It is clearly very important to treat adequately persistent adenoid enlargement, chronic sinusitis and nasal allergy, particularly during the first two decades of life. In addition to treatment of the primary condition there must be supplementary rigorous tuition in the act of auto-inflation to reopen the Eustachian tube and aerate the middle ear. When upper respiratory conditions, neglected in childhood, result in C.S.O.M. in the second and third decades of life then surgical treatment of the ear itself is urgently called for.

Simply, this is tympanoplastic surgery and consists of removal of all diseased tissue from the middle ear cleft, followed by reconstruction of the drum and sound conducting mechanism using a combined approach through the mastoid and through the external auditory meatus. The operation which I currently practice has been evolved from the tympanoplasty technique developed by Wullstein and Zoellner in Germany in 1953. Worldwide disappointment with early results from the German operation has led to much alteration of the original concepts and as a result we

have "combined approach tympanoplasty" which is a practical method devised by surgeons in Los Angeles, Cologne and Belfast, eliminates discharge and pain, and eradicates the disease in practically every case. In "combined approach tympanoplasty" the anatomy of the outer and middle ears is maintained and the cursed post-operative mastoid cavity is avoided. In some cases there are still difficulties in restoring useful hearing, but thanks to the generosity of the Royal Victoria Hospital and Northern Ireland Hospitals Authority, the problems are being tackled in the Research Laboratory of the Eye and Ear Clinic, and we hope these will, as a result of this work and work in other centres, be gradually overcome. Failure most often occurs where the disease is far advanced—there is a definite inverse relation between the extent of the disease and the hearing improvement which can be gained from operation. We hope that, in future, results of operation will be improved by the early referral of all patients with ear symptoms for specialist opinion. Delay in surgical treatment usually means progression of the disease with destruction of the middle ear structures and often irreversible toxic damage to the organ of Corti. As far as the results of these operations are concerned, I have to report that in over 400 cases, 320 have been followed up from 6 months to 5 years, and of these 98 per cent have closure of the perforation and eradication of the disease process. In 75 per cent socially adequate hearing was obtained as a result of the operation, but unfortunately complete loss of hearing occurred in 2 per cent.

Finally, to close my comments on chronic suppurative otitis media, I must emphasise that, in my opinion, the only worthwhile treatment for this disease is surgical. Antibiotics given either systematically or topically, except as short-term treatment of an acute flare-up of a chronic condition, or where there are complications such as intracranial spread, have no place whatever in the treatment of chronic middle ear disease.

Until this new form of surgical treatment was developed, many of these patients were eventually treated surgically by radical and modified radical mastoidectomy. These were excellent and trustworthy operations when well performed, but strictly limited in their advantage. Granted, they could be relied upon to prevent intracranial complication, clearly a most important consideration, but they failed to improve hearing and indeed often greatly worsened it. In addition, the patient was often left with that enigma of modern surgery—the wound which will not heal. Surveys in several large E.N.T. centres in the U.S.A. and Great Britain have shown that a high proportion of patients who have been treated with modified radical and radical mastoidectomy—often as great as 40 per cent of them—have post-operative cavities which are either permanently or intermittently infected. Certainly even the best cavities only remain healthy if wax and epithelial debris are periodically removed by an otologist. Every head cold, hairwash or swim threatens the stability of the cavity; outbreaks of fungal infection occur and are difficult to cure. Many hours are spent in the care of these ears, and the problem arises as to how to deal with the unhealed post-operative mastoidectomy cavity when this is already a fait accompli. Fortunately the muscleplasty operation developed by Rambo of New York offers a satisfactory solution. In this, after total clearance of the mastoid cell system, a pedicled graft fashioned from the temporalis muscle and adjacent periosteum is mobilised and rotated into the cavity. This obliterates the dead space leaving a 'meatus' of near-normal dimensions.

Some replacement of muscle by fibrous tissue occurs in the first year following operation with shrinkage of the 'filler', but fortunately the lining of the resultant slowly-forming small cavity has an excellent blood supply and remains healthy. This improved blood supply is the main reason for the success of the operation. This operation has been performed in 60 ears—50 patients have been followed-up from 6 to 24 months, and the operated ears of all but two patients, one of whom has intractable gross facial acne and dandruff, have remained clean and healthy throughout. This is gratifying, as in every case the cavities were grossly infected and very troublesome to both doctor and patient.

Otosclerosis is a disease where hardness of hearing is caused by the formation of abnormal bone in and around the footplate of the stapes. Formerly it was treated surgically by fenestration of the lateral semicircular canal, which was introduced as a practical operation by Lempert of New York in 1938, and rapidly adopted by otologists throughout the world. This was an excellent operation, the first of the microsurgical techniques, but its successful application was limited to early cases of the disease, and even in these there was a fairly high relapse rate. At best, the resultant hearing was always about 20 per cent below normal, due to loss of amplification by the middle ear mechanism which is by-passed in the fenestration operation. In addition, the patient suffered severe vertigo for some weeks post-operatively, and was obliged to visit his surgeon at intervals for the rest of his life for care of the large post-operative mastoid cavity, a high proportion of which never completely healed.

The outlook for sufferers from otosclerosis was considerably brightened when in 1953 Rosen of New York introduced mobilisation of the stapes. This operation had an initial success rate of about 60 per cent, but unfortunately about half of these patients subsequently relapsed. However, in 1957, Shea of Memphis, Tennessee took progress in otosclerotis surgery a large step forward when he showed that by removal of the whole stapes and its replacement with a piece of vein laid over the oval window, and a polythene strut between the incus and the vein, conduction hearing loss was eliminated in 90 per cent of ears. He found, however, that in severe forms of the disease (10 per cent) this operation was often followed by early return of symptoms, and often inner ear damage. This, he and others discovered, was usually the result of irritation of the diseased bone by the surgical procedure. Shea, therefore, in 1963 introduced the teflon piston operation —an ingenious modification of his original method—in which, after removal of the stapes crura, a small hole is made in the stapes footplate, as far away from the otosclerotic bone as possible, and a piston connected to the incus passed through this hole, thus by-passing the fixed part of the ossicular chain. This operation has lived up to all that was hoped of it, and I have used no other method during the past 3 years.

The advantages for the patient of Shea's operation over fenestration are overwhelming. For example, the results are much better—90 per cent are successful in the teflon piston operation as compared with 60 per cent in fenestration. Practically every patient with otosclerosis is a suitable candidate for the teflon piston operation. The post-operative effects are minimal—most patients leave hospital on the fifth day after operation and can resume normal duties in two weeks. Prolonged follow-up is unnecessary, and there are no restrictions on swimming and hairwashing.

The results in 130 teflon piston operations which I have followed-up from 3 years to 6 months—90 per cent got closure of ear-bone gap to 10 d.b. or less—the kind of result which fulfills the hopes of Shea, the originator of the teflon piston operation. In patients in whom there is also severe perceptive hearing loss due to otosclerosis, successful elimination of the conduction component of their hearing loss by operation may not restore socially adequate hearing, because the perceptive loss remains unchanged. However, the operation is still often worthwhile as it is now usually possible to make good use of a hearing aid when pre-operatively it was valueless. Surgeons elsewhere have shown that, in their hands, stapes operations have resulted in damage to either the vestibule or cochlea or both, in a significant proportion of operated ears. A detailed investigation of this risk in Mr. Hunter's and my patients has recently been completed by Mr. Rodney England, who has shown that in these patients the teflon piston operation has caused damage to inner ear function in less than 1 per cent of cases. This can give us confidence to recommend operation to practically all outpatients whose hearing is impaired by otosclerosis—the disturbance to the patient is minimal, and the result is usually satisfactory for the patient.

Acknowledgements

I would like to take this opportunity to acknowledge my gratitude to my teachers, Mr. Kennedy Hunter, Mr. R. S. McCrea, Dr. John Shea of Memphis, Tennessee, and Dr. William House of Los Angeles. Mr. Hunter and Mr. McCrea have been unfailing in their support, both moral and otherwise, during the difficult early developmental stages of the combined approach tympanoplasty operation. Mr. Alan Kerr, Sister McAloon and Sister Ryan of the Eye and Ear Clinic, Royal Victoria Hospital, and our anaesthetic colleagues Dr. Sheila Bell, Dr. Gerald Black and Dr. David Barron have, by their respective skills and many hours of hard work, made our present small progress possible, and I am very grateful to them.

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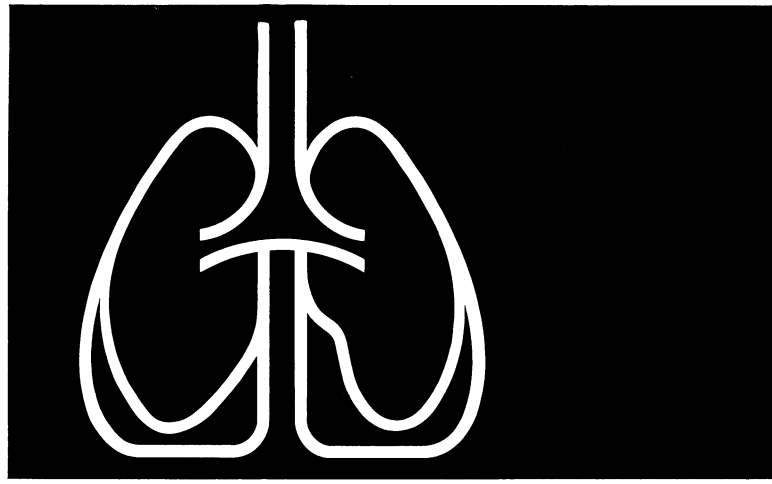

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WHAT'S NEW IN OTOLOGY – THE INNER EAR

By H. W. SHEPPERD, M.B., F.R.C.S., D.L.O.
Royal Victoria Hospital, Belfast

Based on a lecture given to Ulster Medical Society, 29th March 1966

IN Part I Mr. Gordon Smyth has paid tribute to those who have gone before us and pioneered the advances which he has just described for the middle ear and which I am going to describe for the inner ear, and I would like to add my tribute to his.

Symptoms

These are tinnitus, vertigo and deafness, occuring either singly, in pairs, or all together.

1. *Tinnitus*

This is always associated with some degree of hearing loss, and its cause is unknown. The treatment is therefore to treat the cause of the deafness whether this be of middle ear or nerve type. Frequently, however, the tinnitus fails to improve and this had led to a host of procedures for its treatment, none of which are lastingly beneficial. Many patients seek advice, because they are afraid that the tinnitus indicates something sinister occuring either in the brain or the ear itself, and once they can be reassured on this point they are prepared to live with the noise.

2. Vertigo

By which we mean the hallucination of movement. It occurs in diseases of the labyrinth, eighth nerve, and the central nervous system. For successful early diagnosis and treatment, close co-operation between neurologist and otologist is essential, and a team has been formed here in Northern Ireland consisting of neurologist, neurosurgeon, physician, radiologist and otologist.

Meniere's Disease

The most frequent cause of vertigo apart from motion sickness, is Meniere's disease characterized typically by paroxysmal attacks of vertigo, tinnitus, and deafness. The treatment of this condition continues to be a difficult problem, but there have been important recent advances. It is generally accepted that the temporary diminution of labyrinthine activity, which occurs during a typical Meniere attack is due to a sequence of events involving the blood vessels of the inner ear, a sequence very similar to that which occurs in migraine. It is significent that both Meniere's disease and migraine occur in patients with a similar mental make-up and that psychological factors often appear to have a precipitating effect, and that both conditions may occur together, or alternate in the same patient. The changes in the blood vessels result in some way in an increase in the volume of endolymph, eventually causing dilatation of the cochlear duct and the saccule, the well-documented hydrops of the labyrinth. In the early stages of the disease this is entirely reversible but with repeated attacks the changes become permanent with progressive cochlear damage.

Treatment

A. Medical

(i) The psychological aspects of the disease are very important, and if they are adequately dealt with and the mechanism by which the symptoms are produced explained to the patient, then the actual form of medical treatment does not appear to matter much. A recent survey showed that two-thirds of all the patients in a large group receiving most of the accepted diets, injections, tablets etc., got well; and also that a regime which works well with one doctor will often fail with another. It seems certain that for a successful outcome, an extremely good doctor/patient relationship must be established, and if necessary supplemented by tranquillisers or sedatives.

(ii) The elimination of over-work and excess of alcohol and tobacco is an essential part of all treatments.

(iii) The drugs on which we have concentrated in the last few years are those which are intended to aid the metabolism of the inner ear mainly by improving the blood supply. Perdilatal, Cyclospasmol, and Lipoflavonoid have been most useful in many patients who have come into our care early in their disease before much irreversible inner ear damage has taken place. Treatment with these drugs has been disappointing in other centres, possibly because of inadequate dosage, both in amount and duration, or due to a failure to appreciate that this disease eventually results in structural inner ear change which once established no drug can reverse. We are at present engaged in an extensive comparative study of the therepeutic effects of Lipoflavonoid and Perdilatal which supports these views. Treatment may require to be prolonged, even for years and should be supplemented by frequent and regular visits to the medical adviser for encouragement and support, and when necessary by the use of a mild tranquilliser. Drug therapy is most useful, but it must be emphasised that the sympathy and interest of the doctor is an essential and equally important part of the treatment. By these means the great majority of the patients come to terms with their disability and are able to lead normal lives.

B. Surgical

A few patients however, do not respond and continue to have either disabling vertigo or progressive hearing loss, and it is in these patients that surgery may be helpful. There are two types of surgical treatment in current use which may be described as (i) non-destructive and (ii) destructive.

(i) *Non-destructive*

Suitable when medical treatment has failed to halt the progress of the disease, but useful hearing still exists.

(a) *Decompression.* The first operation in this category is that of Georges Portmann to decompress the membranous inner ear, which is grossly distended in Meniere's disease. The approach to the membranous labyrinth is through the mastoid process and along the posterior intracranial surface of the petrous temporal bone to the saccus endolymphaticus where it lies between the layers of dura. Portmann removed sufficient bone from around the saccus to allow it to expand and reduce the pressure elsewhere in the system. Recently House (1962) in Los Angeles has modified this technique by opening the saccus and inserting

a silicone tube through its medial wall so that one end lies in the saccus and the other in the cisterna magna thus permitting a free flow of endolymph into the subarachnoid space.

This operation has in House's, and later in Austin's hands produced most encouraging results. An alternative, though similar method is that of Plester and Fick who approached the membranous inner ear through the footplate of the stapes making a hole in the saccule with a needle, thereby creating a fistula through which the endolymph can drain out of the saccule into the surrounding perilymph. The results of this procedure in the hands of its originators are comparable to those of House and Austin. The aim is to preserve and even improve the faculties of balance and hearing. At present in Belfast all those patients in whom medical treatment has failed and whose symptoms are progressive, but not yet irreversible, are being treated by this method of Plester and Fick, and the early results are encouraging. Twenty cases have been done with a follow-up of at least six months. Of these 18 have shown improvement in their vertigo. Two have shown no improvement and have subsequently required labyrinthectomy. The results so far as hearing is concerned have been disappointing for it has deteriorated in most patients following operation.

(b) *Sympathectomy*. Another non-destructive type of operation which is probably better known, is cervical sympathectomy in which one removes the upper thoracic and lowest cervical sympathetic ganglia and their connecting chain. The rationale of this operation is based on the theory of a vascular mechanism for Meniere's disease and is supported by the dramatic effects on hearing and vestibular function produced by temporary sympathetic block by novocaine injection in an actual attack. Initially results are encouraging, but the long term results less so, presumably due to the regeneration which has plagued other operations on the sympathetic system. Further, removal of the upper sympathetic chain is a major surgical procedure and is followed by some unpleasant side effects such as brachial neuralgia and Horner's syndrome.

(ii) *Destructive procedures*

These rob the patient of the remainder of his hearing function on that side, and they can only be recommended in preference to decompression when all useful inner ear function is lost. These procedures are:

(a) *Ultrasonic irradiation*. In this the vestibular structures, in particular the end organs of the semi-circular canals, are destroyed by ultrasonics. This treatment was introduced by Arslan of Pardua in 1953, and since then has gained acceptance in certain other centres notably with Angell James in Bristol, and Waltner and Altmann in New York. There have been considerable technical difficulties but many have been eliminated, and in particular damage to the facial nerve is now uncommon. The correct dosage of irradiation is difficult to determine and repeat treatments, which are under local anaesthesia and always unpleasant to the patient, are sometimes necessary. We refer to this method since several patients from Belfast have already been treated for us by Mr. Angell James and it is likely to be a therapeutic method about which we shall hear more in the future. It should be emphasised that it destroys the vestibular end organ leaving the cochlear intact, although it does nothing to halt the progress of cochlear dysfunction. Recent communications with Angell James indicate that he has, in the experimental

animal, been able to use his ultrasonic beam to puncture the saccule and thus it is possible that in some patients his treatment achieves the same end result as the operation of Plester and Fick.

(b) *Labyrinthectomy*. This was introduced by Cawthorne in London and by Day in the United States both in 1943. This operation consists of opening the bony lateral semi-circular canal and destroying the membranous canal inside, with usually complete loss of all inner ear function. As a result the intermittent and confusing bursts from the abnormal labyrinth are stopped and gradually the vestibular nuclei learn to function using the information from only one labyrinth, which fortunately is normal in over 85 per cent of patients. This is reasonable therapy for practically all patients with no useful hearing in the affected ear in that it rids them of their episodes of whirling vertigo and vomiting, and eliminates the annoyance of distorted hearing in the operated ear. It should, however, be noted that at least 25 per cent of these patients subsequently suffer from some degree of intermittent unsteadiness for the remainder of their lives, but most patients feel that this is a reasonable price to pay for their escape from paroxysmal vertigo. It should be emphasised that all destructive procedures take something away from the patient permanently, and that the first aim and consideration must always be towards preservation and improvement of function wherever possible. Thus the primary treatment of all patients with Meniere's Disease is by medical means; if this fails and there is useful inner ear function then we try to preserve this function by a decompression operation, and only when the inner ear damage is irreversible will we consent to labyrinthectomy.

Nerve Deafness

This usually occurs as part of the aging process and is subject to a great deal of individual variation. There are however, many other causes such as congenital disease, head injury, virus infections, exposure to noise and treatment with otoxic drugs. When noise is responsible avoidance is obviously the best treatment but if the patient cannot avoid working in noise then he should wear adequate ear defenders. In this country acoustic trauma is not yet an Industrial Disease, but that time is rapidly approaching. Ototoxic drugs should also be avoided unless very clearly indicated and these include streptomycin particularly, but also kanamycin, and neomycin. The custom in many hospitals of treating acute infections routinely with a combination of penicillin and streptomycin is to be deprecated, particularly in the elderly or in any patient whose renal function may be impaired.

Nerve deafness of sudden onset is frequently unilateral and should be treated as an emergency. It may be due to a virus infection, but more probably has a vascular mechanism in which there is a sudden reduction in blood supply due to vasoconstriction. Some patients recover spontaneously, but many fail to do so and are left with a permanent deafness. Treatment should be by sympathetic block on the affected side, followed up by vasodilator drugs and possibly steroids to reduce cellular oedema. The earlier treatment is begun the better the chance of recovery and ideally these patients should be seen within 24 hours.

In the congenitally deaf there is usually some residual cohlear function, and by the use of powerful hearing aids and special training, including lip reading,

these patients can be taught to hear and to speak. Surgical treatment for nerve deafness, apart from that already mentioned for Meniere's disease is not yet available, although this may change in the future. Experimental work is going on in the United States with electrodes placed in the cochlea and auditory nerve, but this is so experimental at the moment that it can offer no hope to the patient with nerve deafness in the immediate future.

In conclusion a word on hearing aids. The government Medresco hearing aid is a most excellent instrument, but for the majority of patients it is not as good as an ear level one. Further, many patients will hear best by wearing two aids, preferably at ear level. This, when successful gives them binaural hearing which not only enables them to judge the source of the sound but also to pick out from a noisy background that sound which is relevant to them. We must therefore hope that the Health Service will in due course produce an ear-level aid and allow those patients who will benefit from two aids to have them.

Summary

The current treatment of disease of the inner ear with special reference to Meniere's disease and the surgical procedures for dealing with it is discussed with some emphasis on decompression.

I wish to thank my colleagues in the Eye and Ear Clinic and particularly Mr. Gordon Smyth for his help and for allowing me to quote some of his results.

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GROUP TRIAL OF PROPANIDID AS AN INTRAVENOUS ANAESTHETIC

By R. S. J. CLARKE, M.D., B.Sc., F.F.A.R.C.S., and
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SINCE the introduction of thiopentone as an intravenous anaesthetic in 1934, it has been realised that the drug is not completely satisfactory for all types of patients and different durations of surgery. Certain difficulties have been overcome by the realization that by using smaller doses the toxicity is reduced to a minimum. However, the fact remains that while anaesthesia is short, recovery results because the drug is stored away in muscle to be released slowly over many hours. This makes it unsatisfactory for outpatients and even patients in hospital often remain drowsy for long periods after the operation unless there is pain or discomfort.

Search for a shorter acting drug led to the synthesis of methohexitone (Brietal) and in many ways it is ideal for outpatients, both dental and those for minor surgery. However even this drug is not rapidly broken but stored up in the body to some extent, and there is cumulation on repeated dosage (Clarke and Dundee, 1966). It has also the drawback that excitatory effects (twiching, hiccough, etc.) occur, though their incidence can be reduced by slow injection and opiate premedication (Dundee et al, 1961).

Of the non-barbiturates tested, propanidid is the first short-acting drug to pass the rigorous clinical trials. It has been shown to produce few excitatory effects, little hypotension, and recovery is as rapid as one could want (Dundee and Clarke, 1964). It is sufficiently insoluble in water to necessitate the use of a "solubilizing agent", and is available as a 5 per cent solution, which remains stable for over two years. Its value is marred by the viscosity of the solution, the prolongation of the action of suxamethonium and a higher incidence of postoperative sickness than follows the use of barbiturates. In order to assess the usefulness of the drug and the general validity of the above conclusions it was decided to carry out a group trial.

Organisation of the Trial

For this study a number of practising anaesthetists of varying seniority and experience were asked to participate. Each was given a minimum of 20 half-gramme ampoules of the drug and a brief factual summary of its pharmacology. They were asked to use it in their routine practise in place of thiopentone or methohexitone. On completion of the study each was asked to fill in a questionnaire comparing propanidid with the barbiturates. Completed questionnaires, often with detailed reports, were received from 66 anaesthetists, covering 1402 anaesthetics for a wide variety of surgery (Table I).

TABLE I

Grade of anaesthetist participating in the group study (absolute figures) and numbers of each type of surgical case anaesthetised by them.

Seniority of anaesthetist		
	Consultant and S.H.M.O.	35
	Senior Registrar	6
	Registrar and J.H.M.O.	13
	S.H.O.	12
		66

	Induction only	*Main Agent*	*Sole Agent*
Major surgery in all fields	422	15	—
Minor Inpatients	364	70	23
Outpatients	151	18	61
Obstetrics	19	—	—
E.N.T. and I.P. Dentals	115	—	—
Paediatrics	50	2	—
E.C.T.	—	—	92
Total numbers	1121	105	176

The participants were asked to express a preference with regard to various properties and actions, also whether they thought the drug was likely to be of clinical use. If the answer to the latter question was "yes", they were to suggest likely fields of usefulness. Finally, they were asked about difficulties encountered, particularly those met in certain techniques or types of procedures.

RESULTS

The results from the specific questions (Table II) indicate that in the dosage used (4-6 mg/kg was recommended) propanidid was preferred with regard to cardiovascular and respiratory depression by most people. Recovery was almost

TABLE II

Answers to specific questions on propanidid. Figures refer to percentages of anaesthetists.

Action compared with thiopentone

	Propanidid preferred	*Thiopentone preferred*	*No preference*
Cardiovascular depression	48	11	41
Respiratory depression	47	15	38
Recovery	92	0	8
Physical properties	2	80	18
Postoperative vomiting	5	36	59

General opinion of the drug

Likely to be of clinical use	73
Unlikely to be of clinical use	9
Future uncertain	18

universally found to be more rapid after propanidid. On the other hand it was not popular because of its physical properties. The opinions about vomiting were more equivocal, because the majority of participants found no difference in its incidence but of those who did, most people found that thiopentone was less emetic.

The overall opinion of the drug was that it was likely to have a place in clinical use. Of the 48 taking this view 45 mentioned out-patients specifically, 25 mentioned minor in-patients and 4 suggested its use for "poor risk" patients.

The objections to the drug though expressed in many different ways can be classified as in Table III. The largest group encountered was due to its viscosity, which made injection difficult. As one would expect from a short acting drug, there was difficulty in attaining smooth surgical anaesthesia because of the slow uptake of the volatile supplement. Other less common objections are also given in the table.

TABLE III

Percentage incidence of the major difficulties reported by the anaesthetists in the trial.

Objections to the viscosity, "oiliness" or to the necessity for using a large needle	57
Difficulty in take over of inhalation agent owing to brevity of action	41
Postoperative sickness	27
Prolongation of action of suxamethonium leading to problems	19
Excitatory phenomena (muscle movements)	14
Hypotension with large doses or in ill patients	8

DISCUSSION

The method of group trial gives an opportunity of finding out early in the life of a drug whether anaesthetists would really want to use it. The answers to the specific questions must be judged in the light of the information already supplied to the anaesthetists, which may have influenced some of them. There was a high incidence of "no preference" answers to three of the questions. Where there was a definite comment about the rapid recovery, the participant was not asked whether this was a good thing for the patient and some expressed views to the contrary, except for out-patients. The rapidity of recovery led to the difficulty in take over of an inhalation agent (Table III) but this can be overcome with practise.

The physical properties of the agent were mentioned frequently in the general reports of difficulties encountered. The oiliness, tendency to form bubbles and necessity for use of a large needle were all unpopular. The latter is a particular difficulty with children but can be overcome by diluting the drug from a 5 per cent to a 3.5 per cent solution. A less viscous preparation is on trial at present and seems very similar in its other properties to the older solution, except that it can be injected through a 23 s.w.g. (No. 12) needle with acceptable speed. Against these objections, a drug in solution has advantages over the barbiturates, which are only stable in powder form.

The high incidence of postoperative vomiting after propanidid has been noted by Dundee, Kirwan and Clarke (1965) and by Goldman and Kennedy (1964) but many general surveys of its use have not mentioned this complication (Howells et al, 1964; Swerdlow, 1965; Gunner et al, 1965). Certainly the majority of the sickness is in the first hour after the drug and if atropine premedication alone is used, there is little late nausea or vomiting.

The difficulties mentioned spontaneously included prolongation of the action of suxamethonium. For most work this action presents no problems but during bronchoscopy and electro-convulsive therapy, where nitrous oxide is not normally used, the patient may regain consciousness before the relaxant has worn off. This is particularly unfortunate in E.C.T. where the other properties of the drug would have made it very useful.

A high incidence of spontaneous muscle movements have not been encountered by the authors using optimal dosage (4-6 mg/kg) but higher doses or very rapid injection, increase the incidence of these complications. Certainly the use of large doses is no substitute for a technique of rapid administration of inhalation agents after intravenous induction. Some anaesthetists also found hypotension common after the use of large doses and in ill patients. With such it is particularly essential to have a smooth "take over" after the induction and the injection of tubocurarine immediately before the propanidid in major surgery does achieve this result. In low dosage the drug has less cardiovascular toxicity than thiopentone and could be used for poor-risk cases though it is doubtful whether this is a specific indication.

Many of the anaesthetists who suggested using the drug for outpatients had not themselves used it for this purpose but all of those who had (25) were satisfied with its action. As sole agent for the extraction of 1-2 teeth and for the rapid incision of an abscess the drug is ideal and as main agent for slightly larger procedures it is satisfactory. Another field where the rapid recovery is most valuable is in the electrical conversion of cardiac arrythmias where even if a second shock is necessary the duration of sleep just matches the duration of the procedure. No premedication is necessary and in this field as with most outpatients where the drug was used vomiting has been very rare.

Summary

A group trial of the new intravenous anaesthetic, propanidid, is described in which 66 anaesthetists took part. The main difficulties encountered with the preparation were its high viscosity, its brief action and, less consistently, early postoperative vomiting. Now that a less viscous preparation is available it would appear that the drug has a definite place for outpatient and brief in-patient anaesthesia.

Addendum

Since this study was completed the less viscous form of propanidid has been marketed in the name of Epontol. The lesser viscosity probably affects the clinical acceptability of the drug.

We would like to thank all the anaesthetists who took part in the trial, and Dr. Donald Whitfield of Farbenfabriken Bayer AG for generous supplies of propanidid.

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THE YELLOW FEVER OF IRELAND

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PHYSICIANS practising in Ireland will be surprised to find Ireland listed as a country subject to yellow fever in a comparatively recent publication. Carlos Finlay, ever to be remembered for his work on yellow fever, died in 1915, and in 1955 his medical school, Jefferson Medical College, held a commemorative conference on yellow fever. Doctor Pedro Nogueira (1955) reviewing the history and geography of the disease remarked that "Doctor Graves left a great description of the havoc it played in Dublin in 1826".

It is quite true that Graves in the first volume of his Clinical Lectures on the Practice of Medicine described yellow fever in Dublin, but MacArthur (1957; 1959) has emphasised that the yellow fever of Ireland was relapsing fever. According to MacArthur what Graves was describing was louse-borne relapsing fever with jaundice. The epidemic described by Graves occurred at the height of the winter of 1826-27 in December and January. It does not seem likely that any mosquitoes, let alone the usual vectors, would have been available at that time of the year in Dublin to transmit yellow fever, even if there had been a human source of the virus. Further, according to Graves, the deaths he observed amounted to "nearly twenty". This is not the mortality to be expected of epidemic Central American yellow fever.

Like many another Nogueira has been misled by the difficulty in nomenclature. There is not one yellow fever but several yellow fevers—classical yellow fever of the Caribbean and Central America (the vomito negro of the Spanish), relapsing fever, viral hepatitis and leptospirosis. All four are micro-organismal. All but leptospirosis can occur in large epidemics, leptospirosis being more restricted in locale and more occasional in time. If this error is not to continue, some synonym in English should be found for the Central American mosquito-transmitted viral disease. It is interesting that Noguchi himself thought he had discovered the cause of classical yellow fever when he discovered a leptospira in a jaundiced patient, another example of confusion of one febrile jaundice with another.

It may make little difference if in the military medical appreciations of the powers Ireland is marked as having "yellow fever". It might deter an enemy. It will cause him to dissipate his resources in inoculating his invading troops with yellow fever vaccine. It would be bad, however, for the tourist trade if this false belief persisted. The only communicable "yellow fevers" we see nowadays are viral hepatitis and occasionally leptospirosis. We may safely assume that Central American yellow fever never occurred in Ireland, and happily we can say that relapsing fever has gone too.

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HOUR-GLASS STRICTURE OF THE STOMACH AND IRON DEFICIENCY

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" . . . non contenti . . . ulcera agitare, rerum quoque naturam ex aliqua parte scrutati sunt." Celsus. De re medica.

THE object of this note is to put forward the hypothesis that hour-glass stricture of the stomach occurs in persons with a chronic simple gastric ulcer of the lesser curvature, because these persons also have chronic iron deficiency, and that the stricture would not occur without the co-existence of the iron deficiency. Seven cases are described. An association with Plummer-Vinson stricture of the cervical oesophagus and with duodenal stricture is recorded. The X-ray appearances are reproduced. The haemoglobin and serum iron estimations are recorded. Gastroscopy was done in one case. One case was operated on.

Important reviews of hour-glass stomach include those of Moynihan (1904) Hurst and Stewart (1929) and Bockus (1943).

Hour-glass deformity of the stomach occurs almost exclusively in women. It is due to a stricture of the body of the stomach, by which the greater curvature is deeply drawn in medially towards a point on the lesser curvature. At that point there is, or has been, a chronic simple ulcer. It is probable that the adhesion of the gastric ulcer to adjacent structures provides a fixed point to which the contracting scar draws in. The stricture does not occur unless there has been at some time a chronic simple ulcer. It divides the stomach into an upper and a lower loculus, neither of which is contracted or sclerosed. The passage between the two loculi may get very small, but total obstruction, if it ever occurs, must be rare. Hour-glass stomach does not correctly describe the appearance, because the stomach is not waisted symmetrically at the mid-point of its lumen, but rather eccentrically at the lesser curvature. The appearance is reminiscent of that of the Plummer-Vinson stricture in the cervical oesophagus as seen in lateral X-ray views. Normally a chronic simple gastric ulcer heals with a little local scar. The scar that causes the hour-glass narrowing is an abnormal scar, and is submucosal.

The gastric ulcer is not often very active, but it rarely heals, and some ulcer pain is a regular feature. Difficulty in eating is common. This is partly because of the small size of the upper loculus which produces "small stomach" symptoms, and partly because of obstructive feelings. These the patient may interpret as difficulty in swallowing. Impaired nutrition and impaired strength follow. Plummer-Vinson stricture may co-exist, to add cervical oesophageal dysphagia to the difficulty in eating, and to add a particular difficulty in eating meat. As a rule the gastric mucosa is hypotrophic or atrophic. Gastric mucosal secretory activity is low. Malignant change at the site of the scar does not seem to happen. This is an important difference from Plummer-Vinson stricture.

The incidence of hour-glass stomach has fallen of recent years. This may be due to a fall in the incidence of gastric ulcer, to improved treatment of iron deficiency, to better medical treatment of gastric ulcer, or to earlier surgical

treatment. It is uncommon now in this country for a chronic simple gastric ulcer to go for long unhealed and not operated on.

The stricture is usually discovered at a barium meal examination. It is seldom suspected clinically. Examination with a barium meal should include views in the supine and prone positions. If the stricture is extreme, it will be seen in the erect position. If it is not, it may easily be missed, though the associated ulcer may be identified. The supine view is the most valuable. It regularly demonstrates the stricture when it cannot be seen in erect pictures or in prone pictures. This because in the supine position the barium distends the upper end of the lower loculus and the lower end of the upper loculus, and the gap between is easily seen. This is important both for the initial examination, and for subsequent examinations when the ulcer and the stricture are being reviewed. Occasionally in the erect position a bulge of the upper sac may obscure the narrow area. It is less likely that, when the patient is lying down, a bulge of the lower sac may overlie it. Sometimes the delay occasioned by a very narrow passage keeps not only the upper sac but also the oesophagus full of barium for longer than usual. Nevertheless, there is no obstruction at the gastro-oesophageal junction. Hour-glass deformity is sharply distinguished from the other scar complications of gastric ulcer. It is not at all like sclerosis of the lesser curvature or "coffee pot" stomach (which is also a complication of lesser curvature gastric ulcer) and not like the concentric contraction of the antrum, which is a complication of antral ulceration. Neither these nor carcinoma are likely to give any difficulty in diagnosis of the hour-glass stricture. Rarely there may be a considerable deformity of the mid-stomach due to extensive or multiple simple ulcers, but this too is not confusing. The appearance of hour-glass stricture is highly distinctive and is constant. Once the stricture is formed, it never goes away, whether the ulcer heals or not.

Case Reports

Case 1. C.H. Married woman. Born 1931.

She had three children one of whom died of tuberculous meningitis. Her husband is said to have pulmonary tuberculosis. Her first Royal Victoria Hospital attendance was in 1960 when she was twenty-nine. She was complaining of upper abdominal pain and anorexia. Her weight was ninety-one pounds. Because of the family history and of a sedimentation rate of 95 mm. in one hour, attention was directed to tuberculosis as a possible cause of her complaints, but none was found. Her haemoglobin was 71 per cent. Red cells 3.9 millions. M.C.V. 88 cubic microns. M.C.H.C. 30 per cent. She was advised to take iron but her haemoglobin in November 1960 was 67 per cent and in December was 71 per cent. In November 1961 a barium meal (figure 1) showed a chronic simple lesser curve gastric ulcer. In June 1965 she came to medical extern because of "lumps on the legs". Weight was one hundred and sixteen pounds. Sedimentation rate 2 mm. in one hour. Haemoglobin 80 per cent. Serum iron 45 micrograms per 100 ml. Barium meal (figure 2) showed marked hour-glass deformity. She did not consider the menstrual loss remarkable.

Summary: This case shows progression from having the ulcer only to development of the stricture in five years. It is probable that the patient was iron deficient all that time.

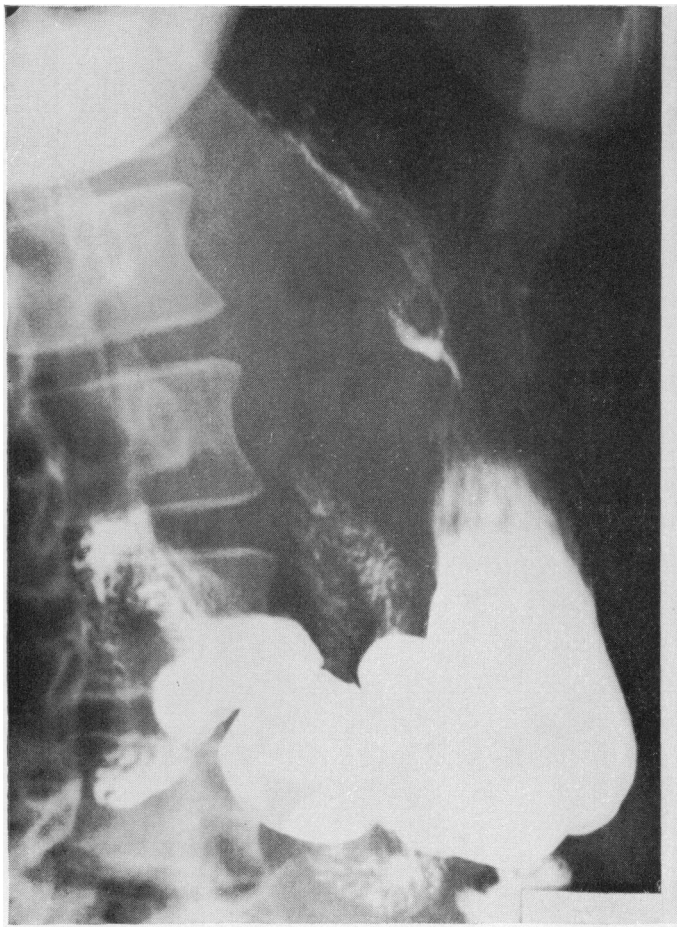

FIG 1. *Case* 1, *in* 1961, *showing lesser curve gastric ulcer*

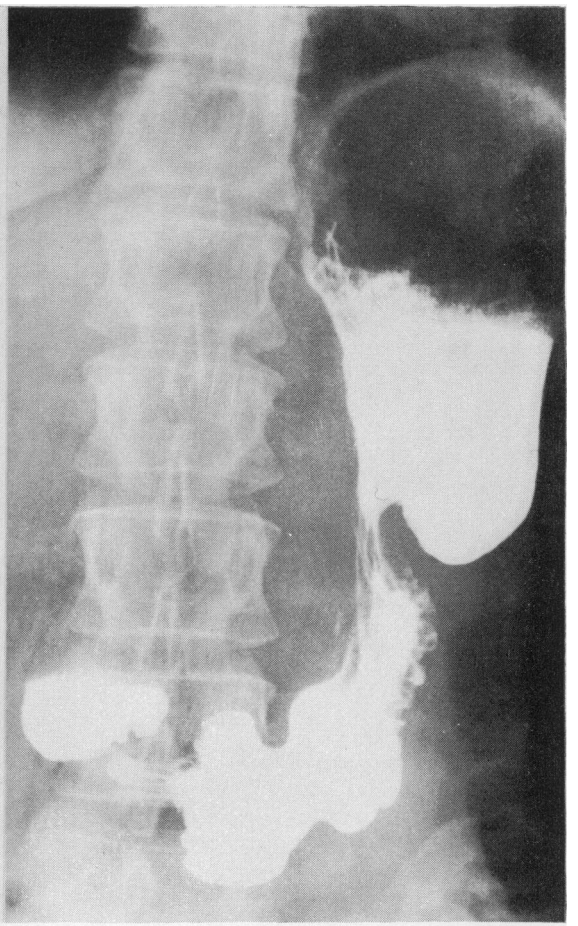

FIG 2. *Case* 1, *in* 1965, *showing hour-glass stricture*

Case 2. E.C. Single woman. Born 1898

She first came to medical extern in 1958 complaining of upper abdominal pain and vomiting. She had had "indigestion" for thirty years, and the pain had lately been bad. There was a doubtful history of rheumatism in the past. Weight was one hundred and seventeen pounds. The barium meal (figure 4) showed a chronic simple lesser curvature gastric ulcer with an hour-glass stricture. The mucosa was hypotrophic. A duodenal web could be seen (figure 3) as a deep narrow incisura of regular outline drawn into the lesser (upper) curvature of the cap from the greater (lower) curvature. In 1960 she came to medical extern complaining of epigastric pain. Weight was one hundred and nineteen pounds. She had been taking an aspirin powder for headache. Haemoglobin 71 per cent. Red cells 4.6 millions per c.mm. M.C.V. 84 cubic microns. M.C.H.C. 28 per cent. The barium meal showed again the ulcer, the adjacent narrowing, and the duodenal web (figure 5). In 1961 she was still having attacks of pain, and the barium meal showed the hour-glass constriction (figure 6). Weight was one hundred and twenty-six pounds. In 1962 the pain was bad. Weight was one hundred and eighteen pounds. In 1963 the pain was bad and she was admitted for surgical treatment. Haemoglobin 80 per cent. Serum iron 99 micrograms per 100 ml. Weight one hundred and seven pounds. No test meal was done. At operation a penetrating

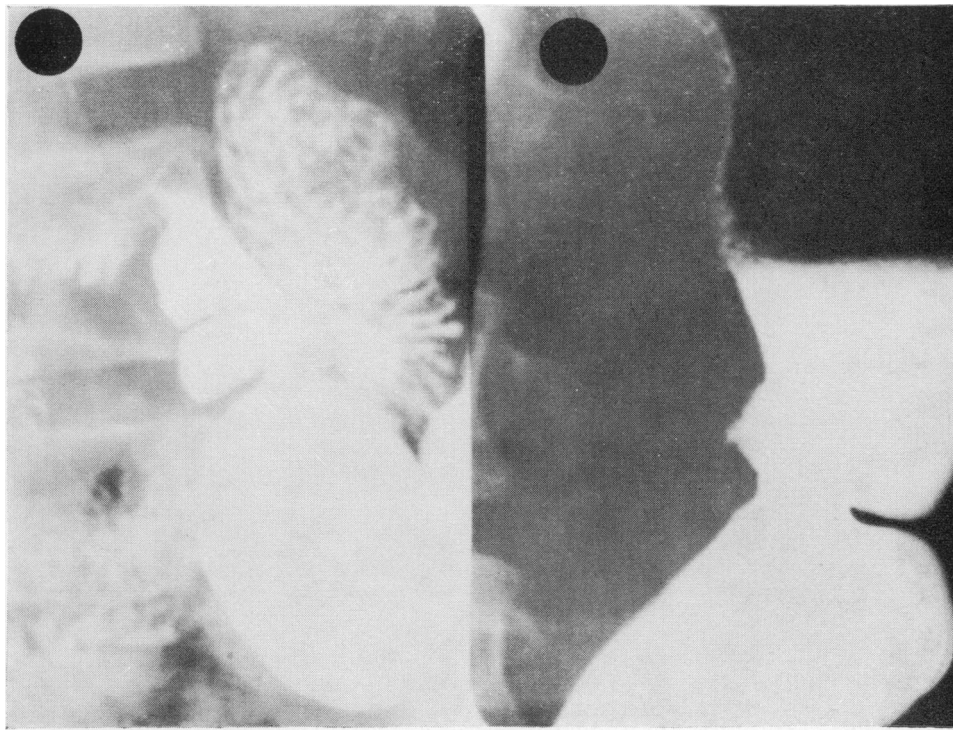

FIG. 3. *Case* 2, *in* 1958, *showing duodenal stricture*

FIG. 4. *Case* 2, *in* 1958, *showing lesser curve gastric ulcer and hour-glass stricture*

FIG. 5. *Case* 2, *in* 1960, *showing the duodenal stricture*

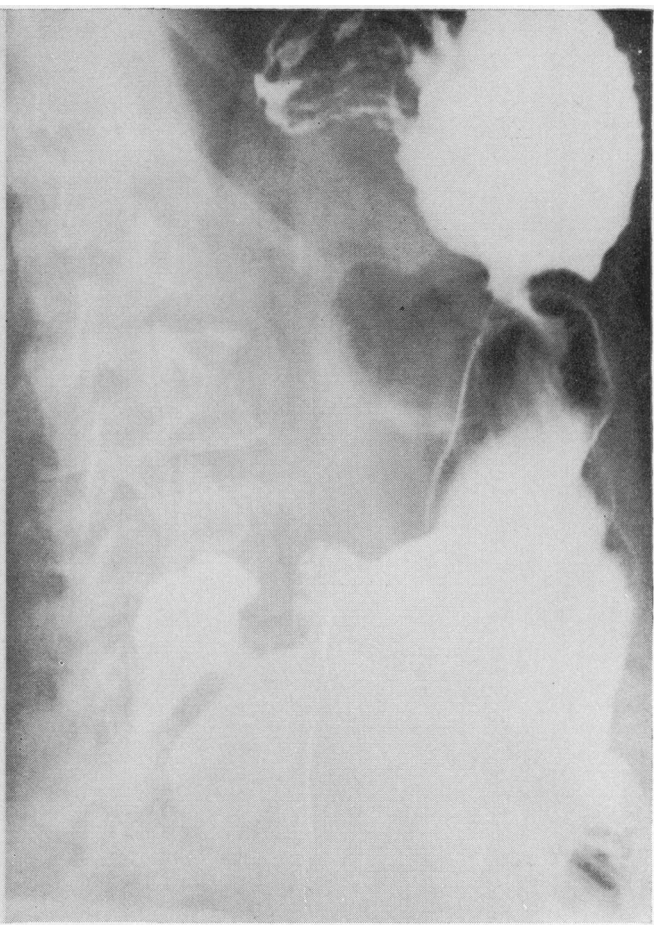

FIG. 6. *Case* 2, *in* 1961, *showing the hour-glass stricture*

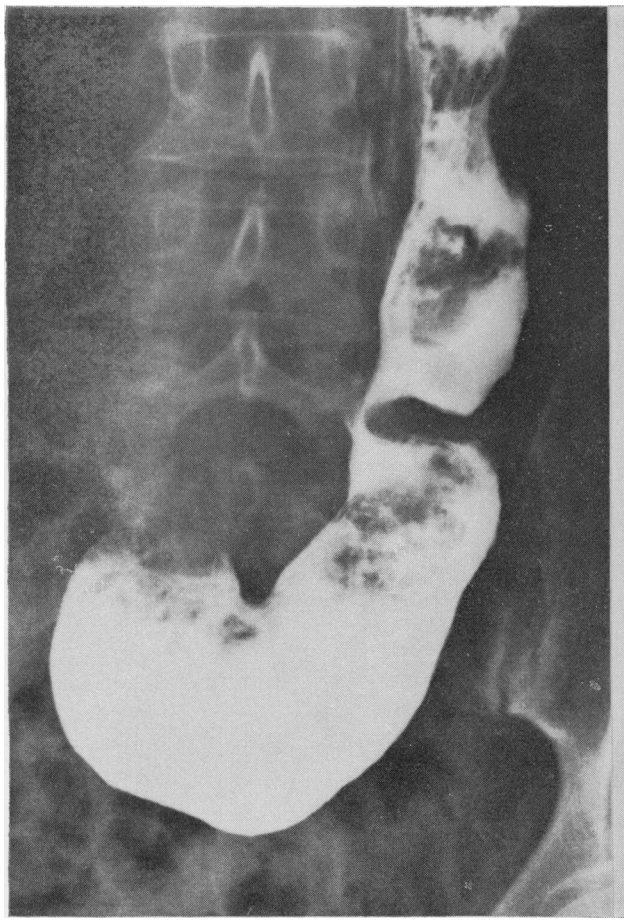

FIG 7. *Case 3, in 1965, showing the hour-glass stricture, erect*

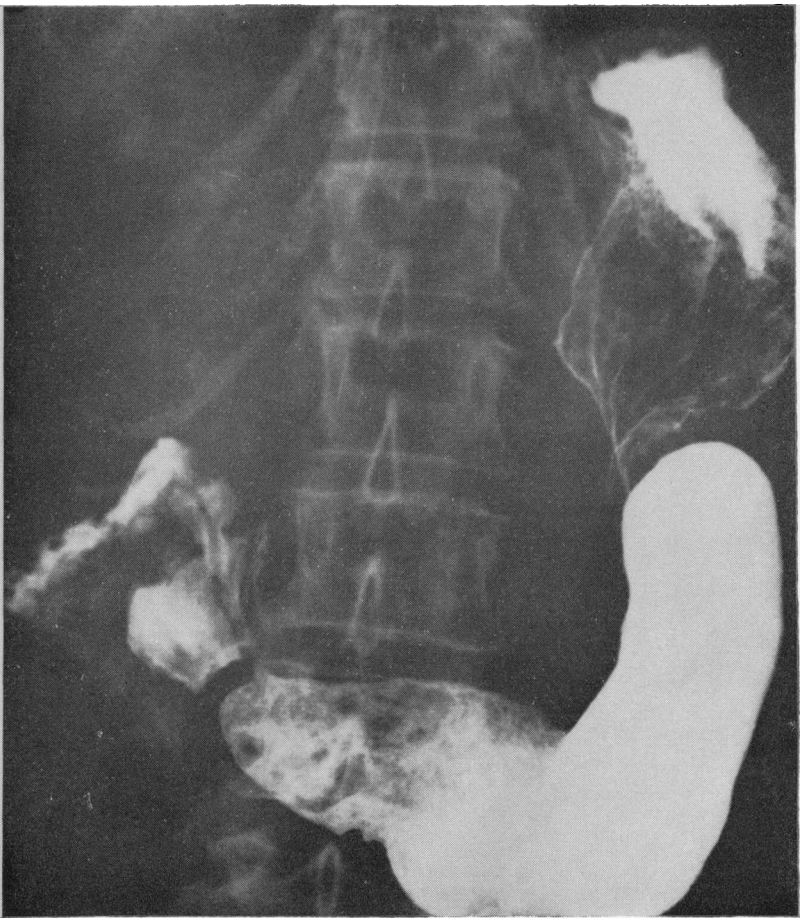

FIG. 8. *Case 3, in 1965, supine view showing the upper end of the lower loculus filled*

gastric ulcer was found with the stricture, corresponding to the X-ray appearances. A partial gastrectomy of Bilroth I type was done by Mr. Morrison, with a gastro-duodenal anastomosis.

Summary: Chronic simple gastric ulcer, hour-glass deformity, stricture or web in the first part of the duodenum, iron deficiency, anaemia and calcified fibroid. The duodenal web was present at the first examination. The hour-glass stricture was also present then.

Case 3. F.McA. Married woman. Born 1896. 2-para.

She first came to the Royal Victoria Hospital in 1964, complaining of loss of weight and abdominal discomfort of thirty years standing. She had no dysphagia. The heart sounds were normal. Haemoglobin 77 per cent. Red cells 4.0 millions per c.mm. M.C.V. 89 cubic microns. M.C.H.C. 31 per cent. Serum iron 40 micrograms per 100 ml. A gruel test meal showed no free acid. Barium meals showed a probable small lesser curvature gastric ulcer with a marked hour-glass deformity (figures 7 and 8). The mucosa was hypotrophic. She was treated with a soft diet of 4,000 calories and with vitamin supplements and with the injections of iron and cyanocobalamin. After she left the ward she did not follow the planned treatment. In March 1965 the haemoglobin was 83 per cent, the serum iron was 57 micrograms per 100 ml., the weight 103 pounds.

Summary: Lesser curvature gastric ulcer, hour-glass deformity, achlorhydria, iron deficiency, anaemia, monoarthritis of shoulder. It cannot be said when the hour-glass stricture formed.

Case 4. E.A. Married woman. Born 1897.

In 1956 a barium meal examination (figures 9 and 10) showed a lesser curvature chronic simple gastric ulcer. No stricture was demonstrated. At that time her weight was one hundred and fifteen pounds, the haemoglobin was 82 per cent and the M.C.H.C. 30 per cent. In 1958 (figure 11) the narrowing could be seen. The gastric mucosa especially in the antrum seemed hypotrophic. In 1962 she came to medical extern complaining of upper abdominal pain and weight loss. Weight was one hundred and ten pounds. Haemoglobin 62 per cent. The barium meal showed the lesser curvature ulcer and an hour-glass deformity (figure 12). There was a duodenal diverticulum. In 1964 she was admitted to the Royal Victoria Hospital. She had been taking aspirin. Haemoglobin 82 per cent. Serum iron 85 micrograms per 100 ml. A gruel test meal showed a very low acid curve—no specimen containing over 10 m.eq./litre of hydrochloric acid. Barium meal showed the stricture as before, and the mucosa even more had an appearance of strophy (figure 13). In 1965 cervical oesophageal narrowing was demonstrated (figure 14). She was treated by prohibiting aspirin and aspirin compounds, by the injections of iron and cyanocobalamin and by maximum feeding. Since then she has been improved. The haemoglobin is 92 per cent. Serum iron 115 micrograms per 100 ml. Heart sounds normal.

Summary: Chronic simple lesser curvature gastric ulcer with hour-glass stricture, cervical oesophageal narrowing, atrophic gastric mucosa, hypochlorhydria, iron deficiency, anaemia, old right mastectomy, emphysema possibly due to flax dust inhalation, periarthritis of the shoulder. It seems that the stricture formed between the age of fifty-nine and sixty-one.

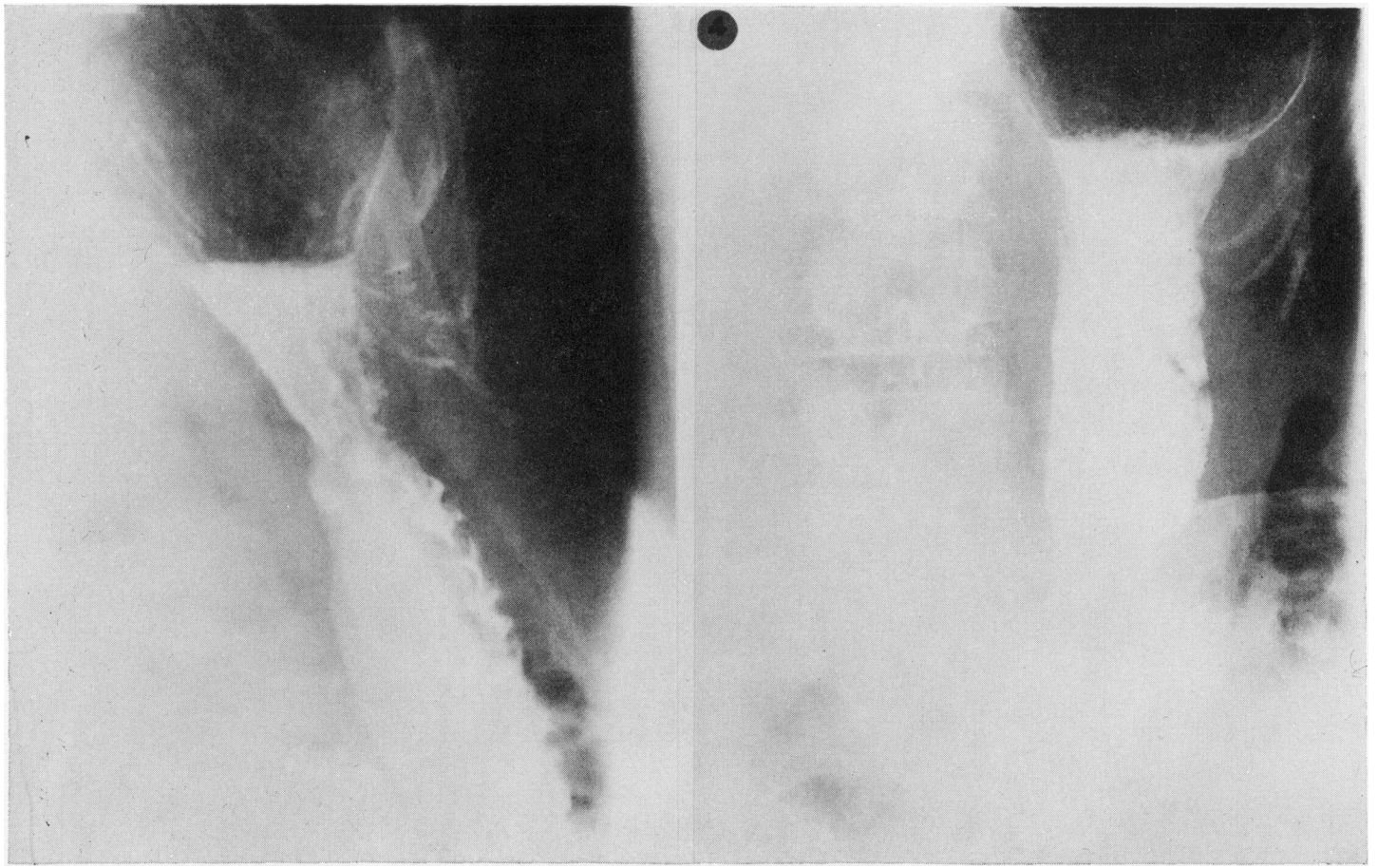

FIG. 9. *Case* 4, *in* 1956, *showing a lesser curve gastric ulcer*

FIG. 10. *Case* 4, *in* 1956, *not showing a stricture*

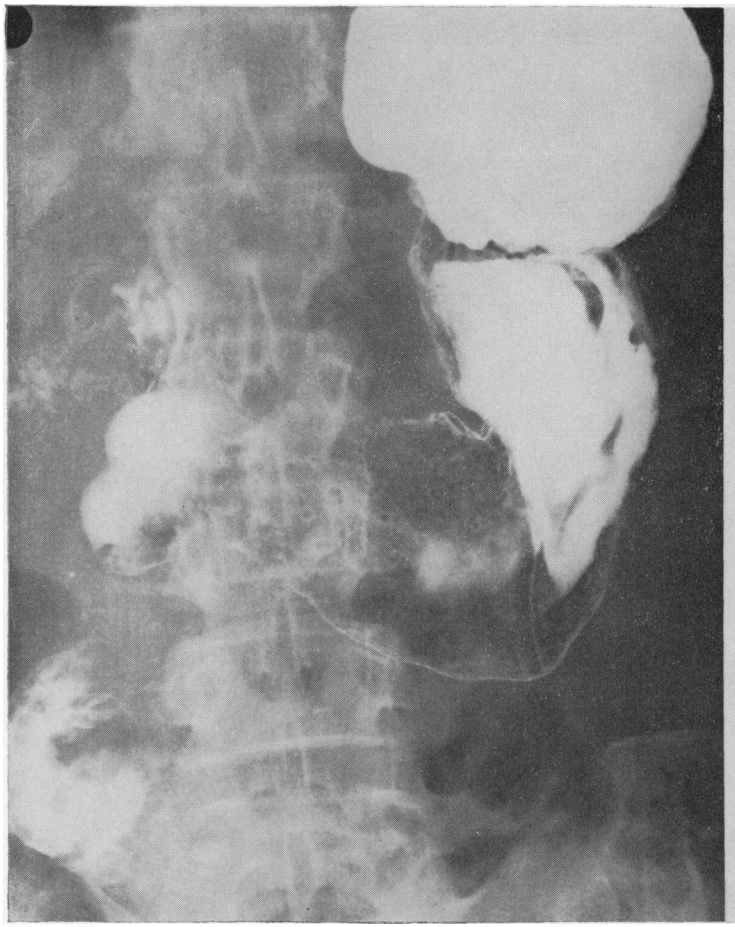

FIG. 11. *Case* 4, *in* 1958, *showing hour-glass stricture.*

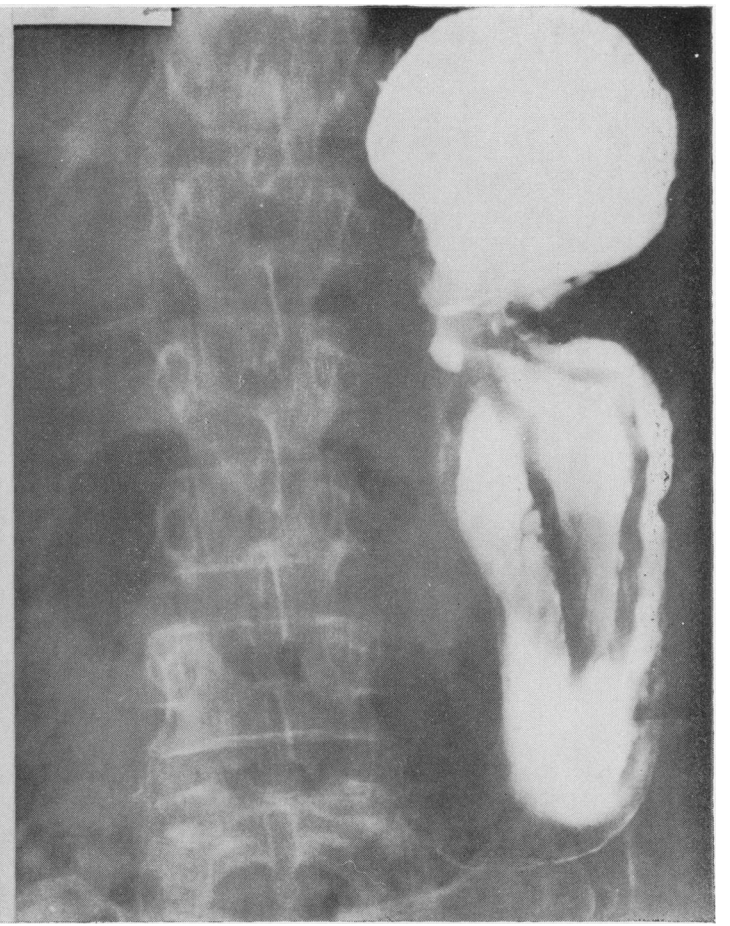

FIG. 12. *Case* 4, *in* 1962, *showing the gastric ulcer and the stricture*

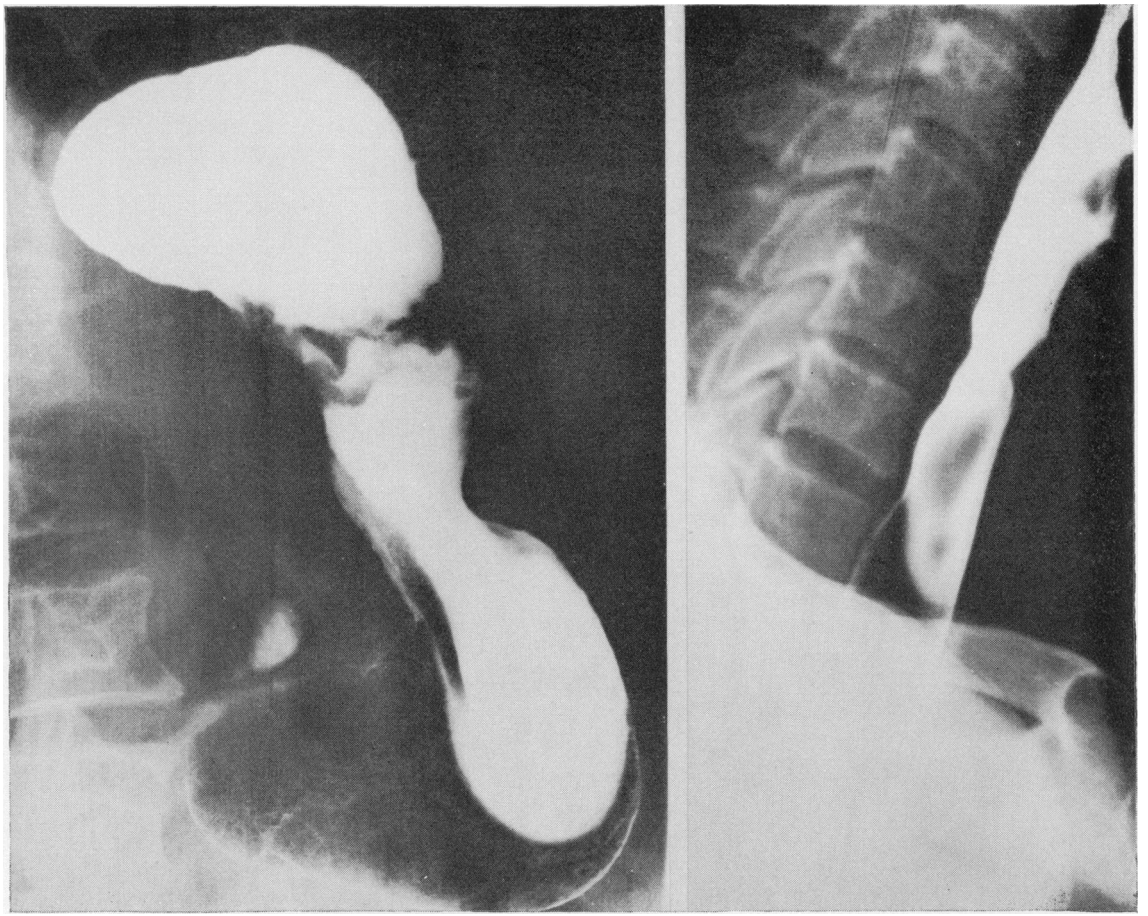

FIG. 13. *Case* 4, *in* 1964, *showing the stricture and hypotrophic mucosa*

FIG. 14. *Case* 4, *in* 1965, *showing cervical oesophageal narrowing*

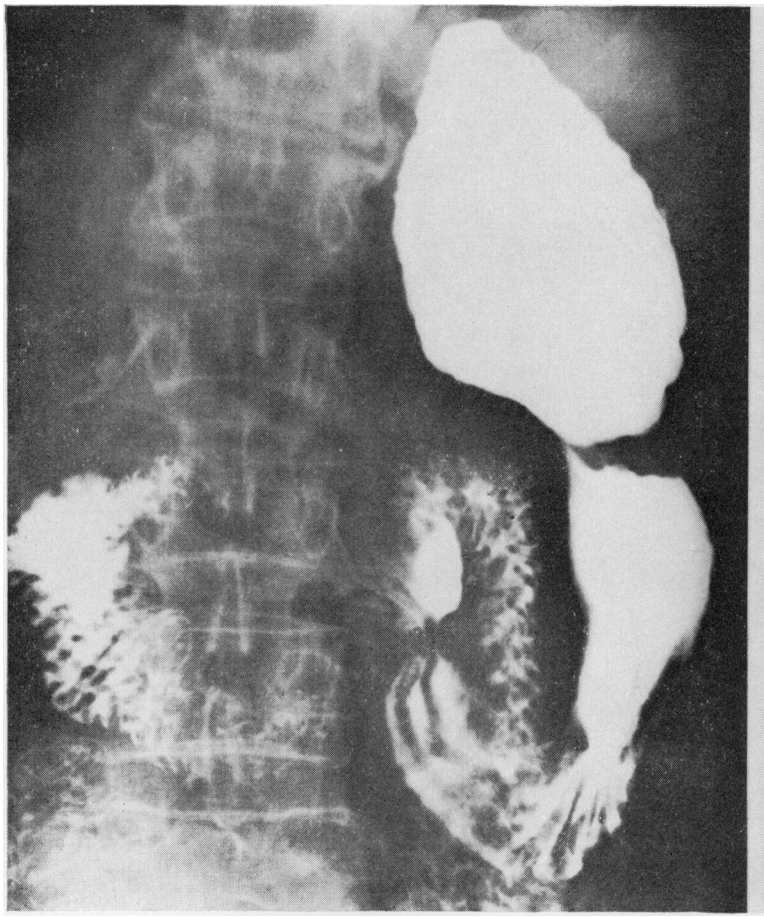

FIG. 15. *Case 5, in 1956, showing the hour-glass stricture*

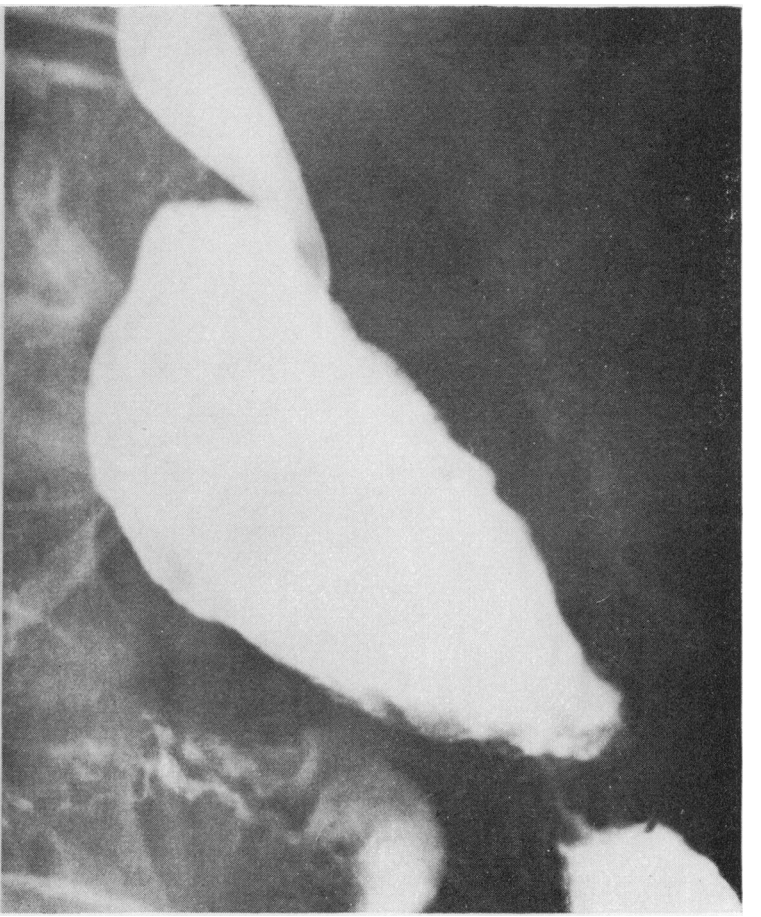

FIG. 16. *Case 5, in 1964, showing hour-glass stricture*

Case 5. M.McC. Married woman. Born 1881. 7-para.

She complained for many years of anorexia, abdominal pain, vomiting after meals, loss of weight and loss of energy. She had had more than one haematemesis in her twenties and thirties. She had been told, after a barium meal examination in 1929, when she was forty-eight, that she had an hour-glass stomach. Her first Royal Victoria Hospital admission was in 1952 when a gruel test meal showed no free acid, a barium meal showed an hour-glass deformity, and gastroscopy (the view was only of the upper loculus) gave a view of the constriction. The mucosa was hypotrophic and no ulcer could be seen. Barium meal X-rays are available for 1956 and 1964 (figures 15 and 16). They show the stricture. An ulcer is not seen. The pyloric channel and cap are normal, as is the antrum. The haemoglobin in 1952 was 88 per cent, 102 per cent in 1954, 105 per cent in 1956, 92 per cent in 1960, and 94 per cent in 1964. On review, in 1964, when she was admitted for congestive heart failure, the serum iron was 40 micrograms per 100 ml. She described some dysphagia which she located in the cervical oesophagus. X-rays showed some narrowing in the cervical oesophagus (figure 17). There was evidence of mitral regurgitation, as there had been for some years.

Summary: Hour-glass deformity of stomach, cervical oesophageal narrowing, iron deficiency, mitral regurgitation. It seems certain that the stricture formed during the child-bearing years.

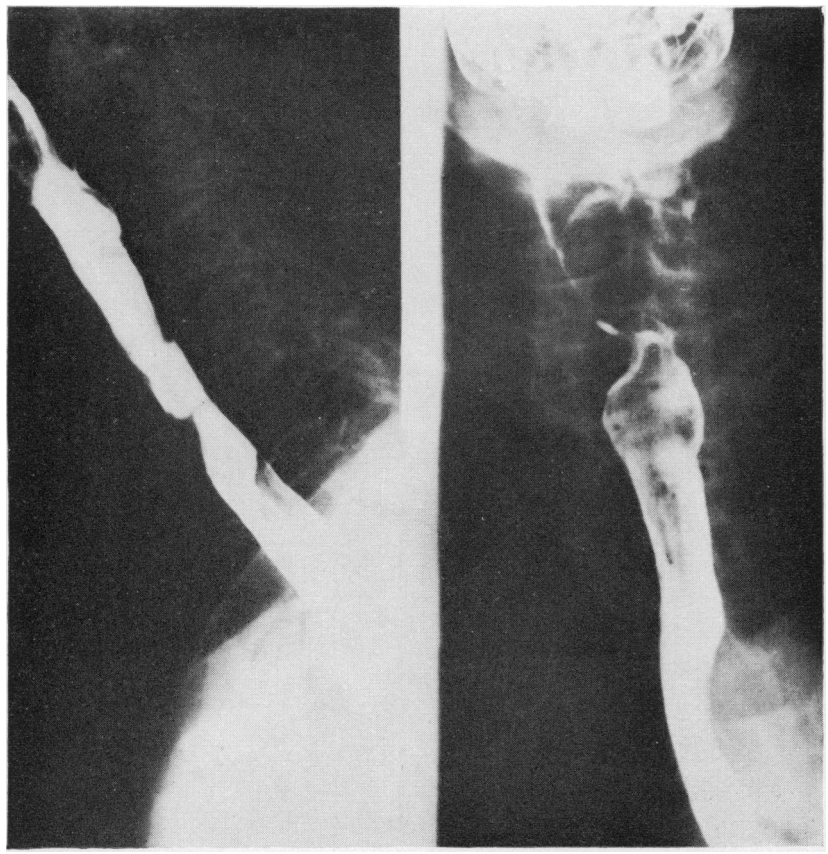

FIG. 17. *Case 5, in 1964, showing cervical oesophageal narrowing*

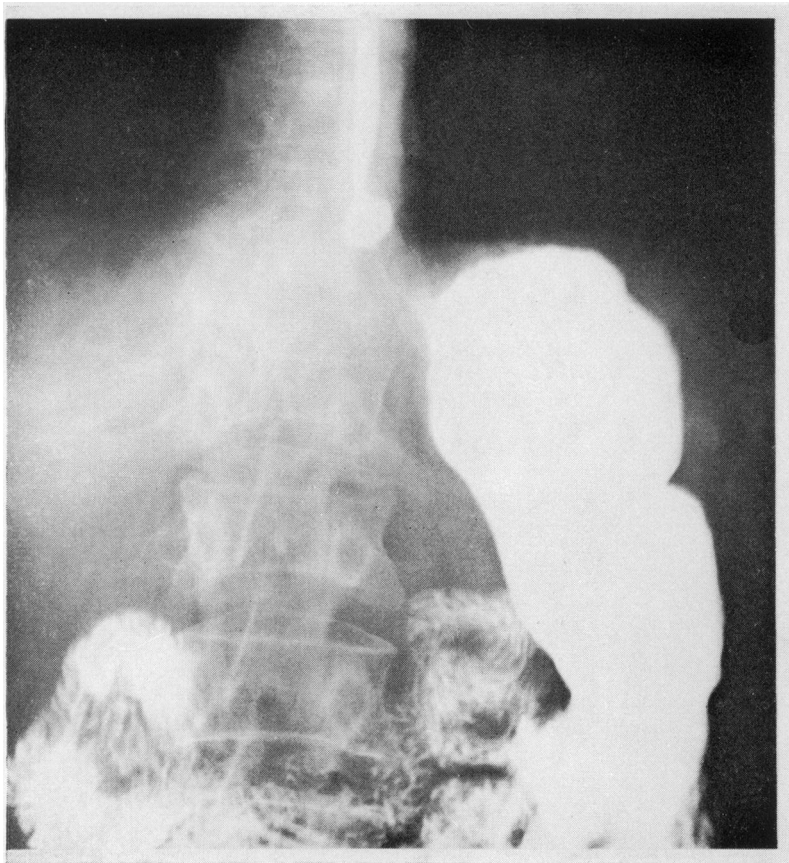

FIG. 18. *Case* 6, *in* 1957, *showing the beginning of the gastric stricture*

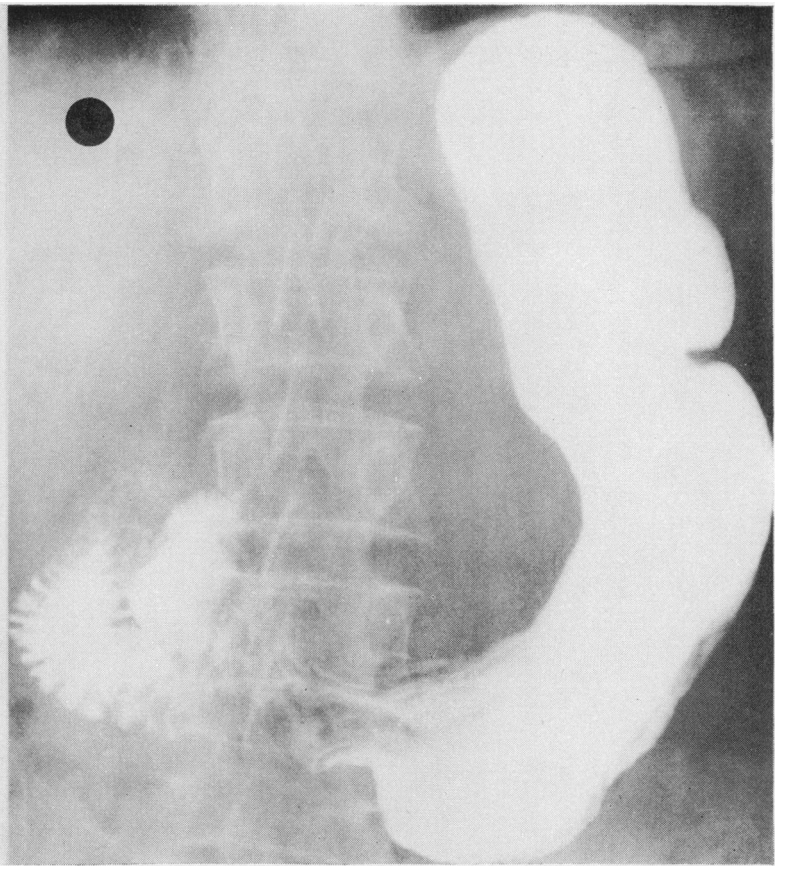

FIG. 19. *Case* 6, *in* 1958, *showing progression of the stricture*

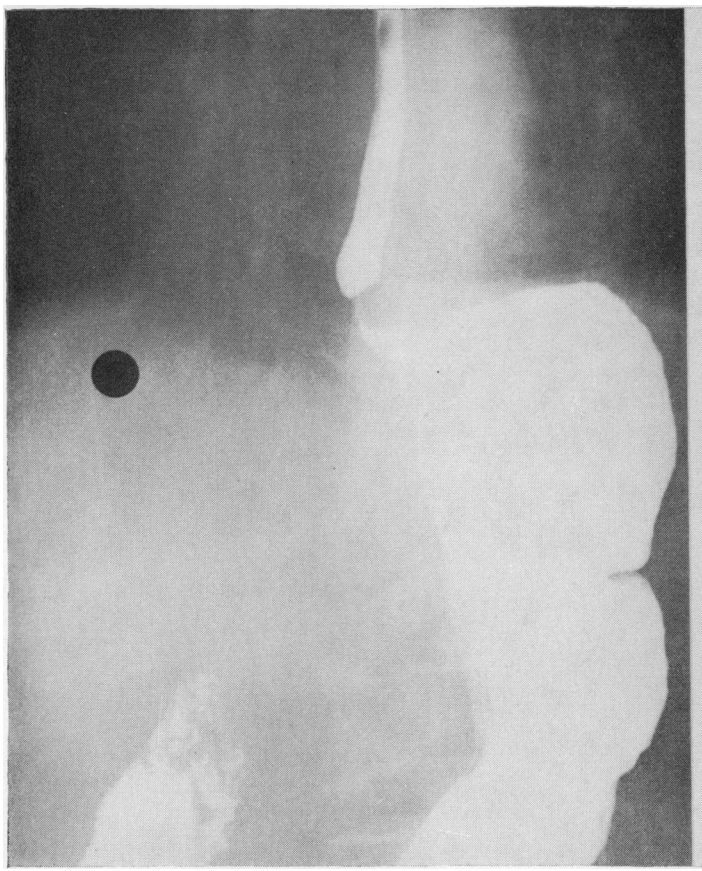

FIG. 20. *Case* 6, *in* 1962, *showing the stricture, hardly more advanced than in* 1958

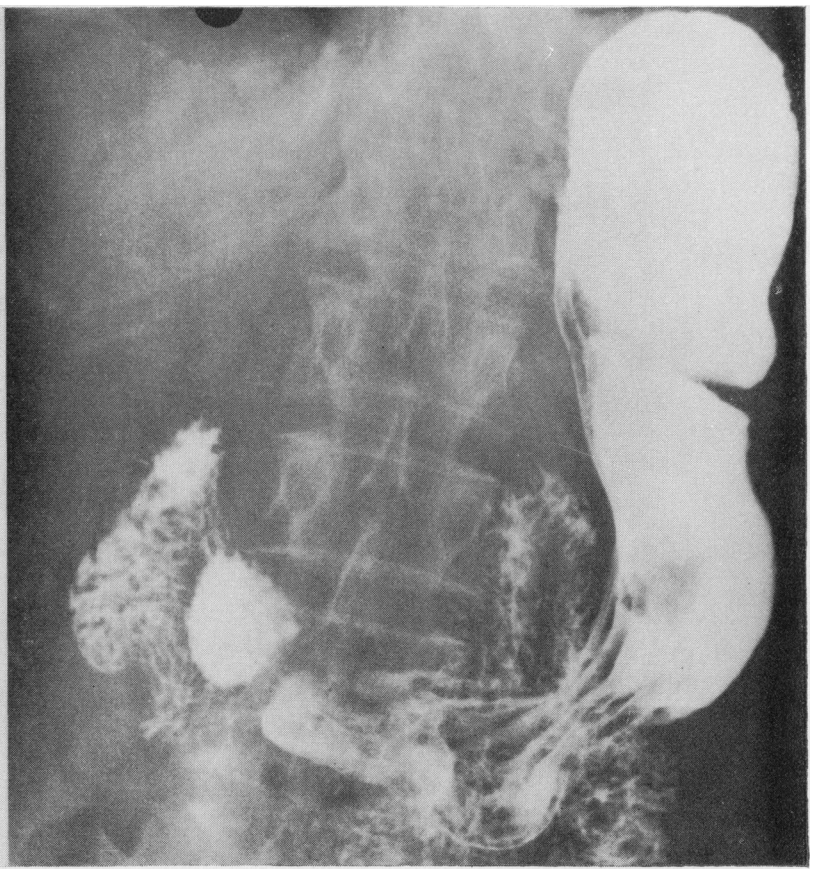

FIG. 21. *Case* 6, *in* 1965, *showing the stricture more advanced*

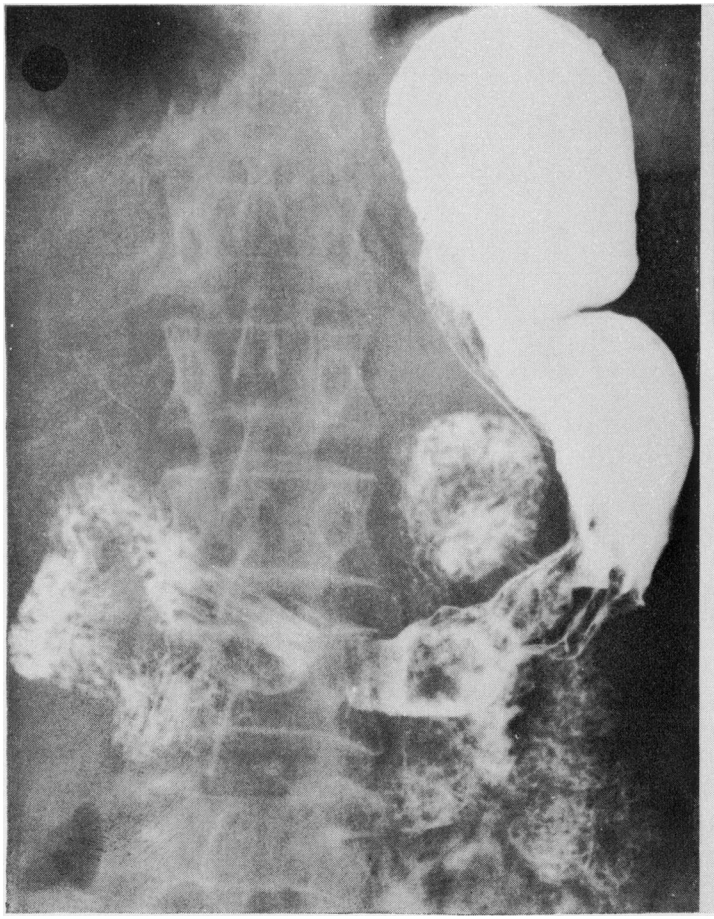

FIG. 22. *Case 6, in 1965, another view of the stricture*

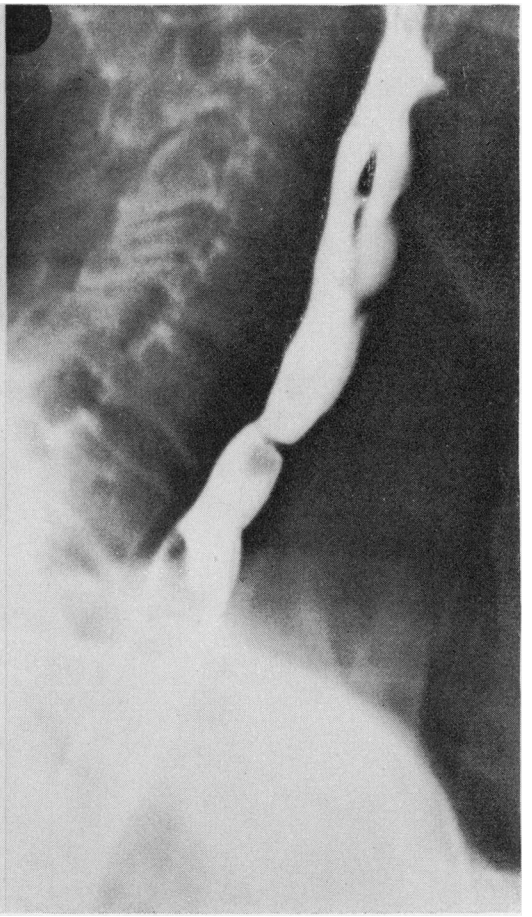

FIG. 23. *Case 6, in 1965, a view of the cervical oesophageal strictures*

Case 6. M.M. Widow. Born 1899. 3-para.

She was first examined in 1949 when she was complaining of a cervical oesophageal dysphagia which was shown to be due to Plummer-Vinson stricturing. She was reported as Case 2 in a previous paper (Bingham and Logan, 1953). Her doctor had treated her for anaemia, sore mouth and angular hacks with iron, including an injection of iron, and vitamins. Her general condition improved but the dysphagia was severe. The Plummer-Vinson stricture was dilated in 1949, and, because the dysphagia recurred, on several occasions since. She was treated off and on with iron, and with cyanocobalamin. In 1957 she mentioned that food "lay" in the upper abdomen, and there was some pain. However, this was transient, and epigastric pain has never been a trouble. In 1959 Mr. Bingham observed at oesophagoscopy that there seemed to be some narrowing in the lower oesophagus. In 1962 it seemed that only a fine bougie could be passed into the stomach. Barium meal examination at those times did not show disease of the lower oesophagus. She was reviewed in 1965 because the cervical oesophageal stricture had recurred. This is seen in figure 23. The stomach at this time was seen to have an indentation of the greater curvature (figures 21 and 22) and on review of the plates it could be seen in those of 1957, 1958 and 1962 (figures 18, 19 and 20). This is believed to be a partial hour-glass stricture. That it did not progress may have been due to partial correction of the iron deficiency, or to a lesser curvature ulcer being transient only. Having the Plummer-Vinson stricture may have protected her from the ulcerogenic effect of aspirin and iron tablets, because she only intermittently could swallow them after a dilatation, and then only for a little while, till the stricture recurred. Her haemoglobin in 1949 was 103 per cent. In 1954 it was 90 per cent. In 1956 it was 84 per cent and the M.C.H.C. was 33 per cent. In 1959 it was 85 per cent. In 1965 it is 82 per cent, the M.C.H.C. 30 per cent and the serum iron (the first ever done) 50 micrograms per 100 ml. It is possible that her iron deficiency was never corrected.

Summary: Plummer-Vinson stricture of the cervical oesophagus, partial gastric stricture, iron deficiency.

Case 7. J.K. Single. Age 92.

This patient was admitted because of a recurrence of abdominal pain. She was transiently jaundiced. Probably the illness was due to gallstones. She had had several hospital admissions and attendances, partly because of her age and infirmity, and partly, among other things, for abdominal pain. The first barium examinations include only erect pictures. The presence of a gastric abnormality may be suspected in the plate of January 1958 (figure 24). A lesser curvature gastric ulcer is seen in April 1958 (figure 25). A supine picture in August 1959 (figure 26) shows the hour-glass stricture well. In 1965 (figure 27) the stricture is still seen. In January 1958 a gruel test meal showed no more than 10 milliequivalents of free acid in any specimen. The haemoglobin, in 1958, was 91 per cent and the M.C.H.C. 31 per cent. In July 1959 the haemoglobin was 81 per cent and the M.C.H.C. 30 per cent. In August 1959 the haemoglobin was 85 per cent and the M.C.H.C. 31 per cent. In November 1959 the haemoglobin was 86 per cent, the M.C.H.C. 32 per cent. In November 1965 the haemoglobin was 84 per cent, the M.C.H.C. 32 per cent and the serum iron was 51 micrograms per 100 ml. In

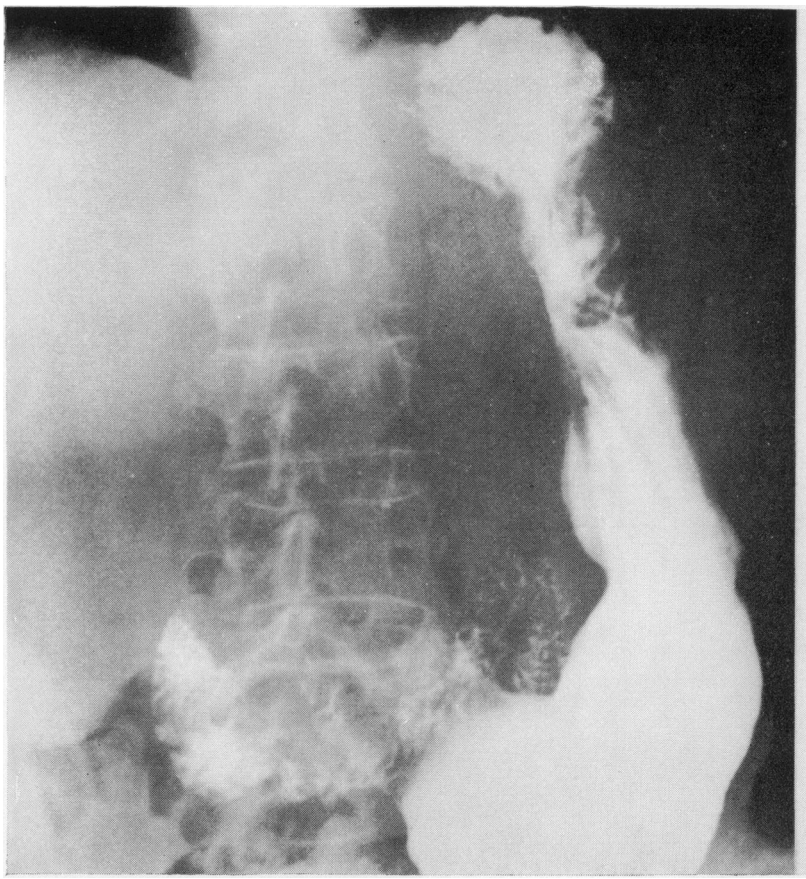

FIG. 24. *Case 7, in 1958, erect view, suspicious of gastric abnormality*

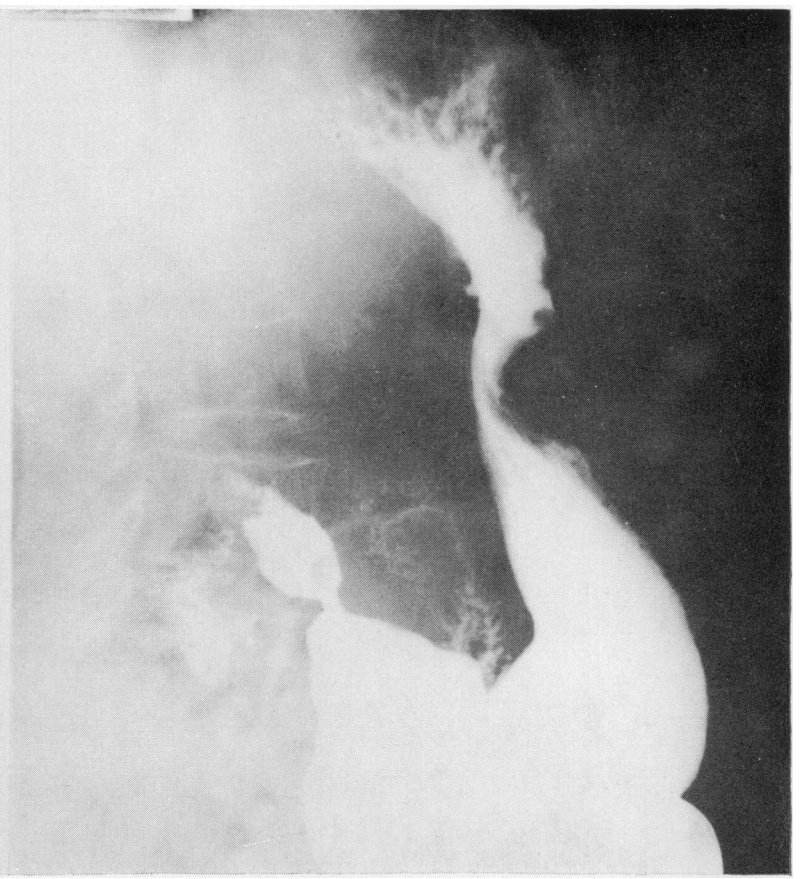

FIG. 25. *Case 7, in 1958, showing a lesser curve gastric ulcer*

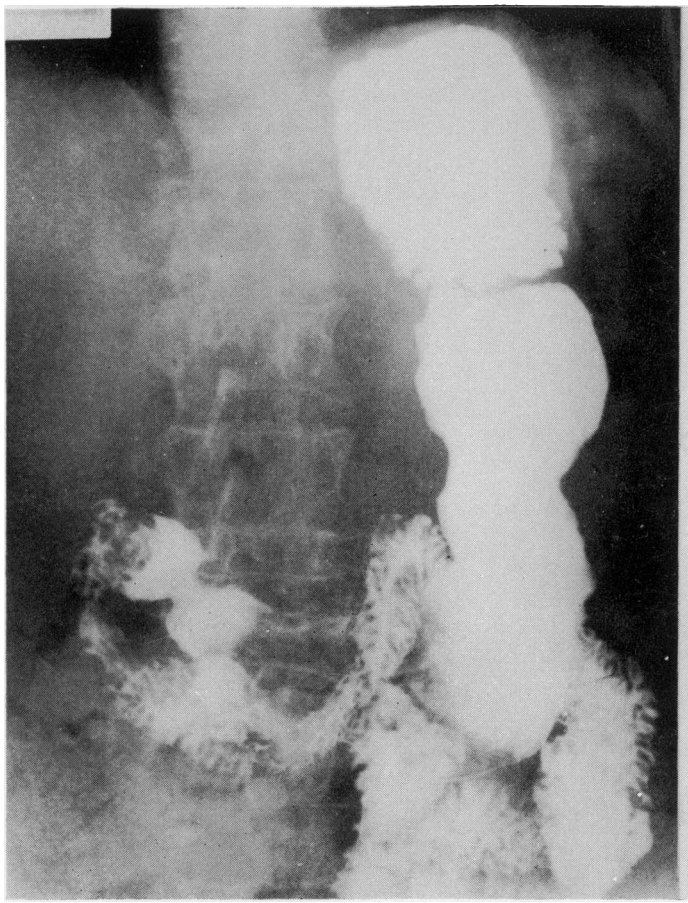

FIG. 26. *Case 7, in 1959, supine view showing an hour-glass stricture*

FIG. 27. *Case 7, in 1965, the stricture is still seen*

July 1959 she had cyanocobalamin injections and in December 1962 she had injections of Imferon. It cannot be said when the hour-glass stricture formed.

Summary: Lesser curvature gastric ulcer, hour-glass stricture, iron deficiency.

Discussion

In view of the association described between hour-glass stricture of the stomach and Plummer-Vinson stricture of the oesophagus and stricture of the duodenum, it is reasonable to think that some general tendency to stricturing in the upper alimentary tract exists in these patients, and that the general tendency occasions abnormal submucosal scarring when, and only when, some local lesion appears, such as the lesser curve ulcer in the case of the stomach. One may think some similar if inconspicuous local lesion occasions the similar scarring in the cervical oesophagus and in the duodenum. All three sites are probably points of special impact of food and drugs, or in the case of the duodenum of the gastric expulsive stream. The question is, is it a reasonable hypothesis that iron deficiency is the cause of the general tendency to stricturing? Is it responsible by itself (or inter alia) for the development of the hour-glass scar in certain cases of lesser curvature gastric ulcer? The hypothesis finds support in the evidence of iron deficiency in the cases reported, in the co-existence of the Plummer-Vinson stricture long thought to be caused by iron deficiency, in the exclusive or almost exclusive incidence in women, and in a clinical impression that iron administration facilitates the normal healing of lesser curvature gastric ulcers. It would be helpful if in future all case records of hour-glass stricture detailed the history of the iron state. Reports of cases of lesser curvature gastric ulcer in patients with long continuing iron deficiency which did *not* form an hour-glass stricture would also be helpful in testing the hypothesis.

The duodenal stricture in case 2 is quite unlike chronic duodenal ulcer deformity, and does resemble hour-glass stricture of the stomach and the Plummer-Vinson stricture. Moyhihan's case XV had what he described as an hour-glass duodenum as well as two gastric strictures. The form of duodenal stricture however is not clear from his report. It could have been as in the present case, or it could have been a duodenal ulcer wasp-waist deformity (Gutmann 1947).

The almost exclusive incidence in women is of great importance, though not conclusive. It cannot be claimed that an incidence predominantly female is necessarily mediated by menorrhagic iron deficiency. This is, however, one of the agents important in making a disease predominantly female in incidence, and so far no other is recognisable in these cases.

It might be said that the iron deficiency is at least in part in many cases due to aspirin taking. This may be so. Such iron deficiency would have the same effect as that due to uterine haemorrhage. No doubt aspirin taking accounts for some cases of postmenopausal iron deficiency. It is probable too that aspirin as a tranquilliser and hypnotic, and perhaps also as an analgesic, is taken more by women than men. Still many men regularly take aspirin, and yet hour-glass stricture is extremely rare in men. Aspirin may cause gastric ulceration, acute and chronic, in both sexes, but again the stricture is rarely found except in women. Some factor above and beyond having an ulcer and taking aspirin must be responsible. It is not a high secretion of acid and enzymes by the gastric mucosa, because the mucosa in these cases is hypotrophic or atrophic. Indeed a gruel test meal shows

that some are hypochlorhydric or achlorhydric (cases 3, 4, 5 and 7). One cannot say whether the hypotrophic mucosa is a causative factor in the forming of the stricture, nor whether the hypotrophy could be caused by iron deficiency. These reports provide no information in that respect.

It might be said that the bleeding and anaemia and iron deficiency are only to be expected of a chronic gastric ulcer, and that the association of gastric ulcer and anaemia is therefore not remarkable, and the anaemia not necessarily due to uncompensated uterine loss of haemoglobin and iron. It would still be possible that the iron deficiency so produced made for chronicity of the ulcer, and also for the formation of the stricture. It is probable, however, that the incidence of bleeding in chronic gastric (and in chronic duodenal) ulcer has been over-estimated. It is now common ground that many of the cases of haematemesis and melaena admitted to medical wards have been occasioned by aspirin erosions of the gastric mucosa, *and that this may be so even if the patient has a co-existing chronic simple gastric (or duodenal) ulcer*. When chronic gastric ulcers bleed, they bleed severely but it is not often.

One must draw attention to the difficulty in studying the effects of iron deficiency on the formation of cervical oesophageal stricture and gastric stricture. These cases commonly come to notice when the stricture is well established, and sometimes years after it is well established. Six of the seven cases described were post-menopausal, though in case 4 the stricture seems to have formed after the meno-pause. Once the menopause occurs, the anaemia may in part or in whole correct. The serum iron may partly or wholly return to normal. Much depends on the aspirin-taking habits of the patient, and much on what treatment with iron she received. In examining postmenopausal patients it is hard to get satisfactory information about the iron state at the time the stricture was being formed. Once the stricture is formed, it never goes away, no matter how much the anaemia and iron deficiency are corrected. But in premenopausal patients adequately treated in the end, and in postmenopausal patients, there may be no, or only imperfect, evidence of the iron deficiency which caused the stricture.

When the stricture can be shown to have formed *after* the menopause, there can be no easy presumption that the patient was iron-deficient at the material time, though that may have been the case. Iron deficiency is not rare in postmenopausal women, sometimes because a premenopausal deficiency was never corrected, and somtimes because it was acquired after the menopause, e.g., by aspirin taking. Sometimes it cannot be explained. Nevertheless, the iron deficiency must be demonstrated, and there must be at least a reasonable presumption that it was present at the time the stricture was forming. In case 4 the stricture may have formed after the menopause but there is evidence of iron deficiency. In case 7 the stricture may have formed in old age or may not, but there is evidence of iron deficiency. It is clear enough that in cases 1 and 5 the stricture formed during the childbearing years. In cases 2, 3, 6 and 7 the stricture cannot be dated. The difficulty in dating the formation of the stricture lies partly in a lack of early barium examinations and partly in a lack of supine views.

Estimation of the serum iron is essential, because the serum iron may be low, even when the haemoglobin is within more or less normal limits. It is iron deficiency we are studying and not haemoglobin deficiency. If the mucosal atrophy

progresses, cyanocobalamin deficiency may be added, and this may give rise to a supposition that the stricture is associated with pernicious anaemia.

Fortunately in planning the treatment of chronic simple gastric ulcer it is not nesessary to decide the question of the relationship of iron deficiency to ulcer healing and stricture formation. General principles require that in gastric ulcer, as in most diseases, if iron deficiency exists, it should be corrected—promptly and completely. Estimation of serum iron as well as of the haemoglobin is essential in assessment of progress. The use of an intramuscular injection of iron is necessary at least in the beginning. Both the haemoglobin and the serum iron should be restored to normal, and kept there. Iron tablets lie under suspicion of causing gastric mucosal erosions as aspirins do, and they should not be used in disease of the gastric mucosa. After the injection of iron, treatment may be carried on by a bland liquid preparation of iron, but if there is difficulty one should return to the injection. Aspirin in all its forms and all salicylate preparations should be forbidden because of their corrosive action on the gastric mucous membrane, and their production of haemorrhage and iron deficiency. The treatment of chronic simple lesser curvature gastric ulcer and the prevention of its complications now seem to lie mainly in these two measures, and in maximal and optimal feeding (assuming always that there is no obstructive delay in gastric emptying also to be coped with).

It remains to be seen if this hypothesis of abnormal gastric scarring being due to iron deficiency has any general application to other visceral injuries which heal by scarring. For instance, observation of the serum iron and the haemoglobin in rheumatism ought to show whether rheumatic subendocardial scarring is worse in iron deficient individuals.

Summary

Seven cases of hour-glass deformity of the stomach are described in whom there was also evidence of iron deficiency. One case had also a duodenal stricture and three had strictures of the cervical oesophagus. It is suggested that in such cases a general tendency to stricturing exists, which acts locally to produce a stricture by an abnormal submucosal scar when, for whatever reason, a local ulcer is formed. It is suggested that iron deficiency may be responsible for the stricturing tendency, hindering normal healing of chronic simple lesser curvature gastric ulcer and inducing the formation of the gastric stricture. It is advocated that the treatment of chronic simple gastric ulcer of the lesser curvature (without any delay in gastric emptying) should rest mainly on correcting iron deficiency, on prohibiting aspirin and salicylates, and on feeding which, if bland, is both maximal in calories and optimal in composition. Where the mucosa is atrophic and the diet inadequate, the injection of cyanocobalamin should be added.

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FOUR CASES OF COUGH FRACTURE

By W. T. WARMINGTON, M.D.
Erne Hospital, Enniskillen

IN the past seven years four cases of cough fracture with eight fractures were found at Killadeas Hospital and the Chest Clinic, Erne Hospital, Enniskillen. No previous record of cough fracture of a rib in Northern Ireland is known to the writer. Yet any patient with a history of cough followed by pleuritic pain may be suffering from one. Other conditions predisposing to fracture may also be present.

Stress fracture is commoner than cough fracture. Unlike cough fracture it usually occurs in the first rib. As the name implies, the cause is muscular strain. Two cases with stress fracture of the first rib and one of the tenth were described in the Ulster Medical Journal ten years ago (Warmington, 1956). A typical example was encountered recently in a physical training instructress. "Fatigue fracture" of the first rib is merely a form of stress fracture affecting hitch-hikers and others who for long distances carry a heavy ruck-sack by a strap over the shoulder (Shanks and Kerley, 1962). Stress fracture of the first rib is often asymptomatic and is usually of little clinical importance.

Cough fracture is easily overlooked. The symptoms are those of the resulting pleurisy. Pleural effusion often follows. Pneumonitis may be mimiced or haemoptysis occur due to bruising or puncture of the lung by the rib ends (Shanks and Kerley, 1962). Pneumothorax or haemopneumothorax may distract attention from the underlying fracture. Also cough fracture usually affects the lower ribs in the axilla. Here rib curvature and tissue density are greatest on a postero-anterior X-ray and combine to obscure a fractured rib. For this reason Shanks and Kerley (1962) state that two oblique films are essential in every radiological investigation of rib fracture from whatever cause. Frequently separation of the fragments in cough fracture is so slight that a fracture is not seen until several weeks after symptoms have disappeared, when callus draws attention to it.

Case 1. G.K.; a male aged 54 on the date of his first cough fracture. He is a life-long bronchitic and asthmatic who for the past ten years has spent much of his time in bed with exacerbations and pneumonitis with uncontrollable cough. Although a tall, strongly built man, the ribs on X-ray are noticeably slender. The frequence of his pneumonitis is seen from the following: Dec. 1963 (right side), Jan. 1964 (left side), June 1964 (left side) and July 1964 (right middle lobe consolidated). The January 1964 pneumonitis was followed by a rib fracture on the same side, observed eight weeks later. In July 1965 a left sided pneumonitis was followed in a few days by a rib fracture on the same side.

His first observed cough fracture occurred in April 1959. It was of the 9th rib on the right side. In May 1963 callus was observed at the site of a presumed cough fracture of the 10th rib on the left side. Two further fractures were seen on the left side, namely of the sixth rib in March 1964 and the seventh rib in July 1965. An encysted pleural effusion overlay the latter fracture in which the rib ends were widely separated.

In November and December 1965 the carbon dioxide combining power was found to be elevated to 40 M.Eq. per litre (Normal 21-28 M.Eq. per litre). The blood urea (48 mgm. per 100 ml.) and the serum calcium (9.6 mg. per 100 ml.) were normal. The alkaline phosphatase was temporarily increased to 16 K.A. units, falling to 10 K.A. units a fortnight after. The plasma proteins showed an increase in gamma globulin to 2.0 gms. per 100 ml. The specific gravity of the urine reached 1020, with neither glycosuria nor proteinuria.

Case 2. R.F., a male aged 68. He was dyspnoeic on hills and slightly in bed also. A hard

cough appeared in February 1964, followed a month after by pain and tenderness over a rib below the angle of the left scapula. On 9th April 1964, X-ray showed a widely displaced (5 mms.) fracture of the 8th rib. No emphysema or costo-chondral calcification were visible on X-ray. The serum calcium was 10 mg. per 100 ml. on 25th July 1964, when he attended as an out-patient and the fracture was seen to be forming callus normally.

Case 3. I.C., aged 24, a married woman with two children born in July 1960 and September 1962. Neither child was breast fed. X-ray revealed slender bones, even for a woman. In February 1964 a cold was followed by severe cough, and this in turn by acute pain in the right chest, aggravated by breathing or coughing. No X-ray was taken until March 1964, after the pain had gone. This showed infiltration typical of pulmonary sarcoidosis, more marked on the right side, with fractures of the seventh and ninth ribs on the same side showing callus already present.

Case 4. T.A.McE., a dairy farmer aged 32. He became dyspnoeic in December 1965, worse following a "cold" on 18th January 1965, and accompanied by a cough. About 26th January he developed acute right-sided chest pain. A 100 mm. film on 27th January was suggestive of farmer's lung but did not show a rib fracture. On 9th February 1966 the clinical picture, serological investigation and history confirmed the presence of farmer's lung. A displaced fracture of the seventh rib on the right side, the side of maximal infiltration, was now clearly shown. He had lost 13 lbs. in weight since December 1965 and felt tired. No other cause predisposing to fracture was found. He stated that no impact or muscular strain preceded the fracture.

Discussion

Cough fracture has been found on both sides from the sixth to the tenth ribs inclusively. Pleuritic pain was present always and pleural effusion frequently, while haemoptysis followed cough fracture in Case 1. In two of the four cases fractures were multiple. In Case 1 four ribs were broken consecutively and in Case 2 two ribs simultaneously. In contrast, stress or fatique fracture commonly affects the first rib alone, when it is often without symptoms, and complications do not occur.

Homolateral pneumonitis occured before two consecutive fractures in Case 1. In cases 3 and 4 mavimal infiltration was on the same side as the fractures. Thus in 5 of 8 cough fractures homolateral pulmonary disease visible on X-ray preceded fracture.

Complications in these four cases included pleural effusion and haemoptysis. Pneumothorax, haemopneumothorax, tension cyst, lung abscess and areas of bruised lung resembling atelectasis are noted as complications by Shanks and Kerley (1962).

Cough fracture should direct attention to a possible predisposing cause, particularly if more than one rib is broken. Predisposing factors were most obvious in Cases 1 and 3 in which fractures were multiple.

In the present cases, predisposing factors included immobilisation in bed and emphysema (Cast 1), slender bones, sarcoidosis, debility and weight loss (Case 3). It is speculative whether respiratory alkalosis might have reduced ionisation of calcium and therefore its deposition in bone in Case 1. James (1965) found a malignant metastasis presenting as cough fracture, and had another case of cough fracture probably predisposed to by sternal depression. Shanks and Kerley (1962) note the occurence of cough fracture with Paget's osteitis deformans, multiple myeloma, osteomalacia, following heavy dosage of X-rays in radiotherapy for breast cancer and in deficiency of the organic bone matrix such as occurs in osteogenesis imperfecta. Cushing's syndrome and the prolonged use of corticosteroid

drugs predispose to fracture; likewise the osteodystrophies of renal origin (milk alkali syndrome and hyperparathyroidism). Either cough or spontaneous fractures following radiotherapy may be multiple, occur in the axis of the therapeutic beam, and usually fail to unite (Shanks and Kerley, 1962). Hence cough fracture and its predisposing causes should be considered in cases with unexplained pleuritic pain.

Summary

In cough fracture the lower ribs are most commonly affected, particularly the sixth to the tenth inclusively. Pleuritic pain is the outstanding symptom. Cough fracture has recently been found on the side of maximal pulmonary infiltration when this is present, whether the infiltration is due to such different causes as pneumonitis, farmers' lung, or sarcoidosis. When more than one rib is broken, whether simultaneously or after a long interval, a cause predisposing to fracture is often present. Some predisposing causes and complications of cough fracture are listed. Both are often serious. Hence cough fracture, though easily overlooked, should be carefully sought, particularly when examining the chest film of any patient with cough and unexplained pleuritic pain.

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BOOK REVIEW

CUNNINGHAM'S MANUAL OF PRACTICAL ANATOMY. Revised by G. J. Romanes, B.A., Ph.D., M.B., Ch.B., F.R.C.S.Ed., F.R.S.E. Thirteenth Edition. Vol. 1. Upper and lower limbs. (Pp. viii+256; figs. 175. Paper 25s.; boards 35s.). London: Oxford University Press, 1966.

THIS well known dissecting manual has been thoroughly revised and recast to bring it into line with the present day requirements of the medical student. The text has been largely rewritten using the *Nomina Anatomica* (1961) throughout and it is now represented in two columns but on a slightly larger page than formerly. All the illustrations have been redrawn and, where possible, simplified by the removal of many of the leaders which tended to confuse the older diagrams. New drawings have been added to illustrate the functional aspect of various structures but some of the radiographic illustrations have been omitted as most departments now have available ample teaching material of this type. The general impression gained from handling this new edition is extremely favourable. The medical student using it will find his work has been made easier as a result of the changes which sacrifice none of the essentials of a first rate guide to dissection. It will be welcome everywhere as a worthy successor to the long line of previous editions and it can be thoroughly recommended as suitable for use by students. W.R.M.M.

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FOETAL HEART RATE MONITORING — PRESENT LESSONS AND FUTURE DEVELOPMENTS

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JUST as the first detailed description of the foetal heart sounds (Le Jumeau de Kergaradec, 1822) eventually led to the view that foetal bradycardia might indicate danger to the foetus (Von Winckel, 1903) and to the present routine practice of periodic auscultation during labour, so the first reported foetal electrocardiogram (Cremer, 1906) has been followed by efforts to develop automatic methods of monitoring the foetal heart (Hon, 1960). The shortcomings of intermittent auscultation as a means of detecting foetal distress or of predicting the condition of the baby at and after delivery prompt the following questions:

Is electronic monitoring any better?

Is it now ready for routine use in our labour wards

Has research in this field provided any lessons which can be applied now to routine practice?

How are future advances likely to help clinical management?

In attempting to make some answer to these questions, the writer draws heavily on recent experience as a member of Dr. Edward H. Hon's research group in Los Angeles and at Yale University. In addition to obtaining practical training in Hon's methods, his unrivalled library of tracings from more than 1,000 fully monitored labours was studied in detail.

The Potential Value of Electronic Monitoring

In contrast to the limitations of intermittent observation and the relative inaccuracy of human counting, the input of continuously recorded electrocardiograms to an accurate cardiotachometer provides a means of obtaining an uninterrupted trace of the exact instantaneous heart rate. With the addition of some technique for cancelling the maternal electrocardiograms, a tachometer will sometimes even count with fair accuracy suitably amplified and filtered foetal signals recorded indirectly from the maternal abdomen provided the patient and her uterus are at rest. To produce a continuous tracing during labour, however, the tachometer must receive the electrocardiograms directly from the foetus so that they are not only free from the unwanted maternal complexes but have a high enough signal-to-noise ratio to remain countable when the patient moves or the uterus contracts. The direct electrocardiogram is obtained with a clip electrode (Hon, 1963). This can usually be applied when the cervix has reached 3 cm. dilatation and with the membranes ruptured. A fine catheter, introduced at the same time, permits simultaneous recording of the intrauterine pressure. In this way, foetal heart rate changes can be related to the frequency, strength and duration of uterine contractions. The identification of the pattern of this relationship between heart rate and uterine action—particularly the recognition of a changing pattern—is the key to interpreting foetal heart rate changes in labour. Because a simultaneous tracing of the heart rate and the intrauterine pressure shows this relationship immediately, clearly and continuously, the answer to the first question is

an affirmative one. Electronic monitoring is potentially much more valuable than intermittent auscultation.

The Feasibility of Routine Monitoring

Unfortunately, the second question must be answered in the negative. No equipment, as yet available commercially, meets the requirements of a practical system for clinical monitoring, namely reliability without prohibitive cost, simplicity of operation and of maintenance, an input from electrodes placed on the mother's abdomen without discomfort or inconvenience to her, and an output in the form of an accurate uninterrupted trace throughout even prolonged and strong labour. A number of electrocardiographs are available for recording foetal signals indirectly via the maternal abdomen but, as their output is unsuitable for automatic counting, they are of little clinical value apart from affording occasional help in the confirmation of foetal life or the early diagnosis of multiple pregnancy. Because, with a normal heart rate and at a standard recording speed, 10 hours of labour produce almost 100,000 foetal electrocardiograms on over half-a-mile of paper, manual plotting of the rate is not feasible even if the signals can be identified during contractions.

The foetal phonocardiogram is even less suited to accurate automatic counting. A heart beat is not represented by a single sharp sound of constant timing. Instead, the tachometer may be triggered at any time or several times during one or both sounds. The elimination of multiple triggering may be attempted by reducing the sensitivity of the tachometer or by making it unresponsive for a short pause after counting an impulse, but the inherent variability in amplitude of the two sounds will result in some beats being counted at the first sound and some at the second, while others (with both sounds of low amplitude) go undetected. Some comercially available foetal phonocardiographs include a device for recognising and rejecting the crescendo of muscular vibration which obscures the foetal heart sounds when the patient moves or the uterus contracts. A system incorporating such a device will hold its last true count until the muscular noise has ended with the result that, at the very time when the heart rate is most likely to alter and when its pattern is usually diagnostic, the tachometer output may be an erroneous and perhaps dangerously misleading straight line.

Hon's data have been acquired, stored and processed by complex equipment which needs constant development to meet research requirements, carefuly supervision in operation and highly skilled technical maintenance. Elaboration of such equipment continues but its equally important development in the opposite direction, i.e., towards simplification, is proving more difficult. It need not be expected to give birth to an effective clinical monitor in the near future. Nevertheless, research studies with sophisticated instrumentation have provided the data for a "working hypothesis" for foetal distress (Hon, 1962), from which a number of clinically applicable lessons can be drawn.

The Normal Foetal Heart Rate

The normal foetal heart rate, at or near term, is between 120 and 160 beats per minute. Its baseline is not smooth but shows 3 to 5 clinically undetectable fluctuations each minute. The rate represents the balance between vagal and sympathetic tone, while the fluctuations seem to reflect an inherent variability in normal

vagal tone. Gradual development of vagal tone as pregnancy advances is suggested by the progressive slowing of the rate and the increased baseline fluctuation towards and after term.

FOETAL TACHYCARDIA *(i.e. over 180 beats per minute)*

The very premature foetus has a smooth tachycardia which is also seen when the vagus is blocked by atropine. A similar trace may occur with maternal pyrexia, presumably due to vagal release or sympathetic stimulation. It may occur transiently before or after hypoxic bradycardia, suggesting that the foetal response to developing or resolving hypoxia is an adaptive tachycardia, probably brought about by increased sympathetic tone. Transient tachycardia after vagal bradycardia may reflect momentary sympathetic overdrive before normal autonomic balance is restored. Hon and Lee (1963) have suggested that the smooth sustained tachycardia which sometimes follows prolonged bradycardia and precedes foetal death may be due to an unavailing final release of adrenaline in the dying foetus.

FOETAL BRADYCARDIA *(i.e. less than 120 beats per minute)*

Hon (1962) now recognises four bradycardia patterns. Three are related to uterine contractions, the first beginning early and being due to head compression (HC), the second having a variable onset and being due to cord compression (CC), the third occurring late in the contraction phase as a result of utero-placental insufficiency (UPI). A sporadic pattern unrelated to contractions should be regarded as a variant of the CC pattern with which it is associated.

Head Compression (HC) Bradycardia. HC patterns, illustrated diagrammatically in Fig. I, are in-phase with the contractions, reflecting their intensity, duration and "shape". They begin soon after each contraction starts and recover by the end of the contraction. Their early onset points to a reflex origin, and their abolition by

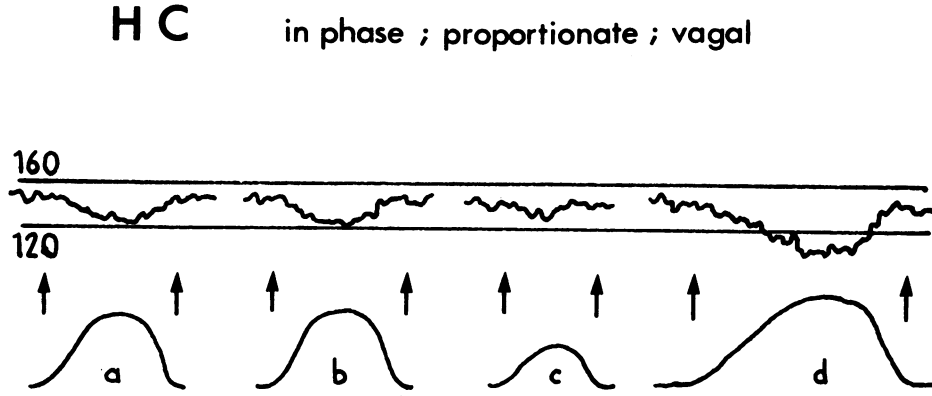

FIG. 1. *Head compression (HC)bradycarda. As in the succeeding diagrams, simultaneous traces of foetal heart rate (above) and intrauterine pressure (below) are depicted. The horizontal lines indicate the normal limits of the foetal heart rate (160 and 120 beats per minute). The paired arrows mark the onset and end of uterine contractions. Note the onset of HC bradycardia soon after a contraction starts and its recovery by the end of the contraction, the small deceleration with a weak contraction (marked c) and the increased pattern during an unusually strong one (marked d).*

atropine suggests the reflex is a vagal one. HS patterns occur in only a few cases and are seen when the cervix reaches about 7 cm. with strong contractions. Their production by equivalent pressure on the fontanelles, but not by manual compression of the head, suggests that the reflex resembles the oculo-cardiac reflex induced by eyeball pressure and that it may be initiated by pressure of the cervical rim on the fontanelles. These patterns are to be regarded as physiological and do not reflect any foetal hazard.

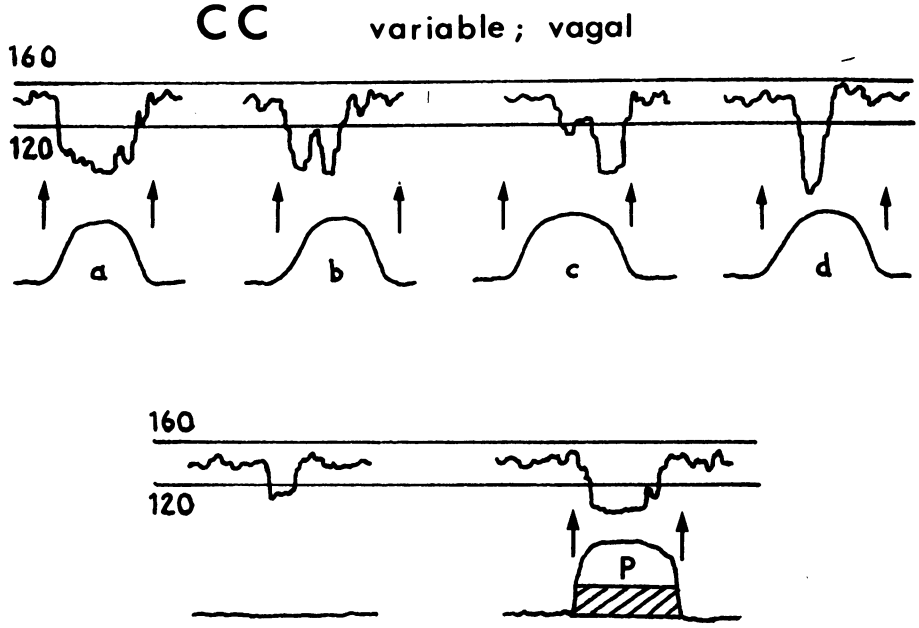

FIG. 2. *Cord compression (CC) bradycardia. Note the variable onset—at the start of some contractions (marked a and b), later during others (marked c and d). Note that the bradycardia recovers by the end of the contraction, although occasional sporadic patterns occur when the uterus is relaxed (bottom left). Typical patterns may be induced by abdominal palpation or pressure (P, bottom right). Note the variability in depth, duration and shape of the patterns. Their steep sides and the increased baseline irregularity are in keeping with a vagal reflex mechanism.*

Cord Compression (CC) Bradycardia. Most labours show at least isolated examples of CC bradycardia (see Fig. 2). Variability is its essential feature—a variability in depth and duration unrelated to strength anl length of the contractions; a variability in rate which may cause clinically detectable baseline irregularity and even beat-to-beat arrhythmia; a variability in the "shape" of the pattern although this is usually steep-sided due to very rapid development and recovery suggesting marked reflex activity. Abolition by atropine again suggests a vagal reflex. CC patterns are often associated with cord entanglement around the foetal neck, trunk or limbs, especially when the cord is also taut and as the head descends or rotates. They may be induced by palpation over a particular part of the uterus (presumably due to an underlying cord), during external cephalic version (pre-

sumably due to torsion or entanglement of the cord), or when the head is manoeuvred into the pelvis (presumably due to similar entanglement or to a loop of cord between head and pelvis). Typical patterns are readily induced by manipulation of the cord at caesarean section, in cases of cord prolapse and in animal experiments. On the other hand, they are not unusually frequent when there is probable placental dysfunction, are not induced when the mother breathes an oxygen-poor mixture and are not modified by increased oxygenation.

In CC patterns, the vagal centre may be triggered by sudden anoxia to it as a result of the abrupt hypotension due to cessation of blood flow from the placenta which must follow short-lived occlusion of the umbilical vein. The purpose of this reflex may be to limit cardiac work during such stress. Thus, some slowing and irregularity during contractions probably indicate an intact rather than failing protective mechanism. The passage of meconium, which sometimes accompanies these patterns, probably also results from vagotonia. Anal relaxation and peristalsis may well result more often from vagal stimulation than from tissue hypoxia. Increasing vagotonia with continuing pregnancy may explain some of those cases of meconium staining past term for which cesarean section is so often carried out in the presumed interests of the foetus. Similarly, meconium which has already reached the anal sphincter as the result of repeated unimportant episodes of ante-

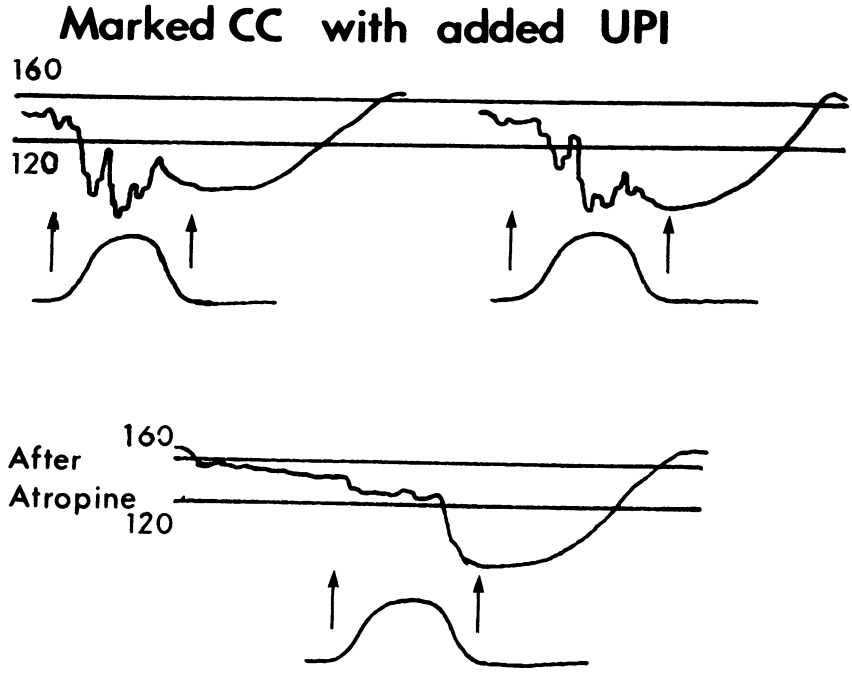

FIG. 3. *Marked CC bradycardia with superimposed utero-placental insufficiency (UPI) patterns. CC patterns (above) are followed by the smooth delayed bradycardia of UPI so that a normal rate is not regained until some time after the contractions end. Atropine (below) has almost completely abolished the vagal CC bradycardia but has not affected the UPI element.*

partum cord compression will be readily passed in labour should even minimal further compression of the cord occur.

CC patterns are not reflected in increased mortality, morbidity or low Apgar scores unless they become persistent and severe, in which case they are then usually accompanied by the late bradycardia of utero-placental insufficiency (see Fig. 3). The superimposed UPI pattern indicates that a potentially dangerous tissue hypoxia has now developed as a probabl result of more prolonged or severe cord occlusion. Atropine will still modify the CC pattern but will not affect the added UPI bradycardia. In profound CC bradycardia beat-to-beat arrhythmia may be pronounced and heart block may occur. This amounts to foetal vasovagal syncope.

Utero-placental Insufficiency (UPI) Bradycardia. UPI bradycardia is less common but more important than CC bradycardia. When these patterns coexist, management should be dictated by the UPI element. Although delayed, UPI patterns (see Fig. 4) are obviously related to the strength, length, "shape" and frequency of the contractions, as is well shown when the uterus is overdriven with intravenous oxytocin. The smooth trace unaffected by atropine excludes vagotonia. Instead, the delayed onset and recovery suggest the gradual development and then the slow correction

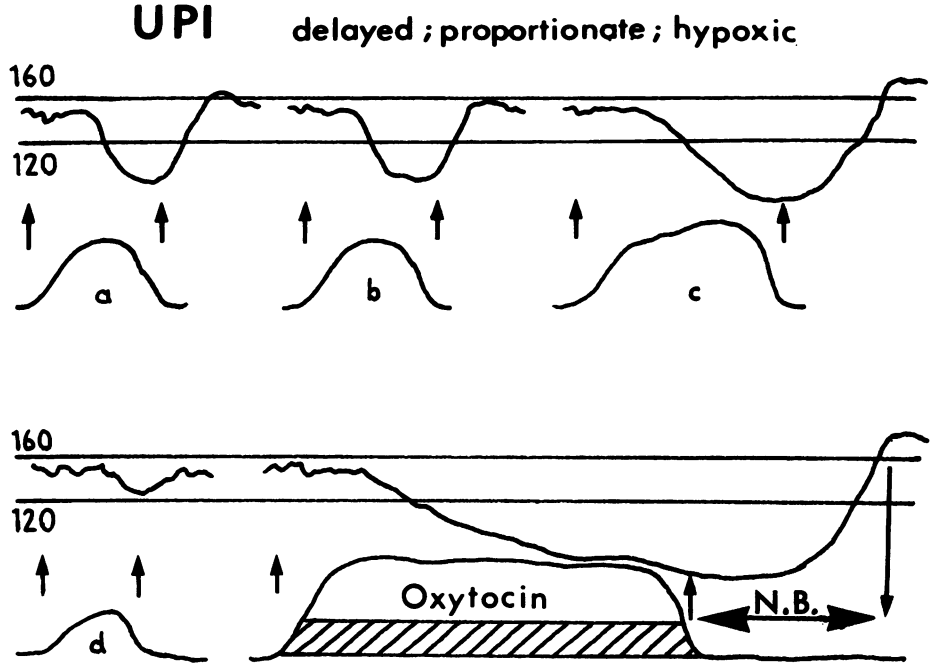

FIG. 4. *Utero-placental insufficiency (UPI) bradycardia. Note the delay in onset after the contractions start and the delay in recovery after they end. The smooth baseline is quite unlike the fluctuation associated with vagal activity. The depth and duration of the patterns reflect the strength and length of the contractions (note especially those marked c and d). Oxytocin-induced uterine tetany results in a particularly severe and prolonged bradycardia with an increased delay in recovery after the uterus relaxes.*

of tissue hypoxia affecting the myocardium directly, an origin further supported by the association of these patterns with conditions interfering with utero-placental blood flow, e.g., uterine tetany and hypertonus, pre-eclampsia, essential hypertension and diabetes. This type of bradycardia is associated with evidence of foetal acidosis in cord blood samples (Quilligan *et al.*, 1965). There is also some experimental evidence, albeit less convincing, that UPI patterns can be reproduced by maternal exercise (Hon and Wohlgemuth, 1961) and alleviated by breathing 100 per cent oxygen (Hon, 1962).

These patterns often increase as labour advances, in which case there may be meconium staining, difficulties in resuscitation and perhaps stillbirth. Tracings preceding foetal death reveal a progressive increase in the patterns, usually over several hours, which should make possible the early recognition of potential danger by automatic monitoring or even by careful repeated auscultation towards and at the end of the contractions. If the bradycardia has been allowed to progress until it persists from contraction to contraction the foetal condition is already critical. If it has given way to a sustained tachycardia foetal death is probably inevitable.

Present Clinical Applications

It is suggested that the lessons to be learned—in some cases to be relearned—are as follows. In the absence of obvious bradycardia the presence of one or more of the triad comprising transient tachycardia, irregularity with contractions and the passage of meconium probably shows that the foetus is adapting successfully to an, as yet, unimportant stress. These signs may however be the forerunners of a bradycardia, the significance of which depends on its pattern in relation to the uterine contractions. HC patterns are infrequent and unimportant. The recognition of CC patterns is important because some will become constant and increasingly obvious, suggesting that the protective vagal reflex may then be reaching its maximum limits. Superimposed UPI patterns, or UPI patterns developing on their own, suggest that foetal adaptation to hypoxia is no longer adequate. These lessons are applicable to current practice provided that auscultation is carried out during and immediately after contractions rather than after waiting for the uterus to relax first. A rough assessment can be made of the interval from the end of a contraction to the correction of the bradycardia, and repeated listening in this manner should reveal any dangerous overall trend towards an increase in this interval.

Fortunately, suitable simpler equipment should soon become available for the less sophisticated but more important further study of the intrapartum foetal heart rate patterns, particularly in conditions associated with placental insufficiency. Although the need for direct recording will preclude its general employment, such equipment could be used in selected high-risk cases, such as pre-eclamptics and diabetics, or when a clinical diagnosis of probable foetal distress has been made. Worthwhile studies could include the correlation of fetal heart changes with biochemical investigations on foetal blood obtained by Saling's method (Saling, 1964) and with foetal anaemia in cases of rhesus sensitisation. The system could also be used to investigate the possibility of significant utero-placental insufficiency during conduction anaesthesia and the effect of hypotensive therapy on the utero-placental insufficiency of pre-eclampsia and essential hypertension.

Future Developments

What of the future? The present concentration by some American groups on the computation of increasing data in increasing detail may well yield little information of immediate practical use because analysis does not amount to interpretation.

The stimulation by an intravenous oxytocic infusion of contractions equivalent to those of strong labour and the assessment of any resultant bradycardia could become the basis of a "trial of placenta" when delivery is indicated or labour has started in the presence of suspected placental impairment.

Another practical proposition would be to explore the feasibility and usefulness of combining the ability of the human brain to "filter out" unwanted noise with a manual device for triggering a tachometer as each heart beat is auscultated. Substitution of the ear and brain for electronic amplification and filtration would eliminate the most critical and expensive components. If the best of both worlds can be achieved in this way, it ought to be possible to produce a relatively cheap and simple system comprising the trigger, a simple tachometer and a single-channel recorder with an event marker to indicate the uterine contractions. The bradycardia of UPI would be easily recognised in the tracing, the delay in its recovery after a contraction could be measured, and periodic comparisons would reveal an increase in this measurement long before it could be appreciated by the usual method of unaided auscultation.

Summary

Attention is drawn to the potential value of automatic foetal heart monitoring during labour.

The unsuitability of present research equipment and techniques for routine clinical use is discussed.

Normal and abnormal intrapartum foetal heart rate patterns are described, and their underlying mechanisms considered briefly.

The significance of the abnormal patterns is indicated and the importance of relating foetal bradycardia to the uterine contractions is stressed.

A number of clinically applicable lessons are drawn.

Some possible useful future developments, including proposed techniques for testing placental sufficiency during uterine contractions and for accurate plotting of the rate of auscultated foetal heart sounds, are outlined.

Acknowledgments

I wish to thank The Queen's University of Belfast for leave of absence and a grant in aid to take up a United States Public Health Service research fellowship (No. 8 TI-HD; 34-03). My thanks are also due to Dr. Edward H. Hon of Yale University, who introduced me to this field and made his data available to me, and to Dr. Stanley T. Lee, formerly of Loma Linda University, California, who trained me in the monitoring techniques.

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ACCIDENTS IN CHILDHOOD

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"The chapter of accidents is the longest chapter in the book"
Attr. to John Wilkes by Southey in "The Doctor" (1837) IV, 166

DURING the last 100 years there has been a remarkable fall in child mortality rates. Before 1860, 4 per cent of children who survived their first year died before attaining the age of fifteen, while in the slums the figure was as high as 20 per cent (Office of Health Economics, 1962). The term "The Survival of the Fittest" was more than an aspect of Darwin's new theory of Evolution: it was a hard fact of life.

The reasons for the improvement in the picture are not difficult to find. Improved Public Health services and possibly superior resistance to infectious diseases transmitted by surviving parents probably account for the earlier, slow but steady fall in the child mortality rate; the rapid increase in tempo since the 1930's is undoubtedly mainly the result of the development of effective antibacterial agents and vaccines. The control of the five infectious diseases—pneumonia, tuberculosis, diphtheria, measles and whooping cough—and their complications accounts for over half the reduction in mortality since 1931 (Office of Health Economics, 1962); only pneumonia remains as an important cause of death to-day (Fig 1).

Deaths attributed to accidents in childhood have also decreased in number during this period, including, surprisingly enough, a 50 per cent fall in the number of deaths from road accidents, but this decline has been much less marked than the fall in deaths from infectious disease, so that accidents have assumed greater relative importance and now account for nearly 30 per cent of all childhood mortality. Indeed, a Lancet annotation in 1961 declared that accidents were the largest single cause of death between the ages of 1 and 9 years in the United Kingdom, while in some countries more persons between 5 and 19 years of age die from accidents than from all other causes combined.

It is convenient to consider accidents in two categories—those inside and those outside the home.

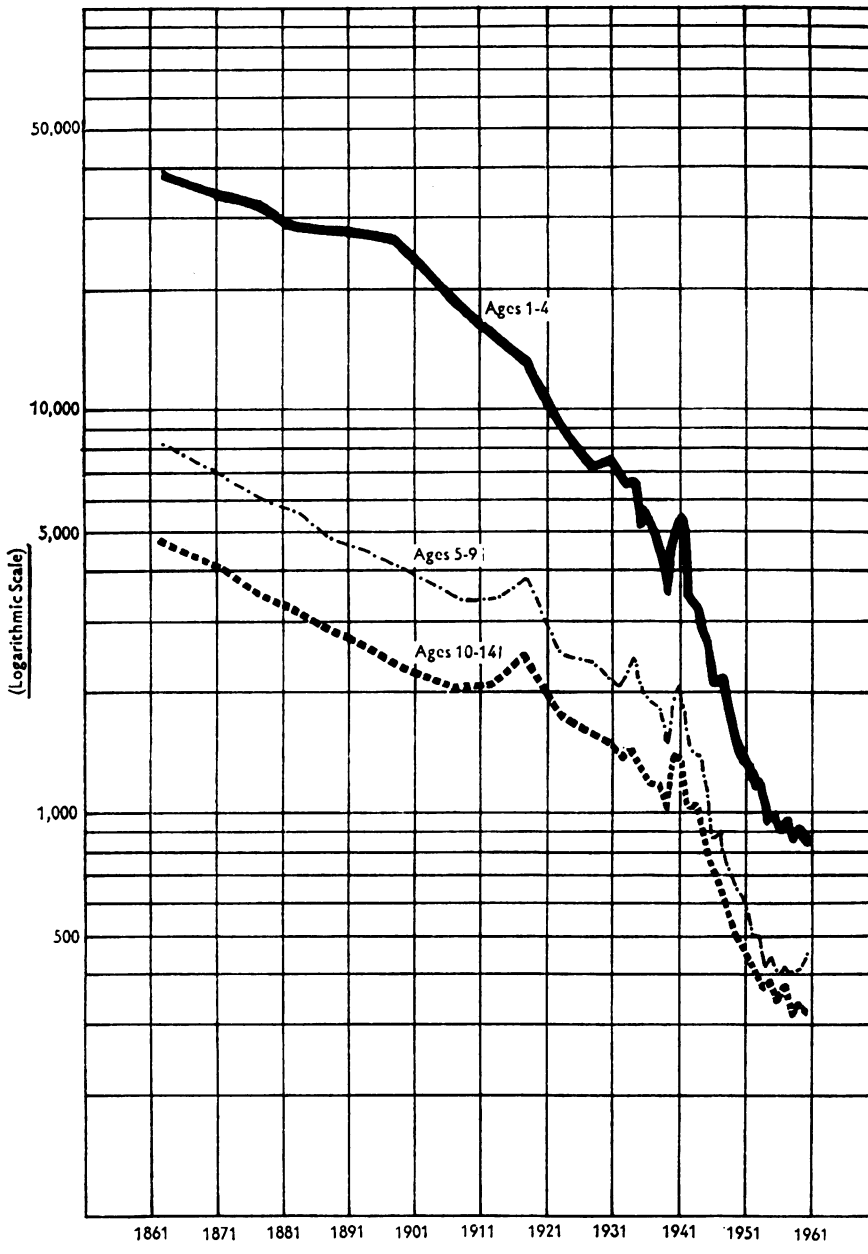
1. Accidents in the Home

A leading article in the British Medical Journal in 1964 revealed that during the previous 10 years 80,000 deaths from home accidents had occurred, 10,000 of them in children under the age of 15 years. Burns, scalds and poisoning figured prominently in this group.

The majority of home accidents are, of course, non-fatal, although they may leave children seriously disabled or disfigured. MacQueen (1960) found in his survey in Aberdeen in 1955-57 that more than half the home accidents occurred in pre-school and school-age children who represent less than a quarter of the population. Moreover, 80 per cent of scalds occur in children under 4 years of age (Tempest, 1956). Surveys of this type are of limited value because the notification of home

Child Death Rates per Million; England and Wales 1861-1960.

Source: Registrar General Statistical Review Part One (1960).



Note: Quinary Averages 1861 to 1930: annual rates 1931 to 1961.

FIG. 1. *Taken from "The Lives of Our Children : A Study in Childhood Mortality." Office of Health Economics* (1962). *(Used by permission)*

accidents is necessarily incomplete, as many trivial injuries are not reported.

The survey conducted between 1947 and 1952 among families in Newcastle gave an incidence of 732 accidents in 847 children during the first five years of life, almost one per child. The definition of a "significant accident" which was adopted included "all injuries sufficiently severe to cause more than a transient upset or for which medical advice was sought; we have also included all substantial falls downstairs, all burns and scalds, and the swallowing of poisons or foreign bodies" (Miller, Court, Walton and Knox, 1960). The authors cite the definition of Clements (1955) which includes accidents which were serious or "could have been serious in slightly different circumstances". This Newcastle survey was conducted prospectively and provided useful information but the numbers involved were relatively small. The greatest number of accidents occurred during the second year of life; during the first three years most accidents occurred inside the home, but during the fourth and fifth years most occurred elsewhere.

In Northern Ireland during the five years 1959-63, 3,211 children under 15 were admitted to hospital, following accidents in the home, and thousands more received outpatient attention or were seen at home by their family doctors. During the six years 1958-63, 135 children died from home accidents (Registrar General, 1958-1963) (Fig. 2.).

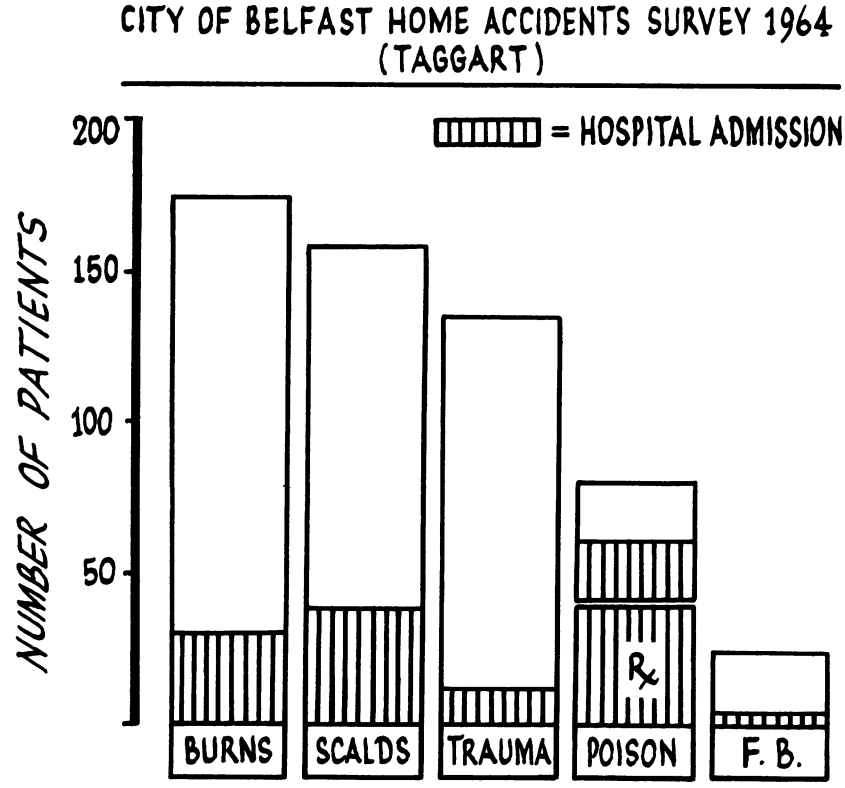


FIG. 2

Suffocation is said to have been the cause in 52 of these (39 per cent). This diagnosis, however, is very much open to question for many of these children, the great majority of whom were under one year of age, are likely to have been examples of "cot death". This condition which is one of the most puzzling problems confronting pathologists is currently being investigated in the Province. Suffocation is a rare cause of death in children, although occasional instances of accidental strangling or true suffocation do occur. Much more commonly the history obtained shows that the young infant was put to bed in normal health and was found dead several hours later. Autopsy reveals no cause of death. Unfortunately, the diagnosis of suffocation (by soft pillows or bedclothes) is still frequently recorded in this situation, resulting in a completely unjustified implication that the parents have contributed to the child's death by their own carelessness Carré, 1964). To spare parents needless mental anguish, and in the interests of accurate record-keeping, the concept of suffocation in the circumstances described should be abandoned. The difficulty of finding an alternative diagnosis remains, although this syndrome of "Sudden, Unexplained Death in Infancy" has been sufficiently well documented to be acceptable without the necessity of further amplification if autopsy reveals no definite findings. About 60-70 instances of the

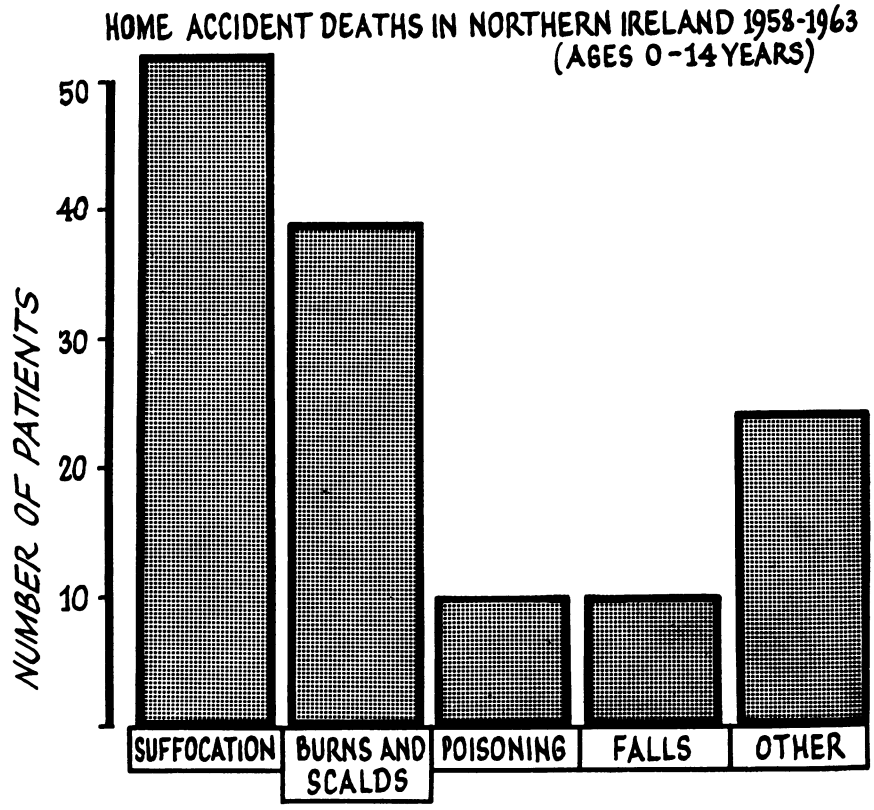


FIG. 3

syndrome occur yearly in Northern Ireland, which is an incidence of about 2/1,000 live births (Carré, 1964). This is of the same order as the incidence quoted by American authors (Bergman and Beckwith, 1965).

If we therefore exclude suffocation, burns or scalds accounted for 47 per cent of the remainder, while poisoning and falls each accounted for 10 deaths, or 12 per cent.

A recent survey conducted in Belfast indicates the kinds of accident which occur most frequently (Taggart, 1964) (Fig. 3). The information was derived from notifications of children seen in the outpatient and casualty departments of the Royal Belfast Hospital for Sick Children and the Ulster Hospital following a domestic accident. It includes the more severe accidents which may take a child to hospital but obviously takes no account of minor accidents which may be treated at home. It is noticeable that although poisoning is fourth in numerical incidence (13 per cent of the total), it accounted for 43 per cent of hospital admissions. More than half of these children had taken some kind of medicine or tablets. One child with lead poisoning died.

2. Accidents Outside the Home

The majority of these are road accidents, although some children are killed or injured in falls and others are drowned. During the years 1959-63, 50 children were drowned in Northern Ireland, and 15 died from falls (although 8 of the latter occurred at home) but 143 were killed on the roads (Registrar General, 1959-63).

Road Accidents

In 1964 there were 6,584 casualties on the roads of Northern Ireland, of whom 219 died (Royal Ulster Constabulary, 1964). This is an incidence of one person in 221 of the population, which is more than half the incidence in Great Britain despite the fact that in Northern Ireland there are little over one-third as many vehicles and slightly less than half the number of persons per mile of road. Thus, road congestion is not the only factor responsible for accidents, nor is it perhaps the most important.

Of the Northern Ireland casualties, about one-third (1,275) were children, but of all injured pedestrians 54 per cent were children. Twenty-eight children died, which represents 29 per cent of the deaths among pedestrians and 17.3 per cent of the total road accident deaths. Just over half the children killed and injured (52 per cent) were aged between 3 and 8 years. In their assessment of accidents, the Royal Ulster Constabulary considered that children under the age of 10 years figured prominently among those persons mainly responsible. In fact, children during their fourth and fifth years were thought to have caused more accidents than any other road users at any one age. Motorists in their early twenties formed the next largest group. After the age of 8 years there is a conspicuous fall in the number of children involved in accidents and in the number of accidents attributed to them (Royal Ulster Constabulary, 1964).

Although the worst months for road accidents in general were August and December, the months of June to September were almost uniformly bad where children are considered alone.

3. Developmental Factors in Accidents

The type of accident in which a child is involved depends to some extent upon his age and developmental maturity.

(1) *Infancy*

Accidents involving infants are usually attributable to carelessness by adults. The child may be given wrong food, or wrong medicine, or the wrong dose. He may be given a dangerous object to play with, or a toy which is unsafe, such as one with eyes which may be removed and swallowed, or with sharp internal parts which are easily exposed. There are few legal controls over toy manufacture, although the British Standards Institute has a code of Safety Requirements which is accepted by the British Toy Manufacturers' Association. The infant may fall from a table while he is being changed, or from a cot whose side has been left down. He may fall from a high chair, unless he is properly strapped in. Outside the home his chief danger is that of falling from a pram.

(2) *The Crawling and Toddling Stages*

During the latter part of the first year the child becomes mobile and increasingly curious. Important sources of danger are open and electric fires, oil stoves, electric sockets and cords, gas taps, matches, hot water taps, cookers with saucepan handles projecting outwards, tablecloths which may be pulled, unguarded staircases, plastic bags, garden pools, and bottles containing cleaning fluids, paraffin turpentine, insecticides or bleach. These latter substances are frequently kept under the kitchen sink, often in old lemonade bottles. At this age the child may choke on nuts or raw vegetables which are easily aspirated into the bronchi.

(3) *The Pre-School Stage*

Between the ages of 2 and 5 years the most frequent home accidents are falls, cuts and lacerations, scalds, burns and poisoning. The child is able to climb on chairs or other objects to investigate high cupboards, and drugs need to be kept under lock and key. This is the stage at which most road accidents occur, usually resulting from the child running carelessly into the path of traffic.

(4) *The School Child*

Road accidents involving pedestrians are still important in this group, particularly during the first few years. Later, the cyclist begins to figure in the lists of the killed and injured. Falls still occur, and drowning becomes more frequent at this stage. Most accidents now occur outside the Home.

4. Children at Risk

"Accidents will occur in the best regulated families"
(Mr. Micawber—Charles Dickens, 'David Copperfield')

Some children are thought to be more liable to serious accidents than others, although all must be at risk at times. Those from poor home conditions appear to be more liable to accidents within the home (Rowntree, 1950; Seiler and Ramsay, 1954).

On the other hand, many workers have suggested that accident proneness in the child is an important factor in many cases. In an investigation by Craig (1955) of

children who had been poisoned, there was a high percentage with exaggerated oral tendencies. Many of these were in the habit of eating small pieces of dirt, or tablets, or small objects, and in 50 per cent he considered that the degree of this habit amounted to pica. These children swallowed things quickly, whereas others were more selective and usually tasted, then spat out the poisonous substances. As he said: "The gourmet lives, the glutton dies".

Burton (1965) has studied a group of children injured in road accidents in Belfast. She found that compared with control children they were impulsive, preoccupied and in conflict with their parents and environment. They tended to be restless and hyperactive from early infancy. There was often a history of a difficult pregnancy or of a difficult birth. There was evidence of considerable emotional distress prior to the accident, yet none of these children had been regarded as abnormal. Burton concluded that attention should be directed to the detection of such stressed children at school and that they should be offered emotional support. Prechtl (1962) has shown that behaviour disorders may be the only residual sign of perinatal damage, while minor varieties of cerebral palsy may be unrecognised unless specially looked for. It would be interesting to investigate whether children who suffer perinatal anoxia are subsequently accident-prone by comparison with controls.

Langford and his co-workers (1953) found that there was a more distant relationship between the parents of accident-prone children and their offspring than that between parents and control children, and that the parents of the accident children were remarkably unconcerned about the injury. A similar distant family relationship was observed by Sobel and Margolis (1965) in a study of children involved in repetitive poisoning.

Haggerty and his co-workers (1962) in Boston, Massachusetts, conducted a survey among children admitted to hospital after accidents and found that 75 per cent of the children were hungry or tired at the time. Slightly more than half the mother were ill, pregnant or menstruating when the accident occurred, and in 54 per cent the children had been in the care of an unfamiliar person. Illness in other members of the family, demanding much of the mother's attention was present in 35 per cent, and 44 per cent of parents had an unstable marital relationship or were separated. The hazard, such as a busy street, a knife or a bottle of aspirin, was easily accessible in 63 per cent, and in 39 per cent the hazard was attractive, such as "candy aspirin". Unfortunately many tablets are made up in an attractive colour and may look very similar to certain types of children's sweets. An extreme example was seen in an American hospital shop, where sweets were on sale in the guise of capsules, packed in imitation medicine bottles with appropriate labels. It would be difficult to explain to a child who had been given these sweets that medicinal preparations are for sickness only and potentially dangerous.

5. Prevention of Accidents

"Prevention is better than cure" (Proverb)

The Lancet suggested in 1961 that the problem of home accidents should be approached by investigating their epidemiology, enacting legislation and applying educational methods. These approaches have also been used in dealing with the increasing road accident problem.

(1) *Epidemiology*

Patterns of incidence of both home and road accidents have been widely studied. Home accident notification is always incomplete, but we already know a great deal about causes and types of accidents, and the data available may be made a basis for action in the spheres of legislation and education.

(2) *Legislation*

It is obviously important to legislate against dangerous equipment in the home, and to provide a system of laws to regulate use of the roads, but the effectiveness of legislation is necessarily limited by the ingenuity of those it tries to protect, and by changing circumstances. Constant modification of our laws is necessary. One example will illustrate this statement.

Between 1958 and 1962, 152 children in England and Wales between the ages of 5 and 14 years died from burns sustained when their clothing caught fire. Many more were severely burned but survived. On March 3rd 1964, Mrs. Patricia McLaughlin, M.P. for Belfast West, introduced a Bill at Westminster "to prohibit the sale and manufacture of flammable materials for certain purposes". Her intention was to prevent readily flammable cloth from being used for the manufacture of pyjamas and party dresses, as well as for nightdresses for children. The Bill was not passed. In September 1964, however, the Home Secretary, Mr. Henry Brooke, introduced a ruling banning the sale of inflammable children's *nightdresses* but not of inflammable pyjamas or cloth. On December 22nd 1964 an 8-year-old boy was admitted to the Royal Belfast Hospital for Sick Children with very severe burns sustained when his pyjamas caught alight while he was warming himself by an open fire before going to bed. He died on Christmas Eve. At the inquest in January 1965 his mother said that she could not buy non-inflammable boys' pyjamas in Belfast. The Coroner's officer telephoned three leading stores and confirmed that although they had non-inflammable girl's pyjamas and nightdresses in stock, they had none for boys (Belfast Telegraph, 14th January 1965).

Undoubtedly, such loopholes in our laws need to be removed, but the human element in the genesis of home accidents will remain. It is necessary to instruct and warn parents, schoolteachers, and others concerned with the care of children, including the children themselves, if the mortality is to be lowered.

(3) *Education*

This is the field in which most progress can be made, and certain situations and individuals commend themselves as being eminently suitable for educational work.

(a) *Antenatal and postnatal clinics*. Mothercraft instruction is available to all mothers at these clinics, and this provides an excellent opportunity to discuss safety in the home. Both here and at

(b) *Infant welfare clinics*, suitable literature detailing common dangers can be given out, such as that published by Ro.S.P.A. (the Royal Society for the Prevention of Accidents).

(c) *Health visitors* have a unique opportunity to draw attention to existing hazards when they visit the home, and to suggest measures for improving safety.

(d) *General practitioners* also have opportunities to point out dangers when they make home visits, and can use propaganda in their surgeries both in the form of posters and of hand-out leaflets.

One thing which all doctors need to remember is to emphasise the potentially poisonous nature of many of the drugs we prescribe, and to instruct parents to keep drugs locked up. This is not always easy to do .Many homes do not have a cupboard which can be locked and which is not frequently opened. Moreover, young children will climb with surprising skill to reach forbidden drugs. The bathroom cabinet seems to be a suitable place to keep dangerous medicines, but it is impossible to obtain in Belfast a bathroom cabinet with a lock. Manufacturers need to be educated, but perhaps if they produced a locking cupboard it would not sell. The firms who have marketed gas and electric cookers with a rim to prevent pots and pans being pulled over, find that they are not popular with the public. If the housewife can be persuaded to demand such safety devices the manufacturers will undoubtedly respond.

(e) *Press and television.* Mass media are relatively unexplored as a means of educating the public in safety, and their use has so far been directed mainly towards the motorist. Perhaps the Ministry of Transport is more aware of their potential value than the Ministry of Health. As we have seen, however, the child mav be more responsible for the accident than the driver, and if we are to prevent child deaths o nthe road we must focus more attention upon the children themselves.

(f) *Schools* Safety taught in schools may help the next generation, but by the time the child reaches school age he is out of the greatest danger period .After the age of 2 or 3 years the home accident rate falls steeply. This means that children are learning. Road accidents are also fewer but still sufficiently numerous for young school children to need to be taught kerb drill. Swimming should be taught as a compulsory subject at school. More adults than children are drowned every year—some might have lived if they had learned to swim while at school. First aid could easily be taught to senior students and this would have great value in the lives of the next generation. It is not unusual to see children with improperly treated burns or having bled from a deep wound, who arrive at hospital in a very serious condition which might have been avoided by prompt emergency treatment. In most schools, only those students sufficiently enthusiastic to join a Red Cross or St. John's Ambulance group are given any instruction in this subject.

Wheatley (1965) in a recent article showed that the introduction of driving instruction in high schools has considerably lowered the incidence of death and injury among young drivers in certain parts of the United States. It is reasonable to assume that the lives of other road users, including children, will similarly be at less risk in these communities.

The main lessons, however, must be learned at home, and are best taught by example. The child who sees his father pour paraffin on to a bonfire will do the same. The child whose parent lights a piece of paper from an electric fire element will try to do the same. To quote Dietrich (1965) of Los Angeles who wrote recently . . . "The years between 1 and 5 must be devoted to promotion of education in safe behaviour, along with bravery in diminishing the dependence on 'Protection'. 'Protection' must always be maintained against lethal and subtle hazards, but the young child must be taught to do with safety all the things he is capable of doing . . .

"By the age of 5 or 6 years, school and play take the youngster from the pro-

tective tent of the home. His safety then becomes much more dependant on what he knows and how he acts, than on any protective laws that the family and community have hopefully enacted on his behalf . . .

"But whether or not he dashes unlooking into the street, drinks kerosene from a coke bottle in a neighbour's garage, sets fire to a pile of waste and himself, plunges a bobby pin into an electric outlet, or slips into a 30 ft. reservoir, depends not on any statutory codes but on *how he behaves*".

Dietrich states his case with conviction. As the child develops, protection is followed by education, and then by responsibility. When the child has been fully taught, and has comprehended, the dangers of his environment and the rules of safety, he must be fearfully, gently, but firmly, pushed into independence.

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LEFT BUNDLE BRANCH BLOCK

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A RECENT STUDY of the prognosis of patients with left bundle branch block (L.B.B.B.) suggested that life expectancy was extremely short (Smith and Hayes 1965). While in general L.B.B.B. has a sinister reputation, we have been struck by a few individuals who have survived many years with L.B.B.B., and we thought an analysis of some of the material available at the Royal Victoria Hospital, Belfast, would be of value.

MATERIAL

In the years 1956 and 1957, 111 of the patients who attended the electrocardiographic department of the Royal Victoria Hospital had L.B.B.B. In all cases the hospital records were studied, and additional data were obtained from the family doctors. Where possible, survivors were examined personally by the authors. In eight cases complete data were not available (a loss to follow-up of 7 per cent).

Electrocardiographic criteria for L.B.B.B. were those of the New York Heart Association (1964). "The QRS interval is 0.12 seconds or more and the components of the QRS are notched and slurred. In leads I, aVL, sometimes aVF, and in leads from the extreme left side of the precordium and the left side of the thorax, the initial deflection is usually an R wave or one of its prominent notches occurs relatively late in the QRS interval. The ST segment is most often displaced in a direction opposite to the principal QRS deflection and the T wave also points in this direction. The appearance of QRS in other leads depends principally on the average direction of the electrical axis of QRS".

In all cases where statistical testing of significance was required the "chi-square" method was used. All differences mentioned in the text were significant at the 1 in 20 level ($P < 0.05$).

RESULTS

Incidence

During the years 1956-57, 9,469 electrocardiographs were recorded at the Royal Victoria Hospital. The 111 patients with L.B.B.B. represent about 1 per cent of this total.

Age and Sex

The age and sex distribution of the patients are shown in Table I. The majority of patients (87 per cent) were aged 50 years or over, and about one-third (34 per cent. were aged 70 years or more. Approximately half (45 per cent) the series were male, and there was no significant difference in the sex incidence between different age groups.

TABLE I
Age and sex distribution

Age (years)	*Male*	*Female*	*Total*
28-49	6 (43%)	8 (57%)	14
50-69	31 (53%)	28 (47%)	59
70-87	14 (37%)	24 (63%)	38
Total	51 (46%)	60 (54%)	111

Aetiology

Aetiological factors were analysed (Table II). Over half the patients (55 per cent) were thought on clinical or autopsy grounds to have coronary arterial disease, and one-third (34 per cent) had hypertension (diastolic pressure greater than 110 mm. Hg.). These two factors between them accounted for 69 per cent of the patients.

TABLE II

Aetiology

Aetiology	*No. of patients*	*Percentage*
Ischaemic heart disease	58	55
Hypertension	36	34
Ischaemic heart disease and/or hypertension	74	69
Rheumatic heart disease	6	6
Syphilis	2	2
Congenital heart disease	1	1
Scleroderma	1	1
Unknown	22	21

A further 10 per cent of patients had rheumatic, congenital, syphilitic or scleroderma heart disease, but in 22 patients (21 per cent) no aetiological factor was known.

Sex did not appear to influence the incidence of any of the aetiological factors though both patients with the syphilitic heart disease were males.

TABLE III

Aetiology in relation to age

Aetiology	*Age Group*		
	28-49 years	50-64 years	70-81 years
Ischaemic heart disease	3 (23%)	37 (66%)	18 (49%)
Hypertension	2 (15%)	26 (46%)	8 (22%)
Ischaemic heart disease and/or hypertension	5 (38%)	47 (84%)	22 (59%)
Rheumatic heart disease	6 (46%)	0	0
Congenital heart disease	1 (8%)	0	0
Syphilis	1 (8%)	0	1 (3%)
Unknown	0	9 (16%)	14 (38%)*

* Includes one case of scleroderma.

As might be expected, patients with rheumatic and congenital heart disease were younger than average (Table III). Patients in whom no aetiological factor was found, occurred more frequently in the older age groups and in fact represented 38 per cent of patients aged 70-87 years, 16 per cent of patients aged 50-69 years and none of the youngest age group (28-49 years).

Clinical Status

The clinical status of the patients at the time the electrocardiographs (ECGS) were recorded is shown in Table IV. Almost half the patients (48 per cent) had no or mild symptoms (Grade I or II functional disability—N.Y.H.A. 1964). Twenty-

TABLE IV

Clinical status in relation to age

Age (years)	*Clinical status* I *and* II	III	IV	*Total*
28-49	9 (69%)	3 (23%)	1 (8%)	13
50-69	28 (50%)	14 (25%)	14 (25%)	56
70-87	14 (38%)	6 (16%)	17 (46%)	37
Total	51 (48%)	23 (27%)	23 (27%)	106

seven per cent were in Grade III and 27 per cent in Grade IV. Sex did not appear to influence the clinical status but the younger patients had a lower incidence of Grade IV disability than the older age group (22 per cent of age group 28-69 years, and 46 per cent of the remainder).

Electrocardiographs

The ECG of all patients were analysed with regard to mean frontal QRS axis and mean frontal gradient. In addition the following patterns which have been said to correlate with myocardial infarction were searched for — (1) slurred $S V_{3 \text{ and } 4}$, (2) $S V_{5 \text{ and } 6}$, (3) broad $qV_{5 \text{ and } 6}$. The results are seen in Table V.

TABLE V

Analysis of electrocardiograms (103 *patients*)

E.C.G.	*Abnormal*
Mean frontal axis (left of -30°)	34 (33%)
Mean frontal gradient (left of -17°)	41 (40%)
$q V_{5 \text{ and } 6}$ (q > 0.02 seconds)	1 (1%)
Slur $SV_{3 \text{ and } 4}$	23 (22%)
$S V_{5 \text{ and } 6}$	61 (61%)

Abnormal mean frontal axis (to the left of -30°) occurred in 33 per cent of patients, and an abnormal gradient (to the left of -17°) in 40 per cent. A q wave greater than 0.02 seconds occurred in one patient only, and a slurred $SV_{3 \text{ and } 4}$ in 22 per cent of patients. None of these electrocardiographic features correlated with age, sex, clinical status, or aetiology.

Radiology

X-ray of patients taken near the time the ECG was recorded were studied, and those in whom the cardiothoracic ratio exceeded 50 per cent were described as having cardiomegaly. Seventy-four patients (71 per cent) had cardiomegaly, and it occurred more frequently (80 per cent) in patients in disability Grade III or IV than in patients in Grade I or II (62 per cent). Cardiomegaly showed no correlation with age, sex or aetiology.

Survival

Survival figures for the group are seen in Table VI. Twenty patients (19 per cent) died within 1 year of the ECG being recorded, and 54 per cent were dead within 4 years. A further 17 per cent died 5-9 years later and 29 per cent are still alive.

TABLE VI

Survival

Survival	*Number of patients*
0-4 years	56 (54%)
5-9 years	18 (17%)
Alive 1966	30 (29%)
Total	104

Various factors were studied to see if they influenced survival. Table VII shows the effect of age. Patients aged 70-87 years at the time the ECG was recorded had a worse prognosis than the remainder, only 16 per cent surviving more than 9 years, compared with 43 per cent of the younger patients.

TABLE VII

Survival in relation to age

Age	*Survival*			*Total*
(years)	*0-4 years*	*5-9 years*	*Alive*	
28-49	4 (8%)	25 (47%)	24 (45%)	53
50-69	4 (19%)	9 (43%)	8 (38%)	21
70-87	5 (16%)	20 (67%)	5 (16%)	30
Total	13	54	37	104

Table VIII correlates survival against clinical status. As might be expected, patients in functional disability Grade I or II did much better than patients with more severe symptoms. No patients in Grade IV survived more than 9 years and 81 per cent died within 5 years. This compares with 47 per cent of patients in Grade I or II who are still alive, and only 35 per cent who died within 5 years.

TABLE VIII

Survival in relation to clinical grading

Clinical	*Survival*			
grading	*0-4 years*	*5-9 years*	*Alive*	*Total*
I-II	19 (37%)	8 (16%)	24 (47%)	51
III	9 (43%)	6 (29%)	6 (29%)	21
IV	26 (81%)	6 (19%)	0	32
Total	54	20	30	104

Cardiomegaly was found to correlate with survival time (Table IX). Patients in whom the cardiothoracic ratio was less than 50 per cent had a higher percentage of patients surviving more than 9 years (47 per cent) than patients in whom the

TABLE IX

Survival in relation to cardiac size

Cardiac size	*Survival*			*Total*
	0-5 years	*5-9 years*	*Alive*	
Cardiothoracic ratio >50%	42 (56%)	16 (22%)	16 (22%)	74
Cardiothoracic ratio < 50%	14 (47%)	2 (7%)	14 (47%)	30
Total	56	18	30	104

cardiothoracic ratio exceeded 50 per cent (22 per cent). However, when heart size was analysed within groups of similar functional disability (Table X) no significant correlation with survival was found.

Other factors studied—sex, aetiology, and ECG findings (mean frontal axis, frontal gradient, $S V_{5 \text{ and } 6}$, slurred $S V_{3 \text{ and } 4}$, wide $qV_{5 \text{ and } 6}$ showed no correlation with survival.

TABLE X

Survival in relation to cardiac size within the clinical grading groups

Clinical grade	*Cardiothoracic ratio*	*Survival* 0-4 *years*	5-9 *years*	*Alive*	*Total*
I and II	< 50%	7 (37%)	1 (5%)	11 (58%)	19
	> 50%	12 (39%)	6 (19%)	13 (44%)	41
III and IV	< 50%	7 (64%)	1 (9%)	3 (27%)	11
	> 50%	30 (70%)	10 (23%)	3 (7%)	43

DISCUSSION

Left bundle branch block is essentially an electrocardiographic diagnosis and thus its true incidence in the general population is difficult to assess: however, it occurs only rarely (Hiss and Lambe 1962; Ostrander 1964). The incidence in patients referred to an ECG department of a hospital has been estimated at 1 per cent (Katz and Pick 1956) and this is similar to our findings. All workers in the field have found L.B.B.B. to occur much more commonly in older age groups than in younger persons.

Aetiological factors have proved similar in most studies of the subject (Johnson *et al.* 1957; Conyers *et al.* 1965). The majority of patients with L.B.B. have coronary arterial disease and/or hypertension. Other findings occurring less frequently include rheumatic or syphilitic heart disease, calcific aortic stenosis and primary myocardial disease. These findings are similar to those in the present study. Our finding of a relatively high percentage of patients in whom no aetiological factor was found, is difficult to evaluate due to a low autopsy rate. It is likely that many of these patients had coronary arterial disease which had not manifested itself as clinical angina or myocardial infarction. However, a number of these patients may have had primary myocardial disease and in at least one patient autopsy findings revealed an essentially normal heart with only minimal coronary arterial atheroma. A comprehensive pathological survey would be necessary to assess its true incidence. In one such study (Conyers *et al.* 1965) 5 per cent had primary myocardial disease and 1 per cent had amyloidosis.

Since L.B.B.B. causes a completely abnormal route for the depolarisation wave in the left ventricle, the QRS-T complexes are markedly altered from normal. The difficulty in diagnosing myocardial infarction in the presence of L.B.B.B. is well known (Wilson *et al.* 1945). However, even in the presence of L.B.B.B. some QRS patterns are said to be suggestive of myocardial infarction (Sodi-Pallares 1956, Massie and Walsh 1960). We have studied $S V_{5 \text{ and } 6}$, $q V_{5 \text{ and } 6}$, and slurred $S V_{3 \text{ and } 4}$. However, like Norris (1961) we found none of these correlated with the clinical diagnosis.

Some workers (Pantridge 1951; Sodi-Pallares 1956) have found that an abnormal ventricular gradient occurs more frequently in patients with severe underlying

disease. It is therefore surprising that we could find no correlation between ventricular gradient and clinical status or survival.

Our main interest in undertaking the present study was to ascertain the prognosis of the condition. While it is generally recognised that the prognosis depends on the underlying heart disease (Rodstein *et al.* 1951), the finding of L.B.B.B. in any patient indicates the need for giving a guarded prognosis (White 1944). A recent study of hospital cases (Smith and Hayes 1965) showed a mean survival time of 36 months, even shorter than the classical study by Johnson *et al.* (1957) who found a mean survival of 40 months. However, both these studies included patients who were still alive at the time the assessment was made. Using the same technique with our figures the mean survival time was 5.6 years. These figures are dependant on the form of the study and do not give a fair estimate of prognosis. The fact that 29 per cent of our patients were still alive at least 8 years after L.B.B.B. had been recorded and that some of these have little cardiac disability gives a more valid indication of prognosis. Even in the older age groups, where life expectancy is under 11 years in females and 9.5 years in males (Registrar General, N.I. 1961), 16 per cent of the patients were still alive 9 years later. It is clear that L.B.B.B. does not of necessity denote a short life span.

Remarkably few of the factors studied seemed to influence the prognosis. Younger patients, whose life expectancy is naturally better, survived longer than older patients. This was also noted by Johnson *et al.* (1957). Cardiac status at the time the patient entered the study also influenced survival and this is as one would have expected. Johnson *et al.* (1957) found that cardiomegaly was associated with a poor prognosis and this is also seen in our figures. However, this appears to be related to the fact that heart size correlated with clinical status: we found that within groups of similar clinical status, heart size did not influence survival time.

All the remaining factors examined—electrocardiographic findings, aetiology and hypertension—were of no value in predicting survival time.

Summary

A study has been made of all patients attending the ECG department of the Royal Victoria Hospital, Belfast in 1956-57 in whom complete L.B.B.B. was found. The incidence of L.B.B.B. in this population was 1 per cent.

L.B.B.B. occurred more frequently in the older age groups.

The commonest aetiological factors were ischaemic heart disease and hypertension alone or in combination. However, in 21 per cent no aetiological cause could be determined.

The ECG itself proved to be of no value in determining aetiology or severity of the condition.

Twenty-nine per cent of patients remain alive 9-10 years after L.B.B.B. was found. The only factors affecting prognosis were age and clinical status at the time L.B.B.B. was discovered.

Acknowledgments

We would like to thank the physicians of the Royal Victoria Hospital, Belfast, and the family doctors for their help.

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BOOK REVIEWS

PSYCHIATRY FOR STUDENTS. By David Stafford-Clark, M.D., F.R.C.P., D.P.M. (Pp. 276; figs. 3. 21s). London: George Allen & Unwin, 1966.

The first edition of this book was reviewed in this Journal. Changes in this edition, now published in paper back form and costing fourteen shillings less, are outlined in the preface. The description of the practical technique for the induction of hypnosis has been omitted. Reference is now made to the application of learning theory to the treatment of neuroses. Chronic schizophrenia and the pychiatric aspects of epilepsy receive more attention.

The early appearance of a second edition reflects the popularity of this book. There is no reference to the Mental Health Act (Northern Ireland) 1961, which, like the Mental Health Scotland Act 1960, does not have a category dealing with psychopathic disorder.

This book will continue to appeal to a wide range of students. General practitioners will find much practical guidance within its pages. J.G.G.

AND AFTER THAT NURSE? Compiled by Roger Brook. (Pp 62. 8s. 6d.). London: Souvenir Press, 1966.

This consists of hundreds of nursing "howlers" taken from examination papers. Especially under stress we all make silly mistakes and only if we acknowledge this should we laugh at these "howlers". Perhaps a rather juvenile sense of humour is necessary for the full enjoyment of this book and its reception will vary widely with the reader.

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A CASE OF A PENETRATING INJURY OF THE GALL BLADDER DUE TO A KNIFE WOUND IN A NINE YEAR OLD BOY

By F. P. S. MARRIOTT

Senior Surgical Registrar, Surgical Unit, Ards Hospital, Newtownards, N. Ireland

PERFORATION of the gall bladder due to injury is extremely rare because of the protected position it occupies under the right costal margin.

Perforation may be caused either by blunt trauma to the upper abdomen or by a penetrating injury. Perforation of the gall bladder by a penetrating injury has not so far been described in a child.

Case Report

The patient, M.M., a nine year old boy was admitted to Ards Hospital on the evening of 25th January, 1964, at 9.30 p.m. An hour previously he had been running along a wall, a few feet above the ground, when he tripped, fell and landed doubled up on the ground. In his right hand trouser pocket he had a knife six inches long, the blade of which was pointing upwards. The point of the knife stuck into his right upper abdomen.

He pulled out the knife himself and ran home. He was seen by a medical practitioner and as the wound appeared superficial two sutures were inserted. He was then transferred to Ards Hospital for observation.

On admission he was a healthy looking, co-operative boy of nine years. His pulse was 84 beats per minute. Blood pressure 130/80 mm. of mercury and temperature 97°F.

There was a transverse wound, 2 cms. long, below the right costal margin. Two sutures had been inserted. Slight tenderness and rigidity were present in the right hypochondrium. The rest of the abdomen was normal to palpation and bowel sounds were present. Other symptoms showed no abnormality.

In view of the slight upper abdominal tenderness, rigidity and the possibility of intra-abdominal injury, exploration of the wound was undertaken.

Operation

The sutures were removed from the stab wound. The wound was probed and the probe passed obliquely into the peritoneal cavity. A right upper paramedian incision was then made to allow a more complete exploration.

On opening the peritoneal cavity a large quantity of bile mixed with blood was found. The liver was uninjured. On further exploration a penetrating wound of the peritoneal surface of the gall bladder was found. This was 8 mm. long and 2-3 cms. from the neck of the organ. The lesser omentum was oedematous. The duodenum, stomach, small intestine and colon were uninjured.

The laceration of the gall bladder wall was closed with a purse string suture of 2/0 chromic catgut, reinforced with sero-muscular sutures of the same material. The wound was closed with intra-peritoneal drainage.

Post-Operative

Drip and suction was continued for 48 hours after operation. The drain was removed in 5 days and drainage ceased 2 days later. He was given penicillin and streptomycin by injection for 9 days and discharged symptom free at the end of two weeks. He was completely well when seen at the follow-up clinic two weeks after discharge from hospital.

Discussion

The gall bladder is rarely injured because of the protected position it occupies, surrounded by the costal margin and liver anteriorly, the right kidney posteriorly and the vertebrae medially.

Damage can occur either due to penetrating injuries of the right upper abdomen with sharp instruments or bullets or by severe blunt trauma to the same region, as may occur with steering wheel injuries, blows and falls from a height.

From the literature from 1942 onwards I have been able to collect 138 cases of penetrating injury of the gall bladder in adults but have not found a single case reported in a child. From 1898 there have been over 50 cases of injury to

TABLE I

Penetrating Injuries of the Gall Bladder
Review of the Literature 1942–1964

Author and Year	*No.*	*Additional Non. Penet. Injuries G.B. in series*	*Injury other organs (Exclud. Liver)*	*Mortality*	*Abdominal Injuries %*	*Cause*	*Remarks*
Gordon-Taylor 1942	15	1 (age 3)	—	? —— 1	1.25%	(a) Military (b) Air Raid	12-599 Abd. inj. 3-600 „
Sako et al. World War II 1942-1945	53	—	18	18	1%	Military	2nd Aux. Surg. Group
Volvinder & Jones 1947	1	—	—	—	0.3%	Needle liver biopsy	Not operated 273 biopsies
Sako et al. Korean War 1952-1953	33	—	—	—	8.2%	Military	Forward Unit
Rubenstone 1952	1	—	—	1	?	Needle liver biopsy	Fatal
Gamble and Sullivan 1953	2	—	—	—	0.6%	Needle liver biopsy	325 biopsies
Sparkeman and Fogelman 1954	1	1	—	—	2%	Needle liver biopsy	Review of 100 liver injuries
Hall 1954	23	2	2	2	3%	14 Bullet 9 Stab	Houston, Texas
Wilson et al. 1961	6	—	2	2	5.8%	2 Stab 4 Bullet	Tenessee
Penn, 1962	3	2	—	—	5.2%	2 Stab 1 Liver biopsy	Johannesburg
Marriott, 1964	1	—	—	—		Stab	N. Ireland
TOTAL	139	6	22	24	3% Av.		

the gall bladder from non-penetrating injury. I have been able to confirm 44 of these, including 26 cases reviewed from the literature by Smith and Hastings in 1954, who added one case of their own.

Penetrating Injuries

Table I gives a review of the 138 cases of penetrating gall bladder injuries reported since 1942. Prior to this the literature is scanty. Of these 101 were caused by injuries sustained in war and this includes 3 civilian air raid victims. In the 37 patients who sustained the injury in peace time, 18 had bullet wounds, 13 stab wounds and in 6 the injury was due to liver biopsy, including one fatal case.

The percentage of gall bladder injuries found at laparotomy for penetrating abdominal injury in war varies from 0.5 per cent in 600 civilian air raid casualties described by Gordon-Taylor in 1942, to 8.2 per cent for 33 gall bladder injuries out of a total of 402 laparotomies for abdominal injury treated by a Forward Surgical Unit during the Korean War (Sako et al). No gall bladder injuries were reported in a series of 64 carefully tabulated laparotomies for abdominal injury in the Western Desert reported by Lowden (1944).

In civilian practice 37 patients had penetrating injuries of the gall bladder. Eighteen were caused by bullet wounds, 13 by stab wounds and in 6, including one fatal case, the injury followed liver biopsy. Hall, from Houston, Texas, describes 25 cases of gall bladder injury, 14 due to bullet wounds, 9 to stab wounds and 2 following blunt trauma. Wilson and Sherman (1961), from Memphis, Tenessee, describe 2 gunshot wounds and 2 stab wounds. Penn, in 1962, from Johannesburg, describes 2 gall bladder injuries due to stab wounds, 1 due to liver biopsy and 2 due to blunt trauma. Sparkman and Fogelman (1954) in a series of 100 consecutive liver injuries describe 2 associated gall bladder injuries, one following liver biopsy. Hannah, Bell and Cochrane (1965) in a series of 112 liver injuries out of 187 fatal accidents, and 48 clinical cases with a mortality of 31 per cent, do not report any gall bladder injuries.

Mortality

In the above series the mortality of injury to the gall bladder alone and treated by operation was nil. Death was caused by injuries to other organs, the liver, colon, duodenum, chest and right kidney being frequently involved. Sako et al (1955) described 53 gall bladder injuries in World War II, and 18 complicated by other injuries died. In the Korean War, 33 patients with uncomplicated gall bladder injury survived following operation. In the civilian series death was again due to associated injuries.

Non Penetrating Injuries (44 cases)

These were due to severe blunt injury to the right upper abdomen, following injury by the steering wheel in automobile accidents, kicks, blows and falls from a height. There were 4 cases of gall bladder injury and 1 case of injury to the common bile duct in children aged between 3-9 years (Gordon-Taylor, Hicks, Coulter, Benson et al.). Smith and Hastings (1954) reviewed 26 cases of non-penetrating injury of the gall bladder and added one of their own. Other series were recorded by Martin et al. (1960), Newell (1948), Manlove et al. (1959), Brickey et al. (1960), and Hogue and Munell (1963).

In non-penetrating injuries the diagnosis was often delayed. The usual history was of a blow to the upper abdomen. Findings on initial abdominal examination were often slight. There followed, however, gradually increasing upper abdominal pain, tenderness and rigidity with increasing adbominal distension. At laparotomy bile was found in the peritoneal cavity and the injury to the gall bladder discovered. The time between the original injury and laparotomy was usually 2-3 days, but was sometimes as long as 2-3 weeks. Whilst infected bile produces a rapidly spreading, fatal peritonitis, the chemical peritonitis produced by sterile bile is much less serious, especially if loculation occurs. If, however, damage to the biliary tract is suspected, laparotomy should be performed as the bile may become infected from the bowel wall.

Treatment of Gall Bladder Injury

The course followed when an injured gall bladder is found at operation will depend on the severity of the injury and the presence of associated injuries. The injury may be treated by (1) Simple Suture; (2) Cholecystostomy; or (3) Cholecystectomy.

Simple Suture was performed in our own case. The results are entirely satisfactory if the laceration is small and the gall bladder wall otherwise healthy. The wound is drained.

Cholecystostomy is the treatment of choice for the more severe injuries, especially when the pateient's condition is poor due to associated injuries (Gordon-Taylor).

Cholecystectomy is performed as a routine by some surgeons but the operation can be a difficult and dangerous one when the anatomy of this area is altered by haemorrhage and oedema. It is, however essential in traumatic avulsion of the gall bladder.

Laparotomy is indicated in all penetrating wounds of the abdomen and in non-penetrating wounds when increasing pain, tenderness and rigidity indicate an intra-abdominal lesion.

Summary

A penetrating injury of the gall bladder in a nine year old boy due to a knife wound is described. This was treated by simple suture of the gall bladder with uneventful recovery.

The literature is surveyed, 138 cases of penetrating injury of the gall bladder are reviewed, in association with 44 non-penetrating injuries. The mortality in cases treated by operation was due to damage to other organs. There were no deaths in cases treated by operation where the gall bladder alone was injured.

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BOOK REVIEW

ISCHIO-FEMORAL ARTHRODESIS. By J. Crawford Adams, M.D.(Lond.), M.S.(Lond.), F.R.C.S.(Eng.). (Pp. vi+112; figs. 107. 35s.). Edinburgh and London: E. & S. Livingstone, 1966.

THIS is an admirable example of a monograph, in that it achieves the object of being a comprehensive study of a subject, which in itself would not warrant book-length treatment.

The history of the development of this type of arthrodesis is outlined. This is followed by clinical and operative experience which is illustrated by beautiful drawings, photographs, and X-rays. Figures of results are also given, including those of earlier authors.

Mr. Crawford Adams then expounds the main theme of the monograph. This is to put forward the claim for this operation to be done by the "nail and graft" technique. Research into the type of bone graft, and the operative techniques has been done by the author and the chapters dealing with this research are most interesting.

His conclusions are not as strongly in support of the technique as the reader would expect from arguments propounded in the earlier parts of the monograph. He does, however, advocate the "blind method" for most cases. Many orthopaedic surgeons will not be encouraged about the safety of the sciatic nerve from injury in this operation . . . especially when Mr. Adams' experiments show that the nerve is "at least one centimetre" from the line of the drill. Anyhow, the figures for fusion by the ischio-femoral method (taken from his tables) show a greater percentage of fusions in the cases in which the "open method" was used. There is a significant absence of comment about the complications of well-leg traction, which forms the preliminary treatment for many cases.

This is an excellent monograph and its study will well repay those interested in the problems of hip surgery. It is also a very comprehensive reference for these problems.

R.I.W.

GAMBLE, R. D. and SULLIVAN, B. H. (1953). *Gastroenterology*, **24**, 394.
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BOOK REVIEW

ISCHIO-FEMORAL ARTHRODESIS. By J. Crawford Adams, M.D.(Lond.), M.S.(Lond.), F.R.C.S.(Eng.). (Pp. vi+112; figs. 107. 35s.). Edinburgh and London: E. & S. Livingstone, 1966.

THIS is an admirable example of a monograph, in that it achieves the object of being a comprehensive study of a subject, which in itself would not warrant book-length treatment.

The history of the development of this type of arthrodesis is outlined. This is followed by clinical and operative experience which is illustrated by beautiful drawings, photographs, and X-rays. Figures of results are also given, including those of earlier authors.

Mr. Crawford Adams then expounds the main theme of the monograph. This is to put forward the claim for this operation to be done by the "nail and graft" technique. Research into the type of bone graft, and the operative techniques has been done by the author and the chapters dealing with this research are most interesting.

His conclusions are not as strongly in support of the technique as the reader would expect from arguments propounded in the earlier parts of the monograph. He does, however, advocate the "blind method" for most cases. Many orthopaedic surgeons will not be encouraged about the safety of the sciatic nerve from injury in this operation . . . especially when Mr. Adams' experiments show that the nerve is "at least one centimetre" from the line of the drill. Anyhow, the figures for fusion by the ischio-femoral method (taken from his tables) show a greater percentage of fusions in the cases in which the "open method" was used. There is a significant absence of comment about the complications of well-leg traction, which forms the preliminary treatment for many cases.

This is an excellent monograph and its study will well repay those interested in the problems of hip surgery. It is also a very comprehensive reference for these problems.

R.I.W.

A CASE OF LYMPHOSARCOMA OF THE SMALL INTESTINE

***By H. L. KHANNA, F.R.C.S.E.**
Surgical Registrar, Darlington Memorial Hospital, and
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Senior Consultant Surgeon, Darlington and Northallerton Hospital Groups
*Now Neurosurgical Registrar, Royal Victoria Hospital, Belfast

THE FOLLOWING CASE of lymphosarcoma of the small intestine is considered worthy of record, not only because it exemplifies many of the characteristic features of this condition but also because it serves as a reminder of a potential cause of recurrent melaena which might otherwise be overlooked.

Case Report

Mr. J. M. S., a surveyor aged 45, suffered from indigestion for many years. During the war his dyspepsia was investigated on several occasions and in Cairo in 1944 he had a barium meal examination, but no evidence of peptic ulcer was discovered. Because of the repeated negative findings at clinical and radiological examination he was considered to be suffering from nervous dyspepsia.

At times he complained of episodes of colicky abdominal pain and he himself thought that he might be suffering from appendix colic. It was not until November 1958, that the pattern of his symptoms changed and he observed that the abdominal colic was followed by vomiting and the passage of loose bowel motions. On the 4th March, 1959, while at work at his office desk he felt faint and subsequently had melaena. He was admitted to St. Thomas' Hospital at which time his haemoglobin had fallen to 70 per cent., but with conservative management on an ulcer regime his condition rapidly improved and after two weeks the haemoglobin level reached 84 per cent. Barium meal showed pylorospasm but no peptic ulcer.

Later the same year, on 26th October, he was admitted to Croydon General Hospital with a more severe episode of melaena, in which his haemoglobin fell to 57 per cent. and a transfusion of four pints of blood was given. Barium meal again did not show peptic ulceration, but in view of the long history of dyspepsia with repeated melaena and occasional episodes of hunger pain, it was considered likely that he was suffering from duodenal ulcer. He was discharged from hospital after three weeks on a strict post ulcer regime.

During the period 1960 to 1963, he continued to have episodes of colicky abdominal pain with sickness and diarrhoea. This occurred as often as six times a year, and during many, but not all, of these attacks he had melaena. He did not seek further medical advice until the 10th December, 1963, when he was admitted during an asymptomatic period to the Friarage Hospital, Northallerton, for investigation. The results of these investigations were as follows:

Barium meal and follow through examination: The oesophageal hiatus was normal and there was no oesophageal reflux on tilting the patient. The stomach and duodenum were normal and there was no evidence of peptic ulcer. Using a small bowel technique no abnormality was detected in the jejunum or ileum and centainly nothing to suggest Crohn's disease.

Gastric secretion tests: The response to histamine, and the level of all night secretions were slightly, but not conspicuously, above the normally accepted levels.

Oesophagoscopy and Gastroscopy: These examinations conducted under general anaesthesia showed no abnormality in either oesophagus or stomach.

Faecal occult blood tests: Three successive stool examinations were negative for occult blood.

Clinical examination at this time showed no abnormality whatever on careful abdominal examination. The possibility that the attacks of pain and diarrhoea were due to food allergy was considered, but there was no supporting evidence for this conjecture. It was decided that the abdomen should be explored as soon as the patient's professional commitments would allow his admission.

While awaiting re-admission he again collapsed in the street in Newcastle-upon-Tyne and was admitted to the Royal Victoria Infirmary as an emergency on 20th November, 1964. The clinical presentation was the same as that in the previous attacks except that on this occasion he passed some red blood per rectum as well as the tarry stools typical of all the previous episodes.

Abdominal examination on admission, showed a suspicion of a mass in the right iliac fossa which significantly had disappeared the following day by which time the patient's pain had cleared up. This was the first occasion on which the possibility of intussusception was considered. Barium enema at this time showed no abnormality after routine blood transfusion. The patient made as good a recovery as on previous occasions and was discharged ten days after admission.

After convalescence he was admitted to Darlington Memorial Hospital for laparotomy.

Operation (5th December, 1964): Under general anaesthesia the abdomen was explored through a lower right paramedian incision. At a position near the middle of the small gut there were two separate tumour masses involving the entire gut wall and separated by a portion of normal looking, but slightly dilated small intestine. In the mesentery, adjacent to the two tumours there were enlarged, soft and congested lymph nodes, with occasional calcified nodules suggestive of former tuberculous mesenteric lymphadenitis. A wide resection of the involved bowel segment together with the related lpmyh nodes was carried out, and intestinal continuity was restored by end-to-end anastomosis. Post-operative recovery was uneventful.

The Specimen: The specimen consisted of 50 cms. of small intestine in which there were two distinct portions, each 7 cms. in length, in which there was gross thickening of the bowel wall. The tumours encroached on, and surrounded the lumen of the bowel. The segment of gut between the two lesions measured 14 cms. and showed some dilatation though the mucosa appeared normal. In the root of the mesentery there were enlarged, soft lymph nodes and old calcified tuberculous foci.

On microscopy the whole thickness of the two affected segments of small intestine were infiltrated by sheets of small round cells. The lymph glands in the root of the mesentery had likewise lost their normal architecture which was similarly replaced by sheets of uniform small round cells. The appearance was typical of lymphosarcoma of small intestine.

Discussion

The outstanding feature of this case was the long dyspeptic history, with episodes of melaena which had lead to the provisional diagnosis of duodenal ulcer, in spite of repeated negative findings on barium meal examination. That tumours of the small bowel may cause gastric upset with pain and sickness at variable intervals after meals has been mentioned by Frazer (1945), but the association of these symptoms with melaena, and negative findings on radiological examination of the small bowel, supported the provisional diagnosis of "post bulbar" duodenal ulcer which is notoriously difficult to demonstrate radiologically. The long duration of symptoms over a period of at least six years was unexpected in a case of malignant disease, though review of the literature shows that similar cases have been recorded by Cape and Grant (1942).

The occurrence of colicky abdominal pain and diarrhoea is a constantly recorded feature of the condition (Lewis, 1934), while alternating constipation and diarrhoea is said to be common (Ullman Abeshouse, 1932).

Sarcoma of small intestine though a rare condition has been recorded sufficiently often to enable its age and sex incidence to be established. The condition is commoner in males than in females, the ratio of frequency being 10-3, and while the condition may occur at any time from infancy to old age, the maximum incidence is in the fourth decade (Marcuse and Stout, 1950). In these respects the recorded case was typical in age and sex incidence.

This case showed the unusual feature of two discrete tumours with apparently healthy gut in the interval between. Both were of the tubular type as distinct from the annular type which tends to present with obstruction, and the polypoid type which shows a tendency to intussusception. In actual fact the shape of both tumours resembled the "aneurysmal dilatation" described by Raiford (1933) in which "the tumour resembling a hollow sphere with a moderate degree of construction at either end".

Microscopically the lesion was shown to be a lymphosarcoma and is the commonest type of growth. Only 6.8 per cent. being myogenous in origin. (Brink and Laing, 1933).

In general the prognosis is said to be bad (Wakeley, 1932), but cases of survival for seven (Mayo and Nettrour, 1936) or even for twenty years have been recorded (Cameron, 1938). In view of the long duration of the symptoms and a wide resection with no microscopic evidence of residual metastasis the outlook in this case can be regarded with guarded optimism.

Comment

The case history here and a review of the literature emphasises that in cases of vague dyspepsia with unexplained melaena the possibility of a sarcoma of the small intestine should be considered, and early laparotomy advised.

We would like to express our thanks to Dr. W. Irwin, Consultant Radiologist, for the radiological reports, to Dr. J. Tregillus, Consultant Pathologist, for the section reports, and to Dr. P. N. Coleman for photomicrographs.

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BOOK REVIEW

PAEDIATRIC PATHOLOGY. By Daniel Stowens, M.D. Second Edition. (Pp xi+847; Illustrated, 212s.). Baltimore: Williams & Wilkins, Edinburgh and London: E. & S. Livingstone, 1966.

The appearance of a second edition of this important and valuable book seven years after the first is welcomed. Dr. Stowens in his preface expresses pleasure at the opportunity it gives him to correct ideas which, as he says, should never be held sacred or inviolable, and to present new concepts.

A book such as this must consider not only the expert reader but also the junior pathologist and the clinician. It is very difficult for anyone to gain an insight into the conflicting views current on many subjects. The question is whether in a book of this size and authority the author should try to present these conflicting views with citation in the text of the key references, or whether he is entitled to present views and ideas with no clear indication as to whether they are those held by many or some workers or by himself alone. There is often little discussion of the interplay of conflicting opinions in this book. Examples of this might be the author's discussion of hyaline membrane disease and of cot deaths. The expert in the field is interested, stimulated or annoyed by the clear presentation of the author's views, but the less experienced reader is inadequately informed; and no number of references at the end of a chapter can take the place of an analytical review of existing knowledge. The omission of an analysis of the literature and the lack of citation of references in the text of course makes for easier revision of a textbook, but the reviewer considers it is rarely possible to allow that such a textbook is one of the highest scholarship. It can only be admitted as such if it concerns a new and limited field of knowledge which the author has personally explored fully and in depth.

This book must also raise the question of the right of paediatric pathology to be considered a separate division of pathology. The descriptive morphology of many of the conditions considered is adequately or even better given in the larger textbooks of general and special pathology. This book fails to define a biological basis for separating the pathology of this period of life from pathology in general. This failure of a fundamental approach is especially apparent in the chapters on the newborn and on the placenta where discussion under isolated headings may sometimes inform factually but will not increase fundamental understanding.

Despite limitations this is a valuable book and should be available wherever the pathology of children is studied. It will provide help on many problems. It is beautifully illustrated though perhaps too largely by microphotographs. There are numerous references grouped under subject headings at the ends of the chapters. Some heavier pruning of older references might have been useful and there are some peculiar inclusions and omissions, but the range is wide and representative. At present no better book on paediatric pathology exists.

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REVIEWS

DISEASE IN INFANCY AND CHILDHOOD. By R. W. B. Ellis, O.B.E., M.A., M.D., F.R.C.P., and R G. Mitchell, M.D., F.R.C.P.E., D.C.H. Fifth Edition. (Pp. 720; figs. 316. 70s). Edinburgh and London : E. & S. Livingstone.

THE fact that this well-known textbook has now reached its 5th edition, and has, also, been translated into both Spanish and Greek, is good evidence that it meets a widespread need. It is admirably suited to the needs of the student, house officer, and senior nurse, and by virtue of a good bibliography, makes a good starting point for post-graduate reading. References are given to work published as recently as 1965, so that the book is as up-to-date as it is possible for any textbook to be. A considerable amount of new material is included, but close pruning of all that is dated or non-essential has kept the size of the book within its original limits. Very wisely, the detailed discussion of nutritional disorders and of tuberculosis has been retained. As the preface says "mass immigration has given them a renewed importance in this country".

The study of the neo-natal period is one of the "growing points" of pediatrics at present and this section is extremely good. The respiratory distress syndrome is dealt with reasonably; the realistic "anoxia" replaces "asphyxia"; and there is a brief but evocative mention of the commoner chromosome abnormalities. Early operation for meningo-myclocele is spoken of with less than fashionable enthusiasm.

Much more than in previous editions is said about Disorders of Metabolism and Storage —an area in which diagnosis far outstrips successful treatment despite the current multiplicity of cunningly contrived synthetic diets.

Leukaemia is placed firmly among the neoplastic diseases (with a cross-reference in the chapter heading on Diseases of the Blood) and the present position is well and sympathetically summarized.

The chapter on Behaviour Disorders is very good indeed, and though one may ruefully wonder whether the attitudes of parents are not as unalterable as the spots of leopards, a gallant attempt is made to show how parents, home, and school, may all be manipulated for the child's benefit.

Rather a surprising amount of space is spared for one or two rarities—actinomycosis for example, and a colour photograph of the hands in smallpox (though perhaps it is only in Northern Ireland that this is such a rarity—and perhaps not for much longer even here). On reflection, however, it is probably a good thing to deal generously with diseases which the student is not likely to see—commoner conditions he can learn at the bedside.

There are, of course, details on which opinions might differ—surely, for example, no unit competent to operate on pyloric stenosis still gives fluids subcutaneously? It would, however, be churlish to stress small seeming defects which depend perhaps as much on the idiosyncrasies of the reviewer as on any fault in the authors. This is a textbook which can be recommended unreservedly. M.J.L.F.

LIVINGSTONE'S POCKET DICTIONARY (Lois Oakes) by Nancy Roper. S.R.N., R.S.C.N., R.N.T. Tenth Edition. (Pp. vxi+557. 8s 6d). London and Edinburgh : E. & S. Livingstone, 1966.

THIS small volume is intended for nurses, but medical students and practitioners may find it useful. It would be easy to be critical of the short and incomplete definitions given but most are essentially informative. However, one must often wonder what the relatively uninformed reader may glean from brief statements on many specialised subjects.

Useful appendages include abbreviations of degrees and diplomas, but are not sufficiently up to date to include the Membership or Fellowship of the College of Pathologists.

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THIS book was first published in 1945 and each succeeding edition makes a detailed survey of every aspect of the development, problems, and treatment of the cleft palate patient in the light of the known deviations from normality and the history of surgical techniques. It reflects the changes that have taken place in the past 20 years.

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It is written by a speech therapist of long standing experience in the field of cleft palates and who worked with Mr. W. E. N. Wardill, so that the emphasis is very much on the speech competence achieved in these patients and gives progress notes on particular children.

Miss Morley states "By four years of age the great majority of the children seen in recent years had achieved normal articulation, speech therapy being required only in those few who had failed to do so." B.S.K.

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Each chapter has a comprehensive bibliography and the text is supplemented by a large number of excellent photographs. This book should be required reading for all medical workers concerned with the care of children but it will be found to be equally readible and comprehensible to lay social workers in this field. C.M.B.F.

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THIS book was first published in 1945 and each succeeding edition makes a detailed survey of every aspect of the development, problems, and treatment of the cleft palate patient in the light of the known deviations from normality and the history of surgical techniques. It reflects the changes that have taken place in the past 20 years.

This particular edition includes a revised and extended section on orthodontic treatment and has additional information concerning assessment of palato-pharyngeal competence, pressure tests, orifice measurement, lateral radiography, and cine radiography.

It is written by a speech therapist of long standing experience in the field of cleft palates and who worked with Mr. W. E. N. Wardill, so that the emphasis is very much on the speech competence achieved in these patients and gives progress notes on particular children.

Miss Morley states "By four years of age the great majority of the children seen in recent years had achieved normal articulation, speech therapy being required only in those few who had failed to do so." B.S.K.

THE DEVELOPMENT OF THE INFANT AND YOUNG CHILD—NORMAL AND ABNORMAL. By R. S. Illingworth. (Pp. ix+378; figs. 378. 37s 6d). Edinburgh and London: E. & S. Livingstone, 1966.

THIS is the third edition of Professor Illingworth's book on child development and its assessment. Most of the second edition has been completely rewritten and an additional chapter by Dr. Holt on "Seeing and Hearing" has been added. This is much the best book of its kind by an English author and it is written in such clear precise prose that it is a pleasure to read. It describes normal development and assessment together with its pitfalls, difficulties and limitations.

Each chapter has a comprehensive bibliography and the text is supplemented by a large number of excellent photographs. This book should be required reading for all medical workers concerned with the care of children but it will be found to be equally readible and comprehensible to lay social workers in this field. C.M.B.F.

CATALOGUE OF LEWIS'S MEDICAL, SCIENTIFIC AND TECHNICAL LENDING LIBRARY. Part II. Classified Index of Subjects. Revised to December 31, 1963. (Pp. vi+372. 30s to library subscribers, 55s to others). London: Lewis, 1966.

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IN 1889 the first edition of this world famous textbook on tropical diseases by Sir Patrick Manson was published. The fact that this book has now reached its sixteenth edition is testimony to its continued popularity. The first five editions were the work of the original author but the last ten have been by the present author who has clearly left the imprint not only of his editorship but also of his wide knowledge and interests.

The book has a marked clinical orientation although many other aspects of diseases in the tropics have been included. Indeed, the scope of the subject matter which is covered is so wide that the book can be used as a reference for much out of the way information, while at the same time it is an invaluable guide to the management of tropical disorders.

While one hesitates to criticise what has always been regarded as the standard work on tropical diseases, yet it must be stated that the coverage attempted in one book is almost too wide. There are chapters on life in the tropics and the diseases likely to occur therein; tropical fevers subdivided by aetiology; deficiency diseases; skin diseases; poisons of exotic snake and insect origin; disease of the eyes; drugs used in the tropics; medical zoology and a section on clinical pathology. This breadth of coverage has meant an inevitable sacrifice in the presentation particularly in the layout, the size of page and the size of print, which I personally think is a little unfortunate. Perhaps the time has come to present this monumental book in two volumes of larger page size and clearer print—one on clinical tropical medicine and the other on its laboratory aspects. M.G.N.

CARE OF THE NEWLY BORN INFANT. By W. S. Craig, B.Sc., M.D., F.R.C.P. (Edin.), F.R.C.P. (Lond.), F.R.S.E. Third Edition. (Pp. vii+635; figs. 329. 55s). Edinburgh and London: E. & S. Livingstone, 1966.

THE preface to the first edition informs us that this book is intended primarily for the midwife and the family doctor. It is now in its third edition, and has been brought up to date with the collaboration of four members of Professor Craig's department at Leeds. There is also a section on Mothercraft contributed by the Matron of the Simpson Maternity Pavilion, Edinburgh.

The book is produced to the publishers' customary high standards, and is profusely illustrated with excellent clinical photographs. It is simply and dogmatically written. However, the dogmatism occasionally appears unwarranted. For example, we are told that the newborn possess a sense of smell, but are given no supporting evidence for this statement (p. 71). Only two pages are devoted to the respiratory distress syndrome, and we are told that it is open to question whether infants of diabetic mothers are prone to this condition, although few would in fact question the association to-day. One hopes that nobody takes blood from the superior sagittal sinus nowadays, although the procedure is described and illustrated with a photograph on p. 555.

The section on feeding is very detailed and some features will seem strange to persons trained in Belfast. In particular, the Belcroy feeder is recommended in certain circumstances, admittedly with some precautions, although it has been shown that this device frequently induces milk to enter the trachea.

Some criticisms can always be levelled against any book which attempts to give a comprehensive view of a broad field. Some of these reflect no more than personal opinions. On the credit side, the section on dysmaturity is first-class, and throughout the text the experience of the authors is constantly in evidence. There is no doubt that further editions will follow and that many midwives and doctors in training will find it a good investment. J.A.D.

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THIS is a new extensively revised edition of a widely known and respected textbook. It is well described as "a textbook for dental students and a reference book for dental and medical practitioners". The book gives an excellent coverage of disease of the mouth and is well written and pleasantly presented. It contains an immense amount of detail and there are many references to classical or recently published works. Because of this attention to detail the book is more suited to those who already have some understanding of disease of the mouth than to the student reading this subject for the first time.

As in any work of this calibre the individual reader may disagree with some of the opinions expressed in it and may find the occasional lack of critical assessment of our knowledge of some conditions irksome. However, these are minor criticisms and in no way detract from the value of this very excellent addition to dental literature.

I would congratulate the authors on the thoroughness of their revision of this book and on the success with which they have incorporated much recent work. I have no hesitation in recommending it to advanced students of dentistry. J.H.J.

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Clinical applications were considered in the afternoon session. The report of Dr. Masheter (Parke, Davis & Co.) on clinical trials of transbuccal oxytocin in the induction of labour provided the main interest. Although the results confirm the relative efficacy of the method, his claim that it was also safe was challenged by some of the discussants. The other main papers dealt with the endocrine control of labour and with techniques for testing oxytocin sensitivity.

In addition to general medical libraries, departmental obstetric and physiological libraries will find this a useful reference book. C.R.W.

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THE author does not express surprise that this is the first work of its size on the topic of "habituation'. By definition, habituation eliminates notice of itself. It is the process by which stored information about earlier stimuli can modify responses to subsequent stimuli. The phenomen can be seen in animals—for example, hares show an inhibition of response to aircraft noise when they sit quite undisturbed beside a runway.

A fresh stimulus can abolish habituation. Anxiety arising in an experimental situation can result in an increase in magnitude of the response. Similarly, in the world of commerce, a new and distinctive wrapping on a product may abolish habituation and increase sales.

This short book was inspired by the author's studies of acclimatisation in the tropics. He reports some interesting experiments, but it is the reviewer's opinion that had a term such as "adaptation" been used for the phenomenon described it would have been more readily understood. The book will be of more interest to those in special fields, such as Physiology, Psychology and Pharmacology. J.G.G.

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BYSSINOSIS IN FLAX WORKERS IN NORTHERN IRELAND. A Report to the Ministry of Labour and National Insurance, the Government of Northern Ireland, from the Department of Social and Preventive Medicine, The Queen's University of Belfast. By G. C. R. Carey, P. C. Elwood, I. R. McAuley, J. D. Merreth and J. Pemberton. (Pp. 171; Illustrated, 15s). Belfast: Her Majesty's Stationery Office, 1965.

IT is a pleasure to have the opportunity of reviewing a Government Publication which is a report to the Minister of Labour and National Insurance, Northern Ireland, by the Department of Social and Preventive Medicine of Queen's University of Belfast. It is the story, clearly and succinctly narrated, of a scientific survey into an important industrial hazard by Professor Pemberton and members of his department.

The spinning and weaving of flax have been carried out in Ireland for many centuries and historically there is no evidence of any respiratory hazard until, with the industrial revolution, mechanization of the various processes took place. It is not surprising that local interest in the Monday syndrome and associated respiratory symptoms was in evidence by studies and observations by Malcolm in 1856. Purden from 1873 to 1877 and much more recently by Smiley (1951 and 1961) and Logan (1959).

The report indicates the origin of the Survey—broadly to enquire into the existence of an industrial disease not prescribed by the National Insurance (Industrial Injuries) Act (N.I.) 1946 resembling byssinosis of workers in the cotton industry. Definitions are made and the complex processing and spinning of flax of today are graphically and pictorially described. With the co-operation of some 23 mills, 3,052 workers aged 35 or over were examined using a questionnaire based on the M.R.C. questionnaire on Respiratory Symptoms (1960). Detailed questions were asked about the symptoms of byssinosis, smoking habits and previous chest illnesses and respiratory function tests were carried out. A 'dust survey' to measure the concentration of airborne dust in the various rooms in the mills. was mounted to determine association between dust levels and the prevalence of byssinosis. The findings in all these exercises are tabulated and discussed and there are some excellent X-rays. Finally, conclusions in relation to the five significant questions to which answers were sought by the Ministry are given in detail. The Monday syndrome is described and the general point is made that flax byssinosis is clinically indistinguishable from emphysema and bronchitis as they occur in the general population. History of the development of the condition from the Monday syndrome combined with the appropriate industrial history enables a diagnosis to be made.

Professor Pemberton and his colleagues are to be congratulated on the production of a very readable report which must be of interest to a wide range of doctors in our Province. Government appreciation of its scientific merit is indicated by legislation consequent on its recommendations not only for Northern Ireland but for Great Britain. C.W.K.

CALLING THE LABORATORY. Edited by William A. R. Thomson, M.D. Second Edition. (Pp. ix+126. 17s 6d). Edinburgh and London: E. & S. Livingstone, 1966.

THIS inexpensive handbook is of much greater value than its price would suggest. It is terse, concise and informed with admirable commonsense. For a busy general practitioner wishing to avail himself of laboratory services it provides not only a quick study, but simple explicit descriptions of essentials. It is also valuable to the trainee in laboratory medicine providing a wide ranging view of the hard practical core of his discipline. There would be few of the medical profession who would not find something of value within its covers.

W.S.

BYSSINOSIS IN FLAX WORKERS IN NORTHERN IRELAND. A Report to the Ministry of Labour and National Insurance, the Government of Northern Ireland, from the Department of Social and Preventive Medicine, The Queen's University of Belfast. By G. C. R. Carey, P. C. Elwood, I. R. McAuley, J. D. Merreth and J. Pemberton. (Pp. 171; Illustrated, 15s). Belfast: Her Majesty's Stationery Office, 1965.

IT is a pleasure to have the opportunity of reviewing a Government Publication which is a report to the Minister of Labour and National Insurance, Northern Ireland, by the Department of Social and Preventive Medicine of Queen's University of Belfast. It is the story, clearly and succinctly narrated, of a scientific survey into an important industrial hazard by Professor Pemberton and members of his department.

The spinning and weaving of flax have been carried out in Ireland for many centuries and historically there is no evidence of any respiratory hazard until, with the industrial revolution, mechanization of the various processes took place. It is not surprising that local interest in the Monday syndrome and associated respiratory symptoms was in evidence by studies and observations by Malcolm in 1856. Purden from 1873 to 1877 and much more recently by Smiley (1951 and 1961) and Logan (1959).

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PRICE'S TEXTBOOK OF THE PRACTICE OF MEDICINE. Edited by Sir Ronald Bodley Scott. Tenth Edition. (Pp. xiv+1259; figs. 34. 90s). London: Oxford University Press, 1966.

THOSE brought up as students on successive editions of Price's Medicine will welcome this new Volume after a lapse of nine years. Sir Ronald Bodley Scott, well known to this school and a new contributor to the Eighth Edition of 1950, has taken over the editorial chair with Dr. R. I. S. Bayliss, Dr. T. Lloyd Davies, Dr. J. D. P. Graham, Mr. R. F. MacNab Jones, Dr. F. O. MacCallum, Dr. G. D. W. McKendrick, Dr. R. W. Riddell, Professor R. A. Shooter and Dr. Denis Williams as new contributors.

Very major improvements are noted in this new edition. The rather stubby rice paper volume of the past has been replaced by a thoroughly up-to-date attractive book in improved print and paper with a two column format. This makes for increased clarity and more comfort in reading. Much of the text has been re-written bringing it up to date and references to major papers and texts are given at the end of certain sections. The introductory remarks regarding definition, aetiology, pathology and physiological derangement are clear and succinct, and treatment is given in detail where required with dosages in the metric system. The line drawings and E.C.G. illustrations are clear.

This is essentially a book of reference for the practitioner, consultant and more senior student. As such, even the rarest conditions receive mention in some detail. One finds it rather peculiar that the section on Connective Tissue Disorders is treated separately from the section on Disorders of the Locomotor System, leaving the latter with no introductory paragraphs. It would appear that these sections could usefully be combined, with a common introduction on connective tissue and remarks on clinical and other evidence of joint and connective tissue diseases. A list of normal values at the end of the book would be advantageous.

These are, however, but small criticisms of the lay-out rather than the substance of the book which is uniformly excellent and presented in a very readable and consistent style.

The new "Price" claims to follow its predecessors in presenting a survey of art and science in Comtemporary British Medicine and in this it has been highly successful. It compares very favourably with similar large texts from the New World and is excellent value at 90s. The Editor and Authors are to be congratulated on this new edition.

M.W.J.B.

HEALTH CENTRES AND GROUP PRACTICES. Articles collected from the British Medical Journal. (Pp. vii+64. 3s 6d). London: British Medical Association, 1966.

DURING 1965 Dr. Stephen Lock, a member of the staff of the British Medical Journal visited five group practices and five health centres in various parts of Great Britain and wrote a series of descriptive articles for the Journal. These articles have now been reprinted in book form, with an introduction by Lord Cohen.

The articles are clearly and concisely written, and illustrated by photographs and (in most cases) plans. They are of considerable interest to all who are concerned about medicine in the community.

What strikes one forcibly is the fact that today, nearly fifty years after the idea of Health Centres was put forward in the Dawson Report, there is still no clear agreement on the essential pattern of their design or organisation. There is clearly much need for operational research in this field.

These articles will be of value especially to the many family doctors who at the present time are looking to health centres and group practices for an answer to the intellectual isolation and administrative problems of smaller practice units. R.P.M.

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TEXTBOOK OF MEDICAL TREATMENT. Edited by Sir Derrick Dunlop, B.A.(Oxon.), M.D., F.R.C.P.Ed., F.R.C.P.Lond., and Stanley Alstead, C.B.E., M.D., F.R.C.P.Ed., F.R.C.P.Glasg., F.R.C.P.Lond. Tenth Edition. (Pp. xvii +1003; figs. 36. 170s). Edinburgh and London: E. & S. Livingstone, 1966.

THE latest edition of this very valuable book contains much up-to-date information. But although it has been kept rigidly to size it is still a heavy book and I doubt whether many students read it from cover to cover before they qualify. By the time they have qualified a lot of the details of treatment will be out of date (indeed in some instances this is already true). This raises the problem as to whether this book is meant to be a textbook for students or a reference book for qualified doctors. It has shortcoming in both respects :

(a) *As a textbook for students.* Apart from being too long and detailed for the undergraduate the book is arranged in an extraordinarily haphazard way. Sections on general principles of treatment (the care of the unconscious patient, the care of old people, the relief of pain, principles of prescribing, disturbances in water and electrolyte balance etc.) are to be found scattered through the book, sandwiched between sections on the treatment of specific diseases. For instance, the use of antibiotics in certain specific diseases is described before the section on the general principles of antibiotic treatment.

(b) *As a reference book for postgraduates.* Here the arrangement of the book is less important. It has an excellent index, a glossary of proprietary names for drugs and a well laid out table of contents. The major shortcoming as a reference book is the almost comp'ete lack of references to the medical literature. As is befitting a textbook for undergraduates many of the sections are beautifully written but express dogmatic opinions as to the correct and the incorrect methods of treatment. Some of these opinions are based on well established scientific knowledge but others ar not. Some of these latter are the accepted opinion of the 'medical establishment' but others are opinions held only by the author. There is no way of distinguishing these three on reading the text.

For the practising doctor, as opposed to the postgraduate student, this book will be used for reference for the treatment of uncommon diseases, especially at the fringes between medicine and such specialities as dermatology and otorhinolaryngology. This provides adequate justification for the inclusion of these special subjects.

This book has been found very useful in the past because there has been no convenient equivalent. It may need mor drastic revision and some alteration to maintain its pre-eminence. P.C.E.

CLINICAL PHARMACOLOGY. By D. R. Laurence, M.D., F.R.C.P. Third Edition. (Pp. x+678; figs. 22. Paper 32s 6d, Cloth 45s). London: Churchill, 1966.

THIS textbook was first conceived seven years ago. It is intended for medical students in their clinical years, it provides the kind of information they need, it is interesting and at times entertaining and it gives references for further reading. The present edition is more than 100 pages longer than th last edition, but it is still an easy book to read, although I think it could with advantage have been kept to its original length.

How sensible that this book is now published in paper back form. It shows an appreciation that textbooks on a subject like modern pharmacology are ephemera : no sooner than they have been used by the current generation of students than they become useless and should be destroyed before they misguide the next generation of students.

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PSYCHIATRIC HOSPITAL CARE. A Symposium. Edited by Hugh Freeman. (Pp. 340. 50s). London: Ballière, Tindall & Cassell, 1965.

DURING the past decade increasing emphasis has been placed on the advantages of treating many mentally ill patients outside the walls of the psychiatric hospital and not isolating them from the community in which they became ill. This attitude has been reflected in the establishment of day hospitals and psychiatric units in general hospitals and in the expansion of out-patient facilities. It has been anticipated that as a result of these trends the existing psychiatric hospitals may have to fulfil a different kind of function than formerly and in the future concentrate their efforts on the care of the chronic psychiatric invalid and psychogeriatric patient.

In view of current interest in the re-shaping of psychiatric services it is not surprising that there has been an increasing number of publications relevant to this.

Here is the report of a symposium held at the Middlesex Hospital in January 1964 at which a group of clinical psychiatrists and others connected in different ways with mental health services met to work out proposals for the future organization of psychiatric hospital care. The book consists of a series of original papers each of which is followed by the edited report of a discussion. The problems of mental hospitals, psychiatric units in general hospitals, rehabilitation, public education, training of psychiatric social workers, the elderly, local authority services and psychiatric nursing are all considered.

A presentaton of this kind seldom makes for consistently absorbing and stimulating reading but, nevertheless, the contributiors have much to offer from their own experience which will be of value to all those interested in the setting up of services in their respective areas. It has the very creditable merit that the contributors are actively engaged in the planning of a comprehensive service and have an awareness of the inherent problems through direct involvement.

Of particular interest and relevance are the chapters by Professor Peter Townsend, Professor W. Ferguson Anderson and Dr. Russell Barton dealing with requirements of the elderly. It is salutary to note that there are far more bedfast old persons living in their own homes in Britain than in all institutions put together and more than three times as many who are severely incapacitated, including the bedfast. As was pointed out in discussion it will take only a very small drop in community tolerance for institutional resources to be completely swamped.

For those interested in the development of pyschiatric services this report makes informative reading. For the less progressive it may prove therapeutic. S.J.K.

A GUIDE TO CARDIOLOGY. By J. C. Leonard, M.D.(Lond.), M.R.C.P. (Lond.), and E. G. Galea, M.B.(Queensland), M.R.C.P.(Lond.), M.R.A.C.P. (Pp. xii+306; figs. 88. 35s). Edinburgh and London: E. & S. Livingstone, 1966.

THE authors state that this book was written primarily for the senior medical student, house physician and general practioner and, as such, is an excellent introduction to cardiology. However, the content and presentation of the book is such that it would also be of value to the post graduate trainee in the field of internal medicine.

Like all texts of this size some dogmatic statements are made which are certainly not widely held by cardiologists, but this defect is fairly well compensated for by a few well chosen references at the end of each chapter.

The text is well balanced, all sections of cardiology have been given the space they warrant and even the more obtruse investigative techniques are briefly described. One is certainly pleased to see that such a topical subject as cardiac arrest and its emergency treatment is dealt with in some detail.

In conclusion, I commend this excellent and inexpensive little book to medical students, general practitioners and general physicians interested in broadening their knowledge of cardiology. S.S.A.F.

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DETERMINANTS OF INFANT BEHAVIOUR III. Edited by B. M. Foss. (Pp. xiii+264; Plates and figures. 50s). London: Methuen, 1965.

THIS book, edited by Professor B. M. Foss, contains the published proceedings of the Third Tavistock Study Group on "Mother and Infand Interaction", held in London in September 1963. The ten separate reports, each of which is followed by discussion, consisting of the authors and invited guests. These reports are grouped into two main sections, one dealing with animal studies and the other one dealing with human studies. It contains the reports of an international group of psychologists, zoologists and psychoanalysts and others working with infants.

As Dr. Bowlby says in his Foreword, the meetings have been convened in the belief that an understanding of mother-infant interaction in humans is of paramount importance in understanding the psychological development of the child and thereby enlarging our knowledge of abnormal reactions in both children and adults. One cannot help feeling that this belief is somewhat narrow as far as the most recent thinking is concerned and one would like to see studies done, not so much on the mother-infant reaction alone as one in which is shown the reactive situations of the family group, especially that of the father.

In Dr. Rosenblatt's paper on "The Basis of Synchrony in the Behavioral Interaction between the Mother and her Offspring in the Laboratory Rat" lays great emphasis on the hormonal basis of behaviour and in particular tries to link behavioral interaction between mother and infant in terms of lactation and other hormonal factors. The induction and maintenance of maternal behaviour by injecting prolactin is shown to be partly successful, especially in the field of maintenance. However, the difficulty, as always in animal studies which arises in attempting to equate these studies with the human, is again very obvious and one is constantly reminded in reading the paper that in fact many social conditions are introduced at the same time as the physical ones. In other words, that mothering has very cogent social references. I feel that this last point is underlined in a later paper in which there is a study made of "Rhesus Monkey Aunts" in which maternal behaviour in these rhesus aunts is described in detail and it is no doubt present.

Prefessor Hinde's paper in this later section introduces the very interesting findings that these aunts, that is, female rhesus monkeys who have not been pregnant can actually be demonstrated to secrete milk when they take on the mothering of an infant who is removed from his mother.

The book moves on in a series of papers to study the interactions in humans and it would appear that there is little resemblance between that of humans and animals in these particular fields investigated.

In some of the discussions which followed these the role of the father is mentioned and this appears to be a factor which is left out of all the experiments, much to the detriment of the entire proceedings. One would also like to see studies which included not only the father but other emotionally relevant people in the child's background—that that would obviously make the study more complicated is not sufficient excuse for leaving it out.

A preliminary report on the Gewirtz's study obviously raises more questions than it answers but in its favour it can be said that it attempts to look at four different methods of child-rearing in a Jewish community and in so doing holds out the promise that interesting data and observation might be obtained which would be of interest in the mother-infant reaction. The account given is a very preliminary one and following this paper the discussion, of which there are many throughout the book, for the first time to me appeared to become alive. This stimulated the discussors to look into the scientific method itself and this I found most rewarding, that is, whether one should collect data first by direct observation and from this evolve a theory or whether the testing of an hypothesis by data collected is the method of choice. Also in this section the use of computers in research gets an airing though one could not say that it is sufficiently good to be of any real value to the reader.

This is a small and expensive book of only 260 pages and is more for the advanced student and presupposes a good deal of knowledge of the subject. Also I feel it is important that the potential reader should realise that these papers consist of preliminary reports only and are indications of work in hand. One can, therefore, not expect very much in the way of hard facts. It does, however, indicate the more sophisticated approach to the

problems of child psychology and psychiatry which is being investigated to-day. It would appear to be an important book to have for reference. Incidentally, there are some twelve pages of references given. W.F.McA.

NOTES ON RADIOLOGICAL EMERGENCIES. By George Ansell. (Cardboard folder. 10s). Edinburgh and London: E. & S. Livingstone, 1966.

MODERN techniques have made it possible to save the lives of many patients whose vital functions cease because of some temporary crisis. Cardiac massage, defibrillation, pacemaking, mouth to mouth ventilation and the understanding of the gross biochemical disturbances which result from a temporary stoppage of the heart beat or of respiration have made this possible. Because of this it is no longer ethical for any member of the hospital staff to stand back and allow the victim of such an emergency to die in peace. The vital factor is speed, not only may a few seconds delay in the application of the correct measure mean the difference between life and death, but it may also mean the difference between a normal survivor and one who is a permanently comatose vegetable.

All hospital staff should have a certain minimal training as to what to do in these emergencies, but they cannot be expected to carry all the detailed information needed in their heads. This detail must be available at obvious points throughout the hospital and with it must be kept certain items of equipment and drugs and directions as to how to get expert assistance and such complex equipment as a defibrillator.

Dr. Ansell has done an excellent job of producing a pamphlet containing the information that is necessary for dealing with emergencies that might arise in a diagnostic X-ray department. Not only is this pamphlet designed for quick reference, but it can be hung on the wall like a calendar and is shocking pink in colour. With a few minor amendments of drug names, etc., it should hang in a prominent place in every X-ray department and beside it should hang the emergency arrangements for cardiac and respiratory resuscitation for that hospital.

Similar pamphlets should be designed to hang in other hospital departments, wards and laboratories. P.C.E.

THE FAMILY AND THE SEXUAL REVOLUTION. Edited by Edwin M. Schur. (Pp. xv+427. 40s.). London: George Allen-Unwin, 1966.

WHILE most sociologists would discredit the view that society is disintegrating because of a sharp decline in sexual morals and a breakdown of the family system there is no doubt that these are passing through a period of profound change and that there is much need for both factual information and informed discussion. These extracts from various sources range from Kinsey and Bertrand Russell to Pope Pius XI and are mainly by American sociologists and psychiatrists, and by professors and chairmen of departments such as child development, family life and education, and by anthropologists, lawyers and marriage guidance experts.

The book is divided into three parts. The first is concerned with facts by Kinsey and discussion by others on changing sex standards. The second is titled the "Women problem" and deals largely with the place of women in society and the limitations placed on her by society. The third deals with birth control and is, at least for the medical reader, of limited interest. It includes a reprint of the Papal Encyclical on Christian marriage.

This book should provide material for informed and responsible discussion and fairly represents a wide range of viewpoints.

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NOTES ON RADIOLOGICAL EMERGENCIES. By George Ansell. (Cardboard folder. 10s). Edinburgh and London: E. & S. Livingstone, 1966.

MODERN techniques have made it possible to save the lives of many patients whose vital functions cease because of some temporary crisis. Cardiac massage, defibrillation, pacemaking, mouth to mouth ventilation and the understanding of the gross biochemical disturbances which result from a temporary stoppage of the heart beat or of respiration have made this possible. Because of this it is no longer ethical for any member of the hospital staff to stand back and allow the victim of such an emergency to die in peace. The vital factor is speed, not only may a few seconds delay in the application of the correct measure mean the difference between life and death, but it may also mean the difference between a normal survivor and one who is a permanently comatose vegetable.

All hospital staff should have a certain minimal training as to what to do in these emergencies, but they cannot be expected to carry all the detailed information needed in their heads. This detail must be available at obvious points throughout the hospital and with it must be kept certain items of equipment and drugs and directions as to how to get expert assistance and such complex equipment as a defibrillator.

Dr. Ansell has done an excellent job of producing a pamphlet containing the information that is necessary for dealing with emergencies that might arise in a diagnostic X-ray department. Not only is this pamphlet designed for quick reference, but it can be hung on the wall like a calendar and is shocking pink in colour. With a few minor amendments of drug names, etc., it should hang in a prominent place in every X-ray department and beside it should hang the emergency arrangements for cardiac and respiratory resuscitation for that hospital.

Similar pamphlets should be designed to hang in other hospital departments, wards and laboratories. P.C.E.

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REPRODUCTIVE PHYSIOLOGY OF THE POST-PARTUM PERIOD. By Albert Sharman, M.D., D.Sc., Ph.D., F.R.C.O.G., F.R.C.S. (Glasg.). (Pp vii + 127; figs. 87. 25s.). Edinburgh and London: E. & S. Livingstone, 1966.

THE title of this monograph is too broad for its limited though valuable approach. The approach is that of morbid anatomy and clinical observation and the term "physiology" is justified only since it is an attempt to define the normal changes of the post-partum period. Essentially these studies are limited to a histological study of the endometrium from soon after delivery till nine months thereafter; the re-establishment of ovulation as indicated by the appearance of the secretory phase of the endometrial cycle and the date of re-appearance of menstruation and its subsequent rhythm.

The basis of the work is 626 endometrial biopsies from 285 women from the fifth day to nine months after parturition and all free from puerperal infection. Of these women 230 had one biopsy only. The remainder had from 2 to 24 biopsies and in these the normality of the later biopsies might be questioned. However the author has an extensive series of biopsies, which he seems to have carefully correlated, and which enables him to describe regeneration of the endometrium following childbirth. Cellular reaction as indicated by the presence of plasma cells is described as late as the third and fourth months in over one-third of the cases. The earliest appearance of secretory endometrial patterns, taken as presumptive evidence of ovulation, was noted during the seventh week.

This is a useful study presented in detail and with an over abundance of microphotographs, many of which show little distinctive change. It would seem that the work in a shortened but unimpaired form might have graced a learned journal. Elegantly presented and beautifully printed and bound as, indeed, one expects from the publishers, it is moderately priced and should be included in the library of all interested departments. J.E.M.

NOTICE

This is volume 35 of the *Ulster Medical Journal*, and the usual two parts are combined. It is the only issue dated for the year 1966. In future, for reasons relating to the dates of delivery of Presidential and other addresses and to difficulties in printing before Christmas, the Journal will be published in late February and late September and the two numbers will be dated as Winter and Summer.

It is regretted that the last issue dated December 1965 was incorrectly described as volume XXXV, No. 2. It was volume XXXIV, No. 2. The volume number on the contents page of the last volume was also incorrectly given.