APRIL, 1940

THE ULSTER MEDICAL JOURNAL

PUBLISHED BY
THE ULSTER MEDICAL SOCIETY

The Ulster Medical Journal

VOL. IX

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CONTENTS

Fellows and Members of the Ulster Medical Society receive the Journal free.

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:: PUBLISHED ON BEHALF OF THE ULSTER MEDICAL SOCIETY ::

Vol. IX 1st APRIL, 1940 No. 1

ULSTER MEDICAL SOCIETY

ACTIVITIES PARTIALLY RESUMED

THE Council of the Ulster Medical Society has decided to resume partially the Society's activities. The Journal is to appear twice in the year if exigencies of war will permit, one issue in April and one issue in October.

A successful clinical meeting has been held at the Royal Victoria Hospital, and other meetings are in contemplation. The following list of exhibits shows the interest taken in the clinical meeting, which was held on Thursday, 22nd February.

1. MR. W. A. ANDERSON.
   Case of otitic hydrocephalus.
2. MR. W. W. BASSETT.
   The Blackburn skull traction appliance in operation. Radiograms.
3. DR. R. M. BEATH AND DR. F. P. MONTGOMERY.
   Radiograms.
4. DR. B. R. CLARKE AND DR. S. L. W. ERSKINE.
   (a) Radiograms of healed cavities after the termination of artificial pneumothorax.
   (b) Radiograms of "early" tuberculous lesions.
5. DR. F. COATES.
   (a) Case of pituitary tumour.
   (b) Case of old standing pericarditis now suffering from spinal caries and paraplegia.
6. MR. J. A. CORKEY.
   Case of arachodoctyly with ectopia lentis.
7. PROF. P. T. CRYMBLE.
   (a) Two cases of gastric carcinoma.
   (b) Three cases of lesser curve ulcer.
   (c) Three cases of gall-stones.
   (d) Three cases of vesical calculi.

8. Dr. E. Hickey.

Case of sclerodermia.

9. Mr. K. Hunter.

Demonstration on use of audiometer.

10. Mr. S. T. Irwin.

(1) Case of fractured neck of femur in a man of 88 years. Result of operation.
(2) Patient with intertrochanteric osteotomy for T.B. hip.
(3) Case of bilateral genu valgum treated by fracture and osteotomy.
(4) Case of fracture through greater trochanter treated by Smith-Petersen nail.
(5) X-rays of slipped epiphysis in a father with Perthe's disease in his son.

11. Dr. I. H. McCaw and Dr. R. Hall.

Some interesting skin conditions.

12. Mr. H. P. Malcolm.

(a) Case of pes equino-cavus.
(b) Case of fracture of neck of femur nailed.
(c) Case of enlarged prostate enucleated under local anæsthesia.
(d) Case of tic douloureux. Injection of gasserian ganglion with .1 per cent. percain.

13. Mr. G. R. B. Purce.

Results of thoracoplasty in pulmonary tuberculosis.

14. Mr. J. R. Wheeler.

Case of orbital tumour (removed).

15. Mr. J. Withers.

Case of fractured femur in a child complicated by femoral artery thrombosis.

Peripheral embolectomy.

16. Mr. C. J. A. Woodside.

(1) Lumbar sympathectomy for thromboangiitis obliterans.
(2) The Bernard Fey incision for renal sympathectomy.
(3) With Mr. Riordan. Adamantinoma of the mandible.

After the meeting the President, Mr. T. S. Holmes, M.B., M.Ch., F.R.C.S.Eng., F.C.O.G., invited the members of the Society to the Grand Central Hotel for lunch. This was one of the pleasantest functions which the Society has enjoyed for some time, the arrangements for which were made by the efficient secretary, Dr. H. Hilton Stewart. Dr. John Rankin expressed the thanks of the Society in a short and witty speech.

Frederick Street

By the late Sir Robert J. Johnston, B.A., M.B., F.R.C.S.ENG.

Reprinted from the Royal Victoria Hospital Magazine, Vol. 1, No. 1
(January 1st, 1931)

It is always salutary and often inspiring to look back on our origins, and to a generation which has been brought up in the Royal Victoria Hospital, one of the most modern and best-equipped hospitals in the world, it may be of interest to learn something of what the "old Royal" was like.

The building still stands, but greatly altered in appearance. Those who, like myself, first approached it in the nineties crossed an empty and windswept court-yard and ascended the steps common to staff and students alike to sign our names in the entrance hall. There was not then a students' union, and the hospital steps in summer, like the dissecting-room fire in winter, served for our club and smoking room. Many an hour was spent there bandying fierce argument or swopping the merry jest, while operations went on unheeded and clinical lectures only reached the ears of the faithful few. For that our system of hospital attendance was in part to blame. Except for a term as dresser in the extern, a term as clinical clerk, and a term as surgical dresser, the powers that were left us to our own devices, and once a student had signed the book he was free to go anywhere he liked in the hospital, or outside it if his occasions so required. Indeed, when we attended in our first hospital year, the second year of medical studies, we were recommended by our teachers to sign the book and get off as soon as possible to the dissecting-room, there to perfect our knowledge of anatomy. Few of us dared to flout this good advice, so far at least as the getaway from hospital was concerned.

Our first real acquaintance with the interior of the old Royal began when we took out our three months' dressing in the extern, and no one who had anything to do with the extern in those days could fail to retain a vivid impression of Lizzie Hanna. Lizzie was great, physically, mentally, and temperamentally. She was a female Falstaff with less humour, as much character, and infinitely more dignity. She sat in the front centre of the waiting hall, beside a bandage-winder, and from that point she ran the whole extern—visiting staff, house surgeon, students, nurses, patients, and all. What would have happened had anyone disputed her authority will never be known—no one ever dared.

From the extern, where Professor Sinclair, who was then assistant surgeon, gave us an occasional taste of his quality as a clinical lecturer, we passed to the wards as surgical dressers. The senior surgeon was John Walton Browne, commonly called "Jack." His confidence, his omniscience, and his side-whiskers were the envy and the exemplar of successive generations of house-surgeons. What was not so easy to copy was his dexterity. I have seen many surgeons operate, but I have never seen one who was more a man of his hands. He was the despair of his assistants, for during an operation his scalpel was as often between his teeth as in his fingers, and he would polish his glasses with his handkerchief and

continue as though nothing had broken the chain of asepsis. As his beautifully dissected wounds had a way of healing by first intention while those made by more scrupulous surgeons not seldom suppurated, he was really a most demoralising influence.

The "hospital alphabet" of our day thus described Surgeon Fagan :—

F is for Fagan, renowned in his art,
Whose motto is "Volkmann" and rest for the part.

He certainly looked the surgeon, bold and resourceful. "No one," he once told us, "can consider himself a surgeon unless he is prepared to tackle a major operation on a bare hillside with only a pocket-knife and a piece of whipcord." Both he and "Jack," who were great friends, were rather irascible while operating, especially if things were not going well. "Jack" usually threw two or three scalpels across the theatre before one was brought to please him, and Fagan, when he was being badly assisted, addressed a full theatre with the words, "I must have committed the unpardonable sin in my youth to be cursed with a house-surgeon like this in my age." It was proof of his progressive spirit that, though barely on nodding terms with asepsis, he was quite eager to practise it.

Henry O'Neill was, however, the figure around which legends of the old Royal cluster. Corporator, public health zealot, humorist, general practitioner, and surgeon, he was always in the limelight. Were he living to-day he would certainly be in the "talkies." He was known commonly as "Health, Belfast," the name of a journal devoted to public health which he founded, edited, and financed, bringing out a series, as he told us, whenever he had saved £1,000, and running it till the money was done. His pet subjects for clinical lectures were talipes and venereal disease, and while he descanted on the pangs of gonorrhœa and the ravages of syphilis even to the second generation and gave us awful examples in illustration, many a heart was touched with anguish. But when he had finished his tour of the ward, armed with Thomas's wrench, the tears in the eyes of the patients showed that as a cause of suffering Vulcan's club-foot could at any rate run a good second to the charms of Venus. He had the pawkiest of wit and a Rabelaisian humour. Of the many stories told of him, here is one. After going over with his class all the usual tests for sugar, he asked : "But suppose you had no chemicals handy, how would you find out if there was sugar in the urine?" He gave the answer himself, "By the sweet taste. Nurse, bring me that specimen glass. Now, gentlemen," he continued, "do as I do, put a finger in the urine and lick your finger." He suited the action to the word, and one by one the class reluctantly followed suit. When the last test had been made Henry quietly said, "Gentlemen, ye haven't much observation or ye would have noticed that the finger I put into the urine was not the one I licked." As the alphabet said :—

H is for Henry, the friend of his race,
An Israelite guileless, as seen by his face.

There was only one theatre for all the surgeons. It had a fine gallery which gave ample accommodation for all the students who wished to see operations, but

it was as well that these operations were very few in number from our point of view, for even then it was no uncommon thing to see one of the surgeons fuming and fretting in the corridor and perhaps going off in disgust while a colleague was taking full advantage of his prior lien on the theatre.

About the end of the second hospital year the student bought a binaural and nerved himself to ascend the staircase to middle and top lobbies, where the physicians stood prepared to initiate the neophyte into their mysteries. Again, as at his first approach to hospital, he met an imposing female figure. Nurse Hanna dominated the extern by sheer force of personality, but "Ma" Henry, who was as angular as the other was rotund, ruled the patients in middle lobby by the gift of the tongue. Woe betide the unfortunate man who did not bring in his nightshirt with him—and striped flannel was the only wear. The hospital alphabet, which spared few, is silent about Mrs. Henry—its author was the house physician and he lived on middle lobby. But a patient, leaving the ward, thus delivered himself to his physician: "Doctor, when I came in I thought that woman was a witch, but now I know that she's a bitch." For all that, many a sick H.P. blessed her taste in medical comforts and in invalid cookery.

In the medical wards we encountered Professor Cuming, the high priest of philosophic doubt, who had long since lost any faith he may have ever had in any form of therapeutics, medical or surgical, and who seemed to question whether diagnosis was possible, and if possible whether it was convenient. Whitla, on the other hand, was absolute, almost infallible, epigrammatic, paradoxical, sometimes oracular, so the earnest student of clinical medicine usually found his way to the bedside where "Jimmy" Lindsay taught his students how to use their eyes, their ears, and their fingers, and encouraged them to venture on deductions from their observations. In a perfectly ordered world, Lindsay would have been set down to a whole-time job as teacher of clinical medicine.

L is for Lindsay, our prize auscultator,
At spotting a murmur there's no one that's "nater."

Top lobby held the female patients, and in it was the four-bed ward which represented the provision for gynæcology. Opposite that was the theatre, but if one had been allowed to make a P.V. on a patient under an anæsthetic and to witness an operation, he was considered to have received a Benjamin's portion of clinical gynæcology.

About once each term we were led across the yard by one of the physicians to visit the fever block. We were shown the chart and gazed from a respectful distance at a case of scarlet or dip. or typhoid, but neither our teacher nor ourselves ventured on too intimate a contact. Still we all got our certificates for attendance on fevers, and we lodged records of twenty cases as required by the regulations.

Space forbids me to carry these random recollections further, but I have always thought that Professor Sinclair summed up the old Royal very well just before we left it for ever: "A hopelessly out-of-date hospital, but there has been a great deal of first-class work done in it."

Colostomy

By ERIC W. McMECHAN, M.B., F.R.C.S.ENG.

Assisting Visiting Surgeon, Belfast Union Infirmary

HISTORICAL.

IN pre-Listerian days surgeons were afraid, and rightly so, to open the peritoneal cavity. The antiseptic methods of the post-Listerian era, and the asceptic technique of the present time, were then unheard of, yet there was no lacking in the anatomical knowledge of the great men of that period, who must be hailed as the founders of the surgical profession. Their vivid anatomical descriptions will stand as monuments to their memory and an inspiration to those who are attempting to follow in their footsteps. It is to Petit that we are indebted for the term colostomy. He suggested in 1886 that its application be limited to permanent artificial anus, and that the older term colotomy be reserved for the temporary variety. In 1710 Littré in "The Memoirs of the Academy of Sciences of Paris" suggested the operation of colostomy for imperforate anus, but there is no record of his having performed it. He advised using the sigmoid flexure via the peritoneal route. In 1776 Pilloré Rouén made a cæcostomy opening for intestinal obstruction in an adult. Then in the next twenty years Duboisé, Dinet, and Désault all performed the operation for imperforate anus. In 1787 Firé did a transverse colostomy during an operation in which he intended opening the small bowel for obstruction. In 1796 Callisen suggested that the descending colon should be reached extraperitoneally by a lumbar incision; this was soon abandoned, however, on account of the difficulty and the mutilation of tissue which was required in order to find the bowel. In addition, the infection that followed this operation was just as fatal as the peritonitis which resulted from the peritoneal route. So we find for the next thirty years the inguinal incision was favoured, but the lumbar operation was again revived by Amussat, who, in a series of articles (1839-41), showed that it could be done without any considerable danger, and in support of this view he records six cases, five of which were successful. In 1849 colostomy was first done for vescio-rectal fistula secondary to ulcerative colitis. Allingham, Reeves, and Cripps were eventually responsible for the abandonment of lumbar colostomy. They showed that it was impossible to do the operation without injuring the peritoneum, and also that the position was a very unsuitable one in which to have a bowel opening. In 1895 F. T. Paul, surgeon to the Royal Infirmary, Liverpool, in an article in the "British Medical Journal" of that year, describes the operation which still bears his name. He was the first to suture the two limbs of the bowel together in cases in which the colostomy was intended as a temporary measure.

Colostomy has never been a popular operation, and a great deal of abhorrence has been attached to it, partly because the patient is able to view the artificial anus, and partly on account of the prejudice which has been handed down from the early days of the operation, when not only the layman but also the physician objected

strongly on religious and sentimental grounds. Even surgical textbooks such as Gross's "System of Surgery" and Smith's "Principles and Practices of Surgery" have aided in this prejudice against the operation. Gross apologised for giving a description of the operation, stating that it was founded on "misdirected sympathy" and that it "ought to be discarded among the obsolete devices of surgery." The expression of such a view by one who was responsible for the teaching of students was most unfortunate. Since the days of Allingham and Paul much has been achieved regarding the technique of the operation, but Allingham's actual incision still stands, and Paul's operation is to-day regarded as a classic. Various surgeons have added to the general knowledge, such as Maydl, Witzel, Paul, and in more recent times, Miles, Lockhart-Mummery, and Gabriel, to mention only a few. It would be a serious omission if I failed to mention St. Mark's Hospital, which has done so much for the advancement of rectal surgery. I propose, therefore, merely to record the technique and after-care of colostomy as it was carried out during the time I was resident at St. Mark's.

Indications for Colostomy.

Carcinoma of the rectum.—All cases of carcinoma of the rectum which are operable will require colostomy, except possibly in some very early cases, where one might perform a conservative resection, either of the local or abdomino-anal type, as described by Lloyd-Davies. The question of whether or not it is worth while doing a colostomy in an inoperable carcinoma of the rectum is a much more difficult problem. One does not want to prolong an existence which has long since become intolerable, unless one can be sure that there is going to be an improvement in both the general and local conditions. Many points will have to be considered, for example, (1) the mental reaction of the patient to the operation, (2) the amount of pain, and whether it is due to involvement of the anal canal, or to extra rectal spread, (3) the severity of tenesmus, diarrhœa, and hæmorrhage, and (4) the possibility of the development of an acute obstruction. The improvement will depend on whether the tumour is of the ulcrative, schirrous, or colloid type; whether it is grade 1, 2, 3, or 4; and whether it is an upper, middle, or lower third growth. Generally speaking, the operation is worth while if there are no liver or peritoneal secondaries. The greatest improvement will be experienced by those cases with subacute obstruction, severe tenesmus, bleeding, diarrhœa, and incontinence, and in cases complaining of pain due to involvement of the anal canal. When pain is due to extra rectal spread there will be no improvement, and in cases of inoperable grade 4 carcinoma, the outlook of life is too short to warrant a colostomy. After the operation in suitable cases, there is an improvement in the general and often in the local condition, due to the now less frequent bowel evacuations. In addition, the appetite is regained, sleep improves, and the tenesmus becomes much less troublesome. This improvement will be maintained right up to a short time before death. The expectation of life is also definitely increased, as shown by Gabriel and Lloyd-Davies in a paper which surveyed the results of five hundred cases of palliative colostomy for carcinoma of the rectum. In the series they found

just over fifty per cent. of the cases, which were without liver and peritoneal secondaries at operation, died within the year, thirty per cent. survived for between one and two years, ten per cent. reached the two to three year period, and six per cent. survived for three years or more, so that providing the operation is done while the patient is still capable of improvement, it is definitely beneficial both as regards comfort and duration of life.

Other indications are much less frequent, for example, recto-vesical fistula which may be due to a carcinoma or diverticulitis, radium burns and their sequelæ, simple stricture of the rectum. Also, patients suffering from large recto-vaginal fistulæ or incontinence will be much more comfortable with colostomy. Lastly, the classical indication is in the newborn, as a temporary or permanent measure, for imperforate anus.

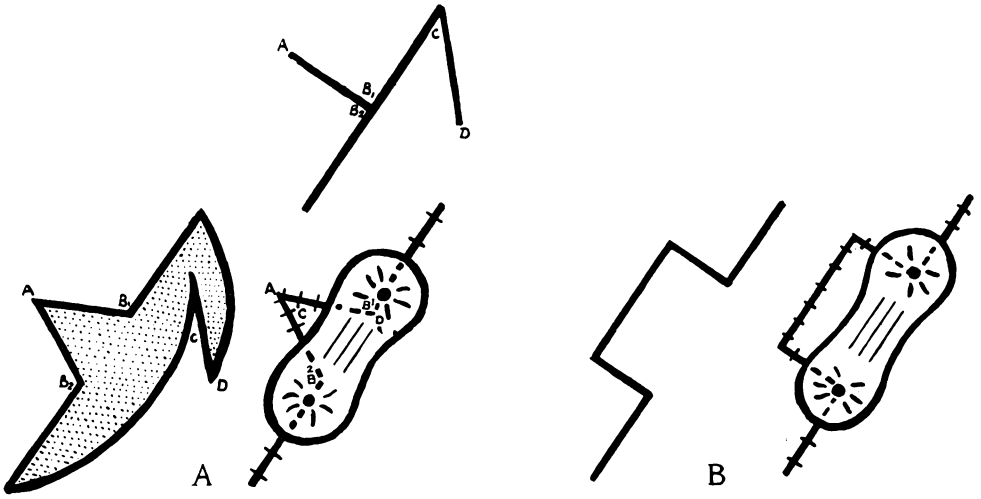

Fig. 1.—Showing variations in the skin incision for the formation of a skin bridge.

Technique of the Operation.

Anæsthetic.—Spinal anæsthesia gives very good relaxation, and permits of a full exploratory laparotomy being done. There are some cases, however, in which its use is contraindicated, for example, in patients with low blood-pressure there is increased risk of thrombotic complications from further lowering of the blood-pressure. In normal patients this fall can be effectively counteracted by the use of ephedrine. For a successful abdominal operation under spinal anæsthesia it is advisable either to employ full pre-operative medication, such as, by the use of omnopon gr. 2/3, scopolamine gr. 1/150, hypodermically, and nembutal gr. 1 to 3 by mouth about an hour before operation, or to combine the spinal anæsthesia with very light gas and oxygen or Evipan anæsthesia.

Incision.—The transrectus incision was first popularised on the mistaken idea that with the muscle-fibres of the rectus abdominus completely surrounding the

bowel, a certain amount of control would be acquired. There is no doubt that it is the most easy colostomy for which to obtain an accurately fitting belt, but on the other hand a transrectus opening is very prone to hernia formation. The most popular incision is an inguinal one, in reality a left-sided muscle-splitting incision. It is more difficult to fit a belt in this situation, but there is less likelihood of the subsequent development of a hernia. Variations in the skin incision have been advocated by different surgeons with a view to the formation of a skin bridge for the colostomy, two of which are illustrated.

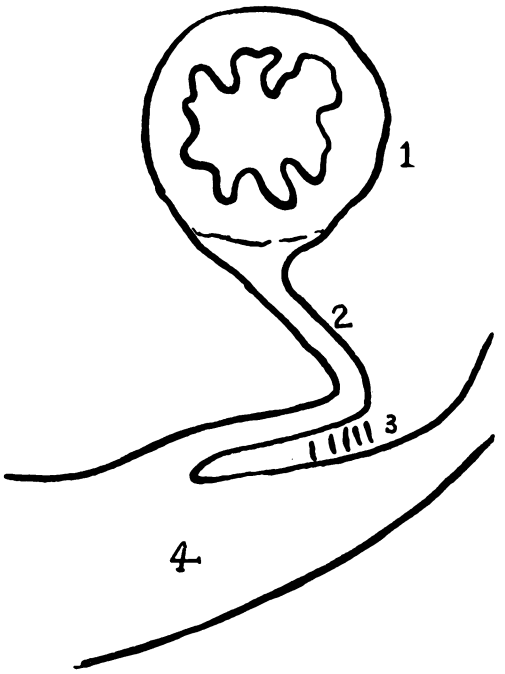

Fig. 2.—To show the adhesions between the mesosigmoid and the posterior abdominal wall, which tend to shorten the mesentry. (1) Sigmoid, (2) mesosigmoid, (3) adhesions, (4) posterior abdominal wall.

The various other so-called valvular colostomies may be mentioned merely to condemn them, as they are such in name only. Thus the oblique variety may retain its valvular action for a few weeks, but with the constant usage the track eventually straightens out. The size of the skin incision does not matter provided it gives sufficient exposure, but the opening through the muscles and peritoneum should be as small as is practicable. The incision should be muscle-splitting, injury to nerves and forcible retraction of the muscles should be avoided as far as is possible. If exploration of the abdomen is indicated, as in cases of carcinoma of the rectum, then a second incision should be made. A complete exploratory laparotomy cannot be done through a left iliac incision, and a paramedian incision does not add to the dangers

of the operation. It is a definite drawback to have to make a colostomy through a large incision, because of the tendency to subsequent prolapse and herniation, and also because there is necessarily an increased risk of infection in a large wound. In the case of a blind colostomy, a finger is passed through the incision and, keeping close to the left lateral wall of the abdomen, it is then passed backwards until it comes into contact with the bowel: this will be the sigmoid colon. The latter is then brought out through the abdominal incision, making certain that the bowel is not twisted by tracing both limbs of the loop backwards. If the mesentery of the colon is short it may be lengthened by dividing peritoneal adhesions on the outer side of the bowel, as there is very often a fold in the peritoneum in this position,

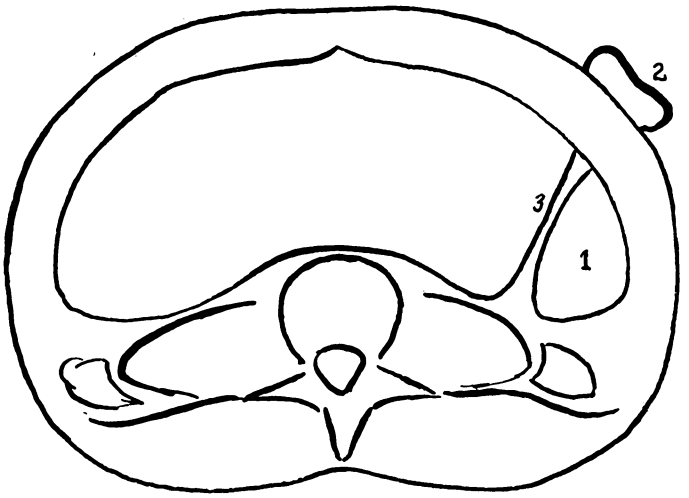

Fig. 3.—To show the posterior space described by Gabriel, and through which small intestines may become strangulated. The space may be easily closed by a purse-string suture. (1) Lateral space, (2) colostomy, (3) mesosigmoid.

as shown in diagram 2. If the mesentery is still too short, one may remove wedges of fat from the subcutaneous part of the abdominal wall. The bowel is then pulled taut on the upper limb; this allows easy passage of a catheter subsequently, and also tends to prevent prolapse or herniation. It is also an advantage to have all the free bowel below, especially if an excision of the rectum is to be performed later. The space lateral to the meso-sigmoid is then closed by putting a catgut suture through the free border of the ridge of peritoneum, which shows when the bowel has been pulled towards the right side. Herniation of small intestine through this space, with acute intestinal obstruction, has been shown to occur by Gabriel in a small percentage of cases in which it has not been closed. A glass rod is then passed through an avascular region of the mesentery. In cases of temporary colostomy, one should suture the two limbs of the bowel together as described by

Paul. In cases of permanent colostomy it is important that one should form some kind of bridge; this may consist either of peritoneum, fascia, or skin, a pedicle of which is carried through the opening in the mesentery for the glass rod. This bridge prevents sinking in of the colostomy, and the passage of fæces into the lower bowel. The peritoneal incision is now sutured, the last stitch on each side of the colostomy being tied over an appendix epiploica. The external oblique is sutured, the internal oblique being caught up in those sutures which lie nearest to the bowel. Finally, skin sutures are inserted, those nearest the bowel catching up the external oblique. No sutures should be inserted into the bowel wall. A small elipse of skin may, with advantage, be excised on both sides of the colostomy. Dressings are now applied around the base of the colostomy, passing underneath the glass rod, and the colostomy is then opened freely with the cautery by a crucial incision, which passes transversely and longitudinally along the whole length of exposed bowel. An finger is passed into the colostomy, it should admit one finger easily but not two. At this stage a few ounces of olive oil may be run into the upper limb of the bowel.

In cases where a bridge of skin is employed it is not essential to put a glass rod in the mesentery, though it is safer to do so until the wound has healed. The bowel intervening between the two openings may be completely divided when the bridge is strong, either with the cautery or by tying a silk ligature tightly round it. This leaves two colostomy openings separated by a piece of skin, and there is now no possible chance of fæces getting into the lower limb. The lower of the two openings will atrophy to a certain extent after a time, but will not close completely.

After-Care of Colostomy.

The opening of the colostomy at the time of the operation prevents the pain from gaseous distension, which may be so distressing to the patient, and which may be an etiological factor in the production of paralytic ileus. After operation the dressings around the colostomy are left undisturbed until the bowel acts, thus giving the wound a good chance to heal by first intention. The sutures in the wound should be removed early, i.e., the sixth to seventh day. The glass rod is kept in position for two weeks or until the patient is getting up, when it can be replaced by a piece of rubbertubing, the ends of which have been bent over and tied with silk. This is more mobile and is less liable to injure the tissues while the patient is going about. It should be kept in position for about a further ten days.

There are two methods used in the after-care of colostomy, each having points in its favour, so that it probably is best to decide which method is to be used in each individual case.

(1) The wash-out method.—This is probably best suited for heavy manual labourers, as they can be sure that after the wash-out, either in the morning or the evening, the bowel will not act in the next twenty-four hours. It is, however, contra-indicated if there is any associated diverticulosis, as there is a definite risk of perforation of a diverticulum by the catheter. On the second night after operation

the patient is given thirty minims of cascara evacuant and one ounce liquid paraffin, and on the third morning a wash-out is given. The wash-out is given through a catheter and funnel, and is run in slowly, one to two pints of water, soap and water, or saline being used. The patient is easily taught the procedure himself, and it should not take much more than half an hour. After the wash-out has been returned, the patient is absolutely safe, in the absence of diatetic indiscretions, for the next twenty-four hours. In some cases using this method, the irritation of the wash-out may give rise to a chronic catarrh of the mucosa of the colon, with a resultant excess production of mucus.

(2) The conservative method in many cases will be quite satisfactory, once the patient has become accustomed to his new life. At first the bowel will probably act at all times of the day, but this will settle down, and subsequently there will be an action after meals, and eventually only once or twice at the most per day, provided care is exercised with regard to diet.

It is most important to get a well-fitting belt to support the abdominal wall, and to give the patient confidence. A layer of soft wool is applied over the colostomy, and then a slightly concave celluloid disc, about four to five inches in diameter, which gives added support in the weakened region of the colostomy. The various cup devices should never be used, as they aid in the production of prolapse of the bowel and hernia formation. A light elastic belt should also be procured for night use.

Diet.

It is important to maintain during the first week a fluid regime, and then gradually increase to a basal diet, such as that described by Bargen and Victor, by the third week.

Breakfast:

Orange-juice, quarter glass.
Bacon and egg.
Toast and butter as desired.
Coffee.

Dinner:

Meat and potato.
Shredded lettuce.
Bland dessert—no fruit.
Bread, toast or crackers if desired.
Boiled milk, one glass, or tea.

Supper:

Cheese.
Meat, one serving, or two eggs.
Potato or substitute.
Fruit—ripe banana or other bland fruit.

Bread or crackers.

Jelly if desired.

Boiled milk, one glass.

Brewers' yeast may be taken with all meals if desired.

An intelligent patient will find out for himself which articles he can safely add to his diet, and which things he will have to avoid, by the trial and error system. He will require warning in the use of laxatives and spirits, condiments, etc. One patient at St. Mark's was so keen on his daily glass of beer that he had the perseverance to try out forty different brands before he found one which suited him. One of the most important points in the after-care is to try and get the patient back to his normal work—for it is in persons with a daily routine to follow out that a colostomy acts most satisfactorily. The colostomy is not a handicap, even in the most arduous of work.

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THE TREATMENT OF SYPHILIS

THE manufacturers of N.A.B.—NOVARSENOBILLON—have sent us a copy of their new publication on this product in the treatment of syphilis.

N.A.B. — NOVARSENOBILLON — which is now widely employed throughout the Empire, is indicated in primary, secondary, and tertiary somatic syphilis. Twenty-three years of consistent use have established the position of this product so firmly that a complete revisal of the previous publication has been considered unnecessary. Certain modifications, however, have been made where recent experience has shown them to be desirable, so that the medical practitioner using N.A.B. — NOVARSENOBILLON—may have access to the latest information available.

Copies of this book will, we understand, be forwarded gladly to any member of the medical profession on request to the manufacturers, Pharmaceutical Specialities (May & Baker), Ltd., Dagenham.

Bread or crackers.

Jelly if desired.

Boiled milk, one glass.

Brewers' yeast may be taken with all meals if desired.

An intelligent patient will find out for himself which articles he can safely add to his diet, and which things he will have to avoid, by the trial and error system. He will require warning in the use of laxatives and spirits, condiments, etc. One patient at St. Mark's was so keen on his daily glass of beer that he had the perseverance to try out forty different brands before he found one which suited him. One of the most important points in the after-care is to try and get the patient back to his normal work—for it is in persons with a daily routine to follow out that a colostomy acts most satisfactorily. The colostomy is not a handicap, even in the most arduous of work.

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SUMMARY.

(1) After accidental or surgical trauma an aseptic pyrexia occurs—it is of rapid onset and short duration.

(2) The pyrexia occurs independently of the presence of anæsthesia or the type of anæsthetic. Therefore anæsthesia plays little part in its production.

(3) The pyrexia appears to depend mainly on the extent of tissue damage and effusion of blood into the tissues, and is due to toxic products of tissue breakdown, including blood, and to increased metabolic disturbance attempting to repair the cellular damage.

(4) The toxins derived from blood and cellular debris are probably protein derivatives.

I wish to thank Mr. H. Stevenson, F.R.C.S.I., for permission to examine his patients, the majority of the patients concerned being under his care. Also Mr. C. J. A. Woodside, F.R.C.S.I., for suggestions and advice.

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INTRAVENOUS UROGRAPHY

THE new organic iodine compound mainly used as a contrast medium in intravenous urography and other radiographic investigations is now being manufactured in Great Britain by Messrs. May & Baker, Ltd., under the registered name of UROPAC.

This preparation — the disodium salt of 3:5-di-iodo-4-pyridoxyl-N-methyl-2:6-dicarboxylic acid—is low in toxicity, and the rapidity with which it is excreted by the kidneys ensures complete safety in use. Previous to the outbreak of war, the drug was not produced in this country. Now supplies of it are available as a British product in the form of UROPAC.

A specimen of the UROPAC booklet has been sent to us by Messrs. Pharmaceutical Specialities (May & Baker), Ltd., Dagenham, who, we understand, will be glad to forward a copy on request to any member of the medical profession.

Thompson (1936) and Whipple (1937) recommend that hæmolytic anæmia be divided into two major groups, "typical" and "atypical." The typical group consists of familial acholuric jaundice which has definite diagnostic criteria — a chronic disease of long duration and of relative mildness with acute exacerbations; chronic variable jaundice, anæmia with evidence of blood regeneration, spherocytosis, and splenomegaly. The characteristic feature of the typical hæmolytic anæmias is the presence of spherical microcytes.

The atypical group includes acquired acholuric jaundice, and is heterogeneous except for the uniform absence of spherocytosis. In some cases the cause is known (infections, chemicals, etc), in others none can be found during life or at post-mortem.

This classification is unsatisfactory, but is useful in the absence of exact knowledge of the mechanism of hæmolysis in idiopathic hæmolytic anæmias—whether it is the result of congenital and acquired defects in the red cell or due to the presence or advent of hæmolysins.

I wish to acknowledge my indebtedness to Dr. S. I. Turkington for permission to publish this case, which was under his care, and to thank Professor W. W. D. Thomson and Professor J. H. Biggart for their encouragement and help in preparing this paper.

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GENITO-URINARY INFECTIONS

A fourth edition of the booklet on DAGENAN (M & B 693) in the treatment of genito-urinary infections has recently been sent to us.

This drug is now widely employed in gonorrhœa. In the course of a discussion concerning the treatment of venereal diseases in the B.E.F., it was stated that "The treatment of gonorrhœa with M & B 693 has shown that in all but very rare instances complete cure is achieved in three weeks." The possibility of treating cases at the front brought forward the opinion that "The proved efficiency of M & B 693 in the treatment of gonorrhœa renders the evacuation of such cases to the base quite unnecessary."

The latest publication on DAGENAN contains a note on the treatment of gonorrhœa in general practice. A copy will be sent, we understand, to any medical practitioner on request to Messrs. Pharmaceutical Specialities (May & Baker), Ltd., Dagenham, England.

Post-Operative Pyrexia

By WILLIAM TOWNSLEY, M.B., B.CH.
House Surgeon, Royal Victoria Hospital, Belfast

ON examining the charts of patients in any surgical ward a rapid rise of temperature is often noticeable, beginning usually a few hours after operation and lasting one to five days. This pyrexia follows the majority of operations—not merely in cases suffering from some inflammatory malady requiring operation, but notably in non-infected cases.

For the purpose of writing this paper I have investigated six hundred surgical cases—having had personal contact with four hundred—in the wards of the Royal Victoria Hospital, Belfast. The remaining two hundred I studied in the form of patients' charts in the record office of that hospital. The operations were of many types, but all were performed on patients who showed no rise of temperature for several days preceding operation — that is, the operations were not performed on patients suffering from infected conditions. The operations included those for goitre, hæmorrhoids, tumours (carcinoma of breast, rectum, etc.), enlarged prostate, gall-stones, fractured bones, hydronephrosis; they also included herniotomies, gastrectomies, gastro-enterostomies, etc.

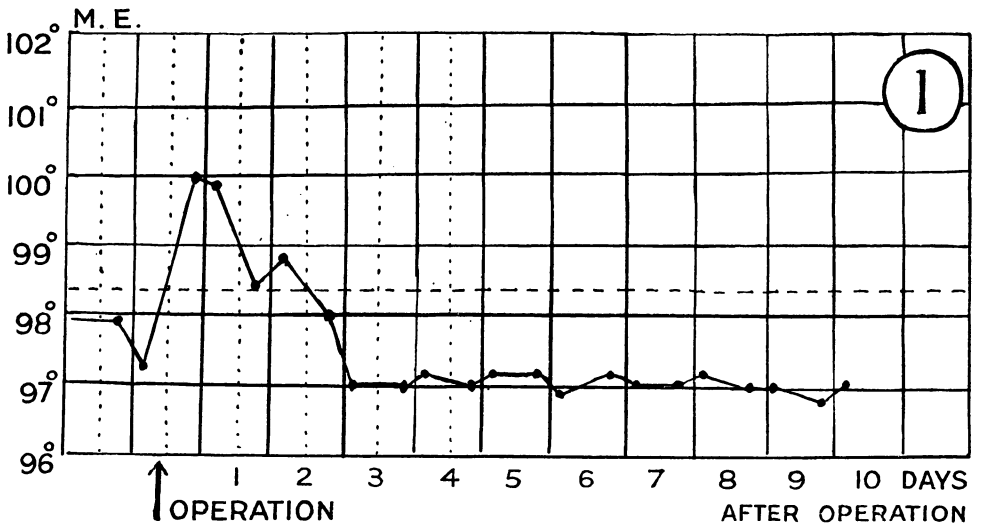

Chart No. 1.
Operation: excision of displaced cartilage of knee. Anæsthetic: chloroform and ether.

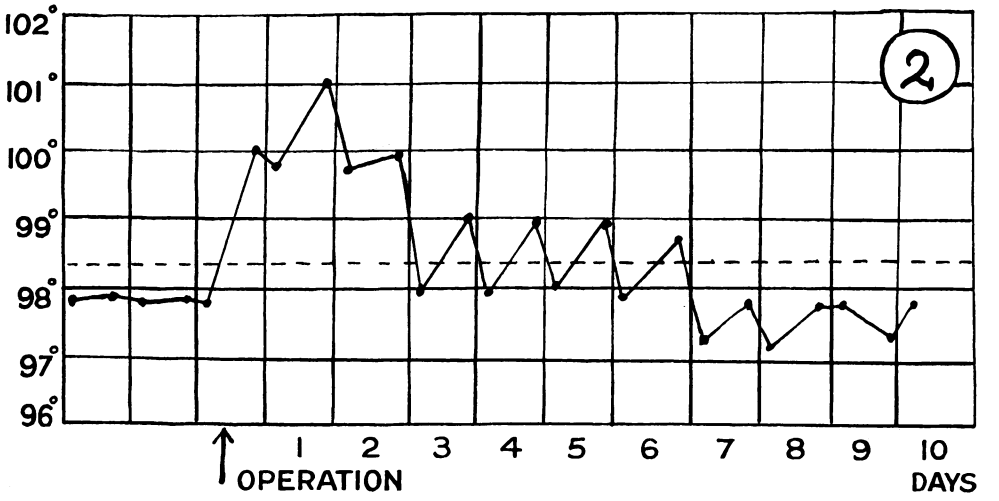

Chart. No. 2.

Operation: herniotomy. Anæsthetic: local (novocaine).

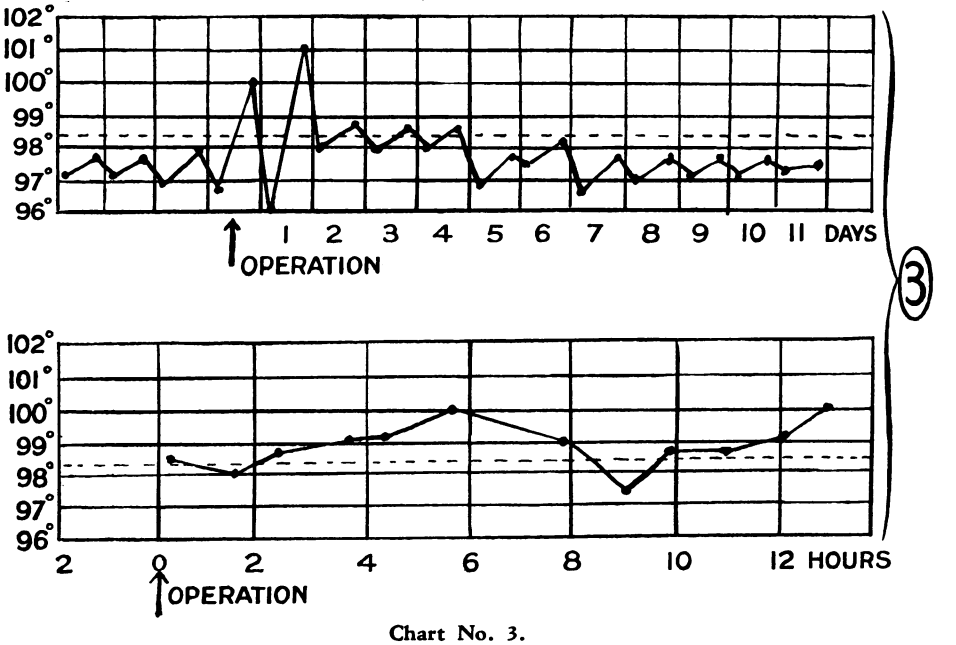

Chart No. 3.

Operations: goitre excisions. Anæsthetics: gas and oxygen.

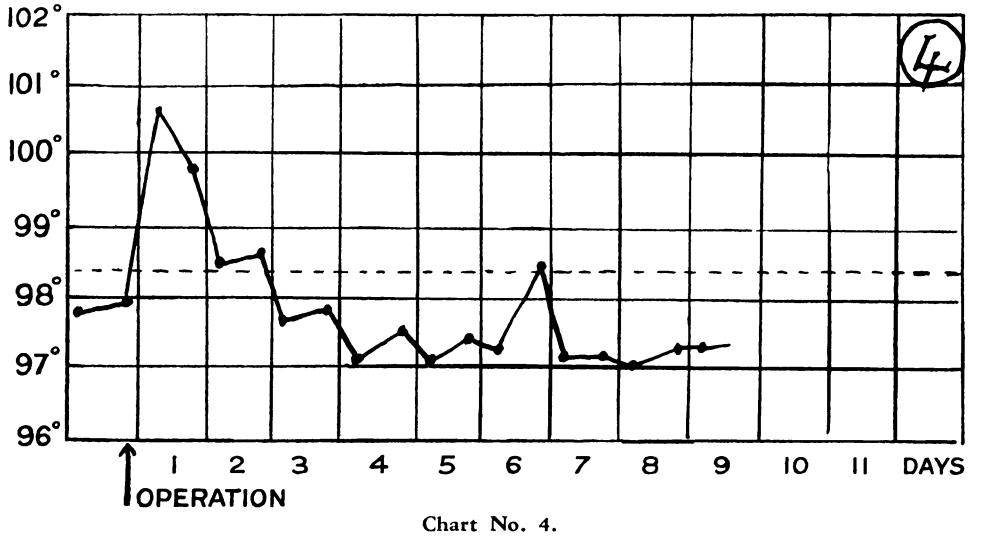

Chart No. 4.

Operation: appendicectomy. Anæsthetic: chloroform and ether.

DESCRIPTION OF CASES.

In the following reproductions of typical charts, it will be noted that a rapid rise of temperature occurs, either within six hours following operation, or on the following morning. The temperature usually reaches its maximum on the day after operation, and then begins to fall, often more gradually than it rose. The duration of the pyrexia is one to five days, usually about three days.

On these temperature charts the temperature is registered twice daily, at 8 a.m. and 5 p.m., and the majority of the operations were performed between 10 a.m. and 1 p.m.

THEORIES OF CAUSATION.

One thinks of several factors which, either singly or in combination, might give rise to the pyrexia. They are :—(1) operative trauma, (2) anæsthesia, (3) infection, (4) physiological reaction, (5) psychic influence.

(1) TRAUMA : After an injury involving damage or destruction of tissue, a rise of temperature can often be demonstrated which resembles closely that following surgical interference. For example in a series of eighty-four cases of fractured bones (femur, tibia, humerus, etc.) admitted to the Royal Victoria Hospital, fifty-four showed a rise of temperature beginning one or two days after injury and gradually disappearing over a period of four to eight days, or even longer—that is, a rise of temperature occurred in seventy per cent. of cases. None of these patients suffered surgical trauma or an anæsthetic during the period of pyrexia, or preceding

it, most of them being treated with splints (Thomas's, plaster of paris, etc.) on admission to hospital. They were subjected to operation, where necessary, only after at least one week in hospital. A typical chart of a fracture case with pyrexia is shown in fig. 5.

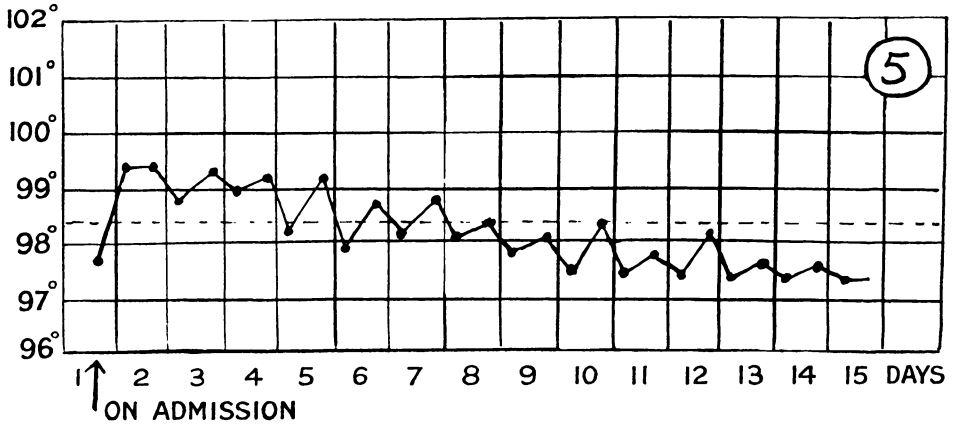

Chart No. 5.

Fractured femur. No operation, no anæsthetic.

In some cases, immediately following injury, there is a fall of temperature to 96°; this is quickly followed in favourable cases by a rise of temperature.

For example: A patient is admitted to hospital with a fractured femur, the accident having happened three-quarters of an hour before. The affected limb is cold and the patient shocked, with a low blood-pressure and low temperature—traumatic shock due to the liberation of histamine following injury is present.

Or again, a patient returns from the operating-theatre in a shocked condition with a low temperature — surgical shock. In both cases, treatment directed at alleviation of shock will quickly raise the temperature to normal, and in a few hours a state of pyrexia will exist. Both of these cases are shown typically in charts 6 and 7.

Only sixteen per cent. of cases in the series of fractures of large bones showed a subnormal temperature on admission.

Febrile reactions can be observed after other forms of tissue trauma, for example, in cardiac infarction and cerebral hæmorrhage (excluding severe hæmorrhage where the pons is affected and a rapidly climbing terminal pyrexia is found). Tidy[1] mentions a febrile reaction following cerebral hæmorrhage—its onset is twelve to forty-eight hours after the attack, and it lasts from one to several weeks; it is due to absorption of blood.

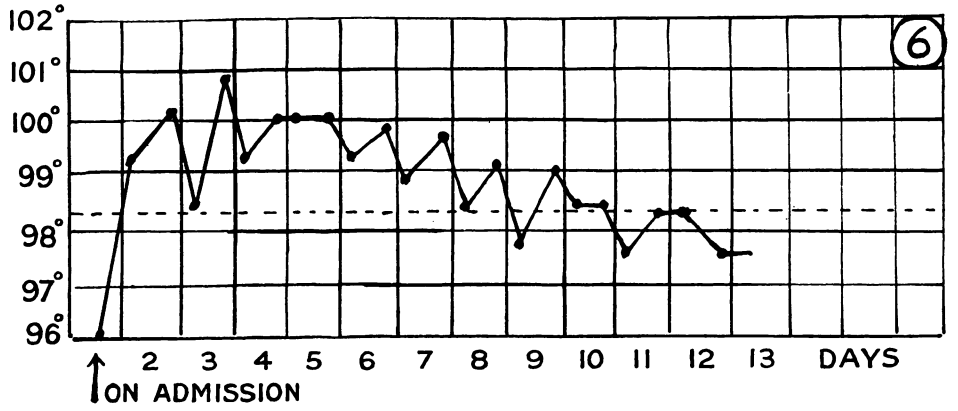

Chart No. 6.

Fractured femur. No operation, no anæsthetic.

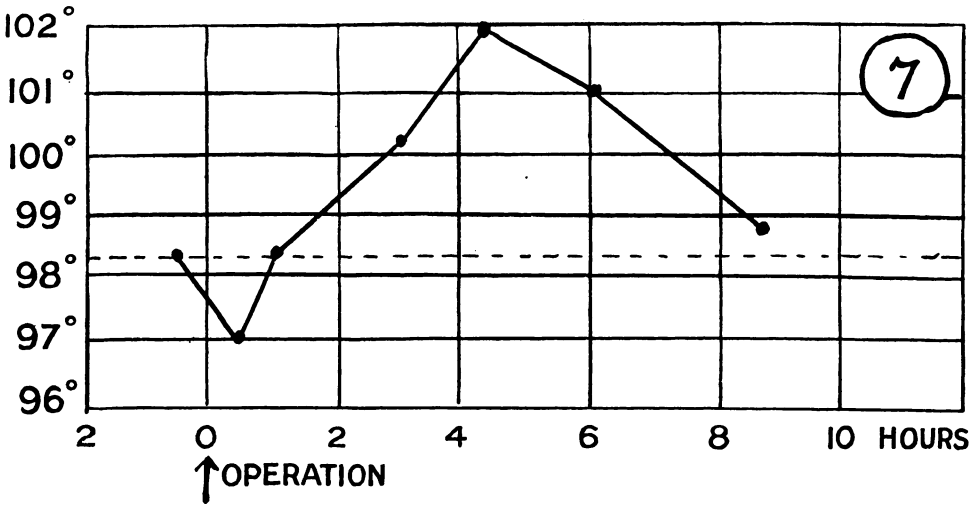

Chart No. 7.

Operation: goitre excision. Anæsthetic: gas and oxygen.

A febrile reaction following injury is described by several authorities, for example :—

Bailey and Love[2] :—"Aseptic traumatic fever : this is due to the absorption of fibrin ferment from extravasated blood and resembles the fever which occurs in any simple wound." (The aseptic traumatic fever referred is that following a fracture.)

Pye[3] :—"An increased temperature is not uncommon for a day or two after severe fractures, even though there is no compound injury and no infection. The febrile reaction disappears as the blood clot undergoes absorption."

Tudor Edwards[4] :—"The absorption of blood from the pleural cavity invariably causes pyrexia sometimes as high as 101°."

Russell Howard,[5] speaking of septic traumatic fever, says :—"Slight rise of temperature, increased pulse-rate and respiration . . . general malaise lasting twenty-four to forty-eight hours, it is seen in cases of simple fracture with considerable extravasation of blood . . . In fever due to mechanical trauma, it is probable that the toxin is fibrin ferment."

During surgical procedures a certain amount of damage is done to the tissues, and some extravasation of blood occurs into the incised area, even after ligature of all visibly bleeding vessels and closure of the wound. Hence, tissue damage must play a major part in the production of post-operative pyrexia.

The relation of post-traumatic pyrexia to biochemical changes in the body has been closely investigated by Cuthbertson,[6] whose work is quoted fully :—

"The loss of body nitrogen, sulphur, and phosphorus which occurs in the urine of otherwise healthy individuals, who have received moderate or serious traumatic injury, was observed to begin within a day or two following injury, to reach a maximum within ten days, and then slowly decline. There generally occur parallel increments in the basal consumption of oxygen, body temperature, and pulse-rate. These phenomena occur in uncomplicated fractures of long bones, dislocations, effusions into joints, laceration of soft tissues, and surgical incisions into knee joints. The wasting of muscle and bone caused by immobilization in fracture and knee-joint cases was insufficient to account for the whole catabolic loss. There was in most cases an evident initial depression of metabolism following injury, followed by a counter-swing. It is believed that these changes were the result of the organism catabolizing its reserves to meet the exigencies of repair and maintenance rather than due to the sweeping out of the disintegration products of damaged tissues. The period of maximum nitrogen excretion was the fourth to the eleventh day following injury, generally the sixth day. The catabolic disturbance is characterised by an increase in basal consumption of oxygen with attendant rise in pulse and temperature."

In another paper, Cuthbertson[7] states that compounds formed from the breaking up of cellular debris in the fractured or injured area may cause a disturbance in the normal balance of the body cells, resulting in an imbalance between anabolism

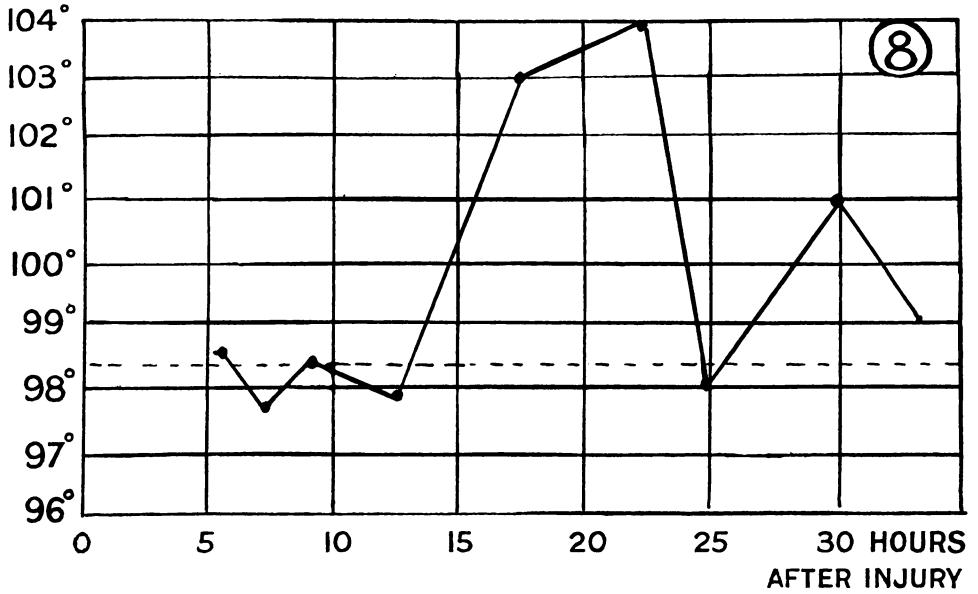

Chart No. 8.

Acute toxæmia following burns. No operation, no anæsthetic.

and catabolism. A small febrile rise is occasionally found in cases of injury. Protein reserves are being catabolized in an endeavour to maintain a constant level of metabolism following on the earlier and more rapid exhaustion of the carbohydrate reserves, and any slight increase in heat production is due to the specific dynamic action of the protein."

Wilson[8] classifies the reactions of the body following injury into four groups (with special reference to burns) : (a) primary shock, (b) secondary shock, (c) acute toxæmia, (d) septic toxæmia. Of acute toxæmia he says :—"It appears to be due to a toxin formed in the injured tissues. This is a product of partial protein degradation, and is precipitated with the globulin fraction." This stage of acute toxæmia occurs six to twenty-four hours after injury, and is characterized in the temperature chart which he reproduces by a raised temperature (fig. 8).

It appears, therefore, that pyrexia following injury or operation is due to either of the following factors : (a) some toxin formed in the damaged tissues—it may be from blood as "fibrin ferment" (that is, thrombin, a protein euzyme), or it may be some breakdown product of proteins from the damaged tissues; (b) increased metabolic disturbance on the part of the body in attempting to repair the injured tissues; (c) It may result from a combination of toxic and metabolic disturbance.

It must be remembered that many protein substances have a toxic and temperature-raising effect on the body—bacterial toxins are albumoses, while ricin, abrin, and vennins are toxalbumins. Protein shock, with a rise of temperature, is produced by the injection of milk, peptone, or dead bacteria parenterally into the body.

Serum sickness and anaphylactic shock are similar febrile phenomena, resulting from the introduction of proteins into the tissues.

In some tumours, apart from the presence of bacterial infection, pyrexia may occur, and is probably due to the absorption of products of protein disintegration or abnormal metabolism. It is recognised also that cardiac infarction may be attended by some pyrexia—probably the result of the absorption of products of autolysis from the necrosed muscle. Finally, MacCallum states that it is injury to cells and consequent decomposition of protein which stimulates the heat-controlling centres.

ANÆSTHESIA.

It is obvious from a consideration of various types of operation and associated anæsthesia, that the anæsthetic plays only a small and insignificant part—or no part—in the production of the succeeding pyrexia. The following table shows clearly that (1) the percentage of cases exhibiting a raised temperature and (2) the extent and duration of the temperature depend on the severity of surgical trauma, rather than on the anæsthetic.

It should be mentioned that in the following series of cases, operations have been arbitrarily divided into "major," "medium," and "minor" operations. "Major" operations are those with severe operative trauma affecting the tissue. This group includes prostatectomy, gastrectomy, gastro-enterostomy, herniotomy, cholecystectomy, nephrectomy, sympathectomy of renal artery for hydronephrosis, and excision of carcinomata of various organs; also goitre. "Medium" operations are those in which tissue damage is less severe than in the previous group. The operations include diathermy excision of tumours of the skin, tongue, etc., insertion of radium needles in growths and the surrounding areas, gynæcological operations, such as dilatation and curettage of the uterus, cauterisation of urethral caruncle or infected cervix, also extraction of cataracts and submucous resection of nasal septum, etc. "Minor" operations are those in which tissue damage is negligible. They include paracentesis abdominis, lumbar puncture, pleural aspiration, etc.

A.—*Major Operations.*

The rise of temperature is marked and prolonged.

Anæsthetic	No. of cases	Temperature raised in
Chloroform and Ether ...	140 ...	82 per cent.
Gas and Oxygen	36 ...	83 per cent.
Spinal	30 ...	73 per cent.
Local (Novocaine)	16 ...	93 per cent.
		Average=83 per cent.

B.—*Medium Operations.*

Temperature is small and of short duration.

Anæsthetic	No. of cases	Temperature raised in
Chloroform and Ether ...	53 ...	25 per cent.
Sodium Evipan	30 ...	60 per cent.
Local (Novocaine)	57 ...	65 per cent.

Average = 50 per cent.

C.—*Minor Operations.*

Transient rise of temperature.

Anæsthetic	No. of cases	Temperature raised in
Local (Novocaine)	21 ...	14 per cent.

D.—*Anæsthesia, with no Operation.*

Transient rise of temperature.

Anæsthetic	No. of cases	Temperature raised in
Chloroform and Ether ...	14 ...	12 per cent.
Spinal	16 ...	25 per cent.
Local	48 ...	10 per cent.

Average = 15 per cent.

E.—*Trauma: No Operation, no Anæsthetic.*

84 Fractures. Temperature raised in 70 per cent.

The same types of anæsthetic were common to groups A, B, C, and D. Yet in group A, sixteen cases of herniotomy were done under local anæsthesia and ninety-three per cent. developed post-operative pyrexia, while in group C, twenty-one cases of lumbar puncture, paracentesis abdominis, etc., were performed under local anæsthesia and showed a rise of temperature in only fourteen per cent. cases. The damage to body tissue was much greater in group A.

Again in group A, operation was carried out on one hundred and forty cases under chloroform and ether anæsthesia, and post-operative rise of temperature was found in eighty-two per cent. cases, while in group D fourteen cases of examination (no surgical interference) were carried out under chloroform and ether, and a slight and transient temperature was present in only twelve per cent. of cases.

Again in group E, tissue damage caused by fractured bones, uncomplicated by anæsthesia, operation, or sepsis, showed a rise of temperature in seventy per cent. of cases in a series of eighty-four fractures.

Some explanation must be offered for the mild and transient pyrexia in a small proportion (twelve per cent.) of cases in group D, where the patient, for purposes of examination only, was subjected to chloroform and ether anæsthesia, but sustained no surgical tissue damage. The pyrexia may be due to :—(1) the effects of anæsthetics on the tissues, (2) increased secretion of adrenaline, (3) psychical causes, (4) physiological reaction.

(1) It is known that chloroform and ether may damage body cells, and produce, for example, albuminuria. Cushny[9] states :—"The kidney appears to be affected in a certain proportion of cases of anæsthesia, as shown by albuminuria." Also, "Acute yellow atrophy may occur in rare cases, while even in ordinary anæsthesia the damage to the liver by chloroform is by no means negligible." And again he says :—"Nitrogen elimination in the urine is considerably increased, and also unoxidised sulphur—there is increased protein destruction and disturbance in oxidation of the tissues." Clark[10] states that albumin and casts appear in urine in twenty-five per cent. of cases after ether anæsthesia.

This destruction of tissue may give rise to cellular toxins, which act like those produced after direct trauma, as in fractures, or operations, and so give rise to pyrexia, but in a much smaller percentage of cases and in lesser degree.

(2) An attractive hypothesis is that which may be based on the fact that anæsthesia increases adrenaline secretion, and Beattie[11] has told us, following his work on hypothalamic stimulation, that increased adrenaline secretion causes a rise in temperature (because adrenaline dilates vessels in muscles, and the continuous release of small quantities of adrenaline is responsible for the production of heat, necessary to maintain body temperature, in the muscles).

Psychical and physiological causes will be considered later.

INFECTION.

Infection plays no definite part in the onset of pyrexia. The rise of temperature is often present six hours after operation, or on the following morning, hence bacterial multiplication and invasion could not have occurred in that short space of time.

Infection of the wound itself certainly plays no part, for it is still clean and free from inflammatory redness and pus. When wound infection does occur, it is only after the lapse of several days that pus is seen, that is, after the primary pyrexia has settled down. A new and greater swing is now noticed on the temperature chart, and this new rise of temperature usually follows an apyrexial interval, or is superimposed on the disappearing "tail" of the aseptic traumatic pyrexia. The same applies to pyrexia due to true bronchitis or pneumonia. Charts 9, 10, and 11 are self-explanatory.

I have examined day by day, before and after operation, the lungs of forty-two patients who had major operations under chloroform and ether anæsthesia, and found the following facts :—

(1) In twenty-five cases (sixty per cent.) there were no adventitious sounds or other evidence of pulmonary trouble following operation (i.e., no bronchitis or pneumonia), and yet twenty-four out of these twenty-five showed post-operative pyrexia.

(2) Seventeen had rhonchi and moist râles following operation (four of these had adventitious sounds before operation) and a raised temperature, but the rhonchi and râles persisted for many days after the temperature had regained normal limits, the average duration of temperature being three days.

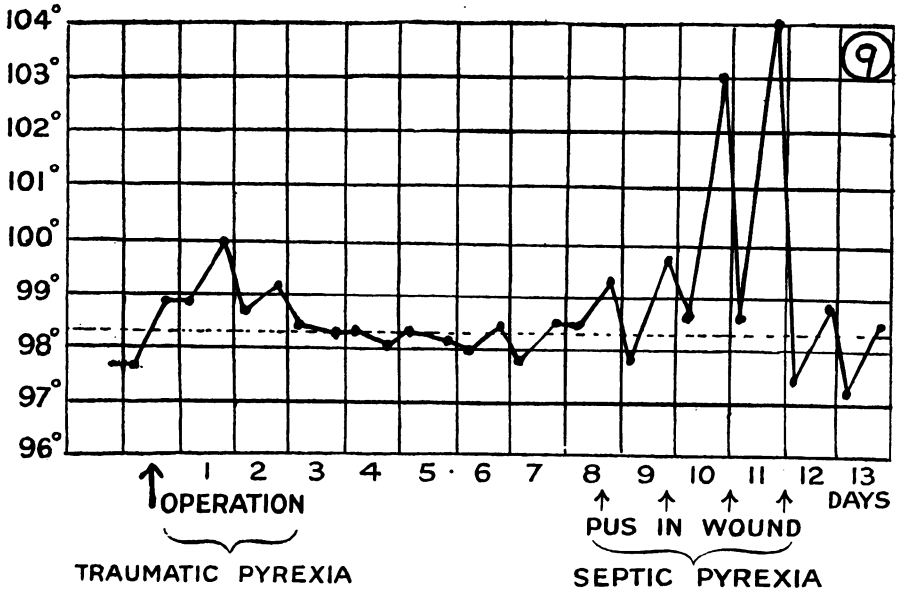

Chart No. 9.

Operation: nephrectomy. Anæsthetic: chloroform and ether.

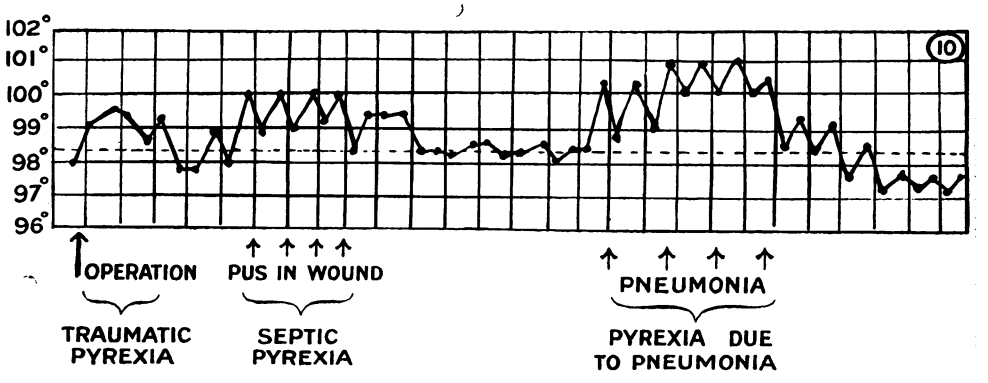

Chart No. 10.

Operation: appendicectomy (gangrenous appendix). Anæsthetic: chloroform and ether.

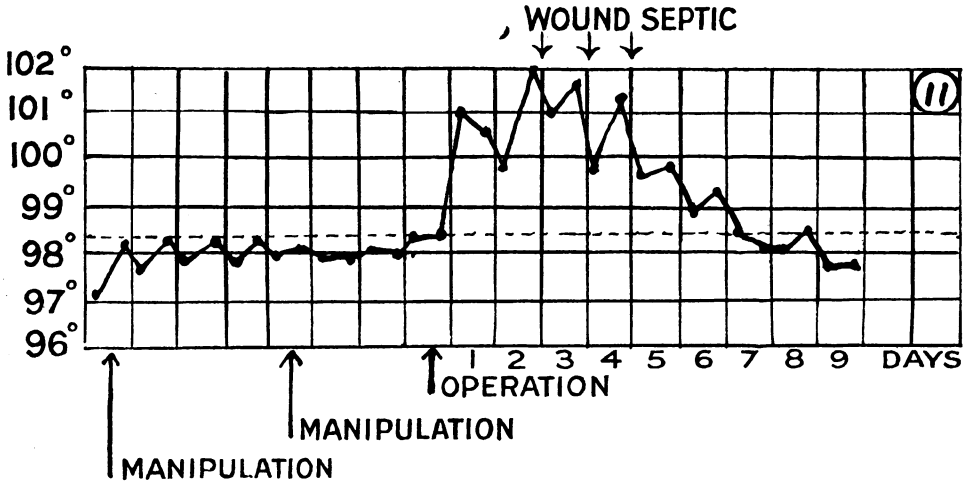

Chart No. 11.

Dislocated shoulder. Three separate anæsthetics: chloroform and ether.

1. Manipulation: no rise in temperature.
2. Manipulation: no rise in temperature.
3. Operation: post-operative pyrexia.

A secondary septic pyrexia, due to wound infection, is superimposed on the disappearing primary aseptic (traumatic) pyrexia.

If these physical signs had represented a bronchitis due to bacterial invasion, and not merely an increased secretion of mucus due to chemical irritation, the pyrexia would surely have been more prolonged. Clark[12] states:—"Ether bronchitis occurs in ten per cent. of cases." Yet the pyrexia occurs in eighty per cent. to ninety per cent of cases. Therefore bronchitis plays little or no part in its actiology.

Again, this high incidence of pyrexia is present after all forms of anæsthesia, not merely after "irritant" vapours like ether or non-irritant inhalations like gas and oxygen, but also after intravenous sodium evipan or pentothal and local anæsthetics. It is also present after all types of operation, not only after upper abdominal ones which might damage the lungs by immobilizing them.

PHYSIOLOGICAL REACTION.

A fall of temperature and blood-pressure are said to occur during surgical operations and anæsthesia, the result of histamine shock. It may be that (a) the lowered body temperature during operation (the result of some degree of shock) causes an increased secretion of thyroxine, which subsequently raises body metabolism and temperature for a few days following operation, or (b) that the subsequent pyrexia is an unexplained physiological "swing of the pendulum."

PSYCHICAL CAUSES.

"Nervousness" following injury or operation may possibly cause the pyrexia in a few cases, but there is no evidence to prove this.

SUMMARY.

(1) After accidental or surgical trauma an aseptic pyrexia occurs—it is of rapid onset and short duration.

(2) The pyrexia occurs independently of the presence of anæsthesia or the type of anæsthetic. Therefore anæsthesia plays little part in its production.

(3) The pyrexia appears to depend mainly on the extent of tissue damage and effusion of blood into the tissues, and is due to toxic products of tissue breakdown, including blood, and to increased metabolic disturbance attempting to repair the cellular damage.

(4) The toxins derived from blood and cellular debris are probably protein derivatives.

I wish to thank Mr. H. Stevenson, F.R.C.S.I., for permission to examine his patients, the majority of the patients concerned being under his care. Also Mr. C. J. A. Woodside, F.R.C.S.I., for suggestions and advice.

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INTRAVENOUS UROGRAPHY

THE new organic iodine compound mainly used as a contrast medium in intravenous urography and other radiographic investigations is now being manufactured in Great Britain by Messrs. May & Baker, Ltd., under the registered name of UROPAC.

This preparation — the disodium salt of 3:5-di-iodo-4-pyridoxyl-N-methyl-2:6-dicarboxylic acid—is low in toxicity, and the rapidity with which it is excreted by the kidneys ensures complete safety in use. Previous to the outbreak of war, the drug was not produced in this country. Now supplies of it are available as a British product in the form of UROPAC.

A specimen of the UROPAC booklet has been sent to us by Messrs. Pharmaceutical Specialities (May & Baker), Ltd., Dagenham, who, we understand, will be glad to forward a copy on request to any member of the medical profession.

Case of Macrocytic Hæmolytic Anæmia

By Thomas G. Milliken

History.

A young woman, aged 30, unmarried, and employed as a shop assistant, was admitted to the Royal Victoria Hospital on 7th November, 1939. She complained of weakness, palpitation, and breathlessness on exertion, present since July, 1939. Since August she had noticed that her skin was a yellow colour. This yellow colour tended to disappear for short periods, but her other symptoms gradually increased in severity during the four months of their presence.

Previous Illnesses.—She had measles when a child, and "abscesses on her gums" in May, 1939.

Personal History.—She had a fair appetite, her bowels were rather loose, and micturition was normal. She was a sound sleeper, but was losing weight. No note was made of her menses. Her parents, brothers, and sisters were all healthy, and there was no family history of jaundice.

Examination.

The patient was a tall, linear young woman of good nutrition, rather nervous and excitable. The scleral conjunctivæ and the skin of the face, limbs and body were a lemon-yellow colour, and the palpebral conjunctivæ showed a considerable degree of anæmia. There were no enlarged cervical or axillary lymph-glands, and no œdema of the face or ankles.

Cardiovascular System.—The pulse-rate was consistently raised to 100 or more, and was easily raised to 120 by any excitement. There was no gross enlargement of the heart. The sounds were regular, but there was a soft localised mitral systolic murmur.

Alimentary System.—The tongue was rough, moist, not sore, and teeth, gums, and throat were healthy. Nothing abnormal was discovered in the abdomen, and the liver and spleen were not palpable.

The respiratory system was normal, and there was no organic nervous disease. The temperature was normal.

Investigations.

Blood.—Red cells : 1,320,000 per cm.
Hæmoglobin : 40 per cent. (Sahli); colour index 1.5.
White cells : 6,552 per cm.

Differential White-Cell Count:

Polymorphonuclear neutrophils ...	63 per cent.
Polymorphonuclear eosinophils ...	1 per cent.
Polymorphonuclear basophils ...	0 per cent.
Small lymphocytes	25 per cent.
Large lymphocytes	7 per cent.
Monocytes	4 per cent.

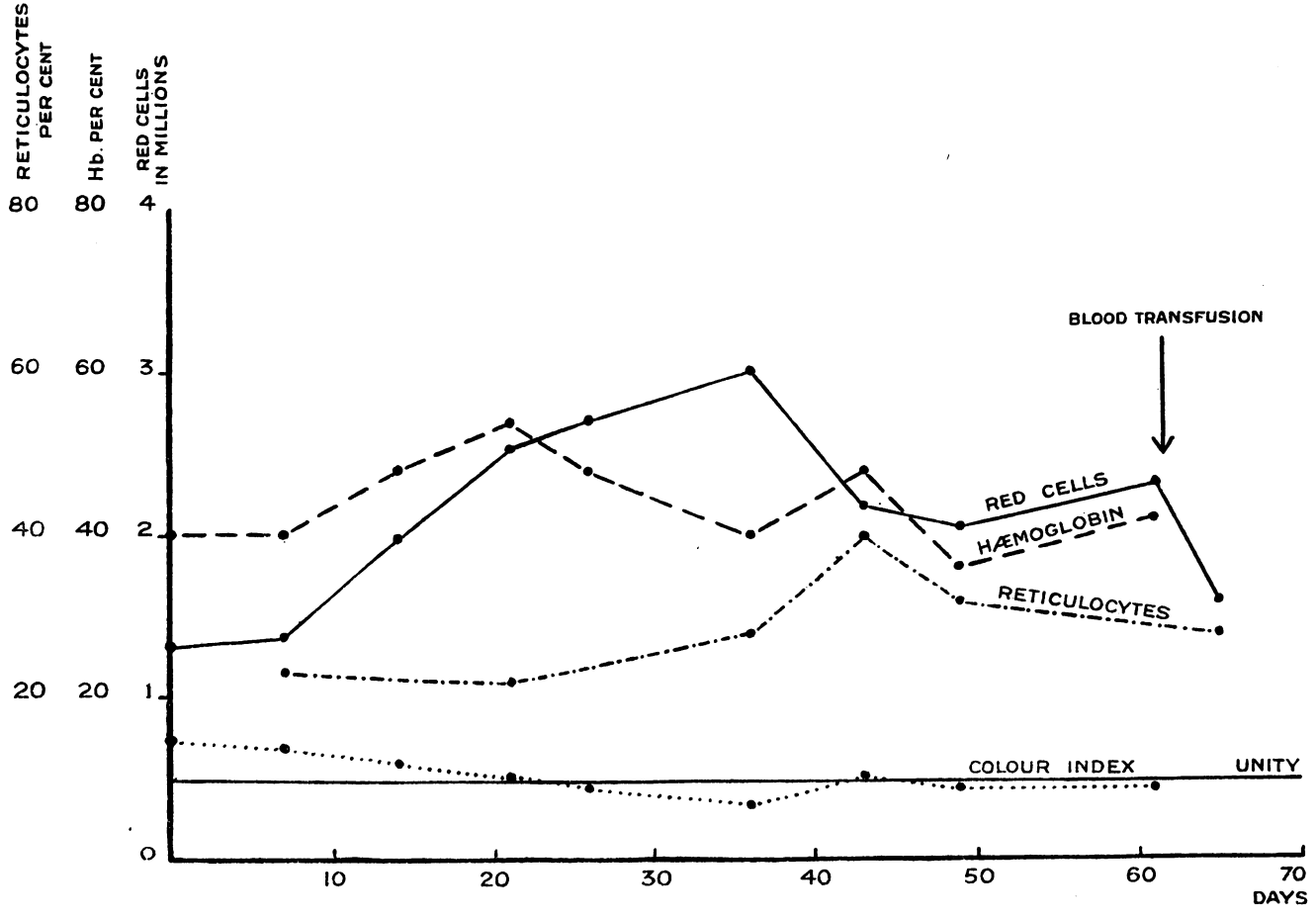
RETICULOCYTES PER CENT
Hb. PER CENT
RED CELLS IN MILLIONS
80
60
40
20
0
4
3
2
1
BLOOD TRANSFUSION
RED CELLS
HÆMOGLOBIN
RETICULOCYTES
COLOUR INDEX
UNITY
10
20
30
40
50
60
70
DAYS

Fig 1.

A blood-film (Leishman) showed poikilocytosis, macrocytosis, microcytosis, anisochromia, polychromasia, and a few normoblasts. White cells appeared normal, and platelets were plentiful.

The van den Bergh reaction was positive by the indirect method, and quantitatively showed a marked increase.

The Urine was amber in colour, acid in reaction, and of specific gravity 1020. The tests for albumen, sugar, bile, and blood were negative.

A fractional test-meal was not carried out at this time, as the patient refused to swallow the Ryle's tube, but even in the absence of certain knowledge of achlorhydria, a diagnosis of pernicious anæmia was made, based on clinical findings, the anæmia with high colour index, the positive indirect van den Bergh, and the reticulocytosis of 23 per cent. apparently in response to liver treatment.

The patient was given an intensive course of liver therapy, and periodic checks were kept on her progress, which is summarised in figure 1 and table 1.

TABLE NO. 1.

	R.B.C. (per cmm.)	Hæmoglobin (per cent.)	Colour Index	Reticulocytes (per cent.)
7.11.39 ...	1,320,000 ...	40 ...	1.5 ...	—
13.11.39 ...	1,380,000 ...	40 ...	1.4 ...	23
21.11.39 ...	1,980,000 ...	48 ...	1.2 ...	—
28.11.39 ...	2,530,000 ...	54 ...	1.08 ...	22
3.12.39 ...	2,720,000 ...	48 ...	0.9 ...	—
13.12.39 ...	3,000,000 ...	40 ..	0.66 ...	28
20.12.39 ...	2,180,000 ...	48 ...	1.09 ...	40
26.12.39 ...	2,050,000 ...	36 ...	0.9 ...	32
7. 1.40 ...	2,320,000 ...	— ...	— ...	—
11. 1.40 ...	1,600,000 ...	— ...	— ...	28

The liver therapy was supplemented with iron, but it became increasingly evident from the poor red-cell response that a revision of the diagnosis was necessary. The erythrocyte count in the relapse stage of Addison's anæmia should rise by 400,000 to 700,000 per week following the injection of adequate amounts of a potent liver extract. In this case the count progressed slowly for five weeks and then deteriorated. There was, however, a constant reticulocytosis of twenty to forty per cent. The conclusion was that although red cells were being produced, as shown by the reticulocytosis, cell destruction was proceeding almost apace with cell formation—Piney's hæmopoietic-hæmoclastic balance.

Also, the following points were noted in retrospect which were not entirely in keeping with a diagnosis of pernicious anæmia :—

1. The tongue was rough and not painful. (Sore tongue is present in fifty per cent. of cases, whether there is glossitis or not.)
2. The jaundice at its height was deeper in hue than is usually found in pernicious anæmia.

3. Quantitatively the positive indirect van den Bergh reaction was markedly increased.

Further investigations were now required.

The Wassermann reaction proved negative, and spectroscopic examination of the blood-serum showed no abnormal pigments. The urine also was free of hæmoglobin and hæmosiderin, but no attempt was made to estimate urobilin excretion. The fragility of the red cells, however, was increased, as shown by decreased resistance to hypotonic saline. Hæmolysis began in 0.68 per cent., and was complete in 0.46 per cent. saline. (Normal : 0.44 and 0.34 per cent. In pernicious anæmia there is an increased resistance to hypotonic saline.) The diagnosis suggested was acholuric jaundice of the acquired type.

An examination of the bone marrow by sternal puncture and of the blood was carried out by Dr. J. T. Lewis, who reported the following :—

"Blood film : Many nucleated red cells, both normoblasts and erythroblasts, and an occasional myelocyte. Reticulocytes 40 per cent. Marrow :—

Myeloblasts	...	2 per cent.
Myelocytes	...	9 per cent.
Polymorphs	...	26 per cent.
Eosinophils	...	1 per cent.
Lymphocytes	...	10 per cent.
Erythroblasts	...	29 per cent.
Normoblasts	...	23 per cent.

52 per cent.

Very marked increase in nucleated red cells at the expense of granular forms—findings suggest prolonged red-cell stimulation, e.g., hæmolytic anæmia."

The patient was now persuaded to swallow the Ryle's tube, and a fractional test-meal was carried out. The specimens obtained showed complete absence of free hydrochloric acid by the methyl orange test—without histamine—and a low total acidity. This achlorhydria would have supported a diagnosis of pernicious anæmia if discovered earlier, but in reality did not prove the absence of the intrinsic factor, as the components of gastric secretion fail in the order :—

1. Hydrochloric acid.
2. Pepsin.
3. Mucus.
4. Intrinsic factor.

Achylia was therefore not proven. In this respect it may be noted that in Davidson and Fullerton's (1938) four cases of macrocytic hæmolytic anæmia resembling the present case, achlorhydria was present in two and hypochlorhydria in the other two. Lescher and Osborn's (1939) case also showed a histamine-fast achlorhydria.

Clinical Course of the Disease.

During the first eight or nine weeks she was in hospital the patient was not confined to bed, but felt well enough to wander round the ward, a listless figure with face alternately pale and yellow. She thoroughly enjoyed the Christmas festivities, and it was only in the early days of January that she began to decline,

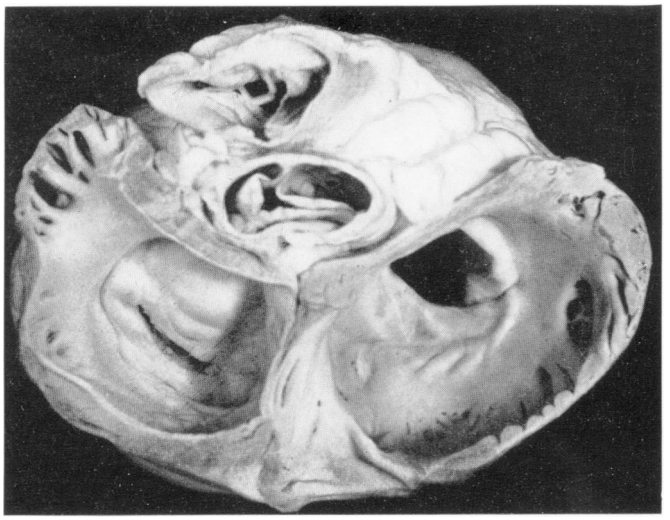

Fig. 1.

Heart cut transversely, showing all the heart-valves. The mitral valve is grossly stenosed, and the valve's cusps thickened; the tricuspid valve shows a mild degree of stenosis.

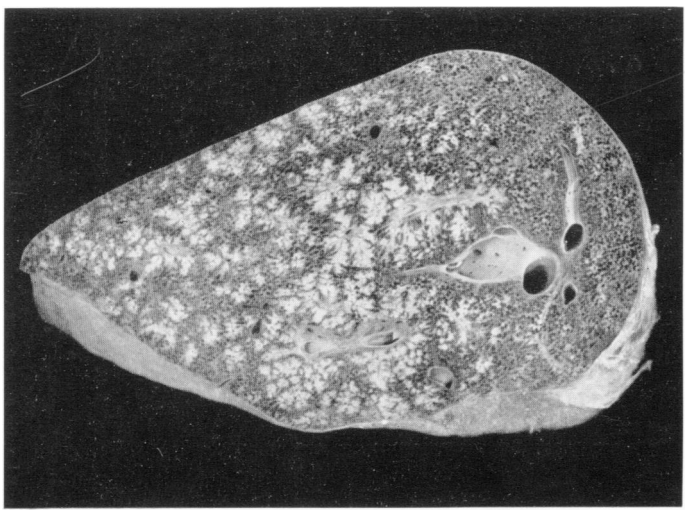

Fig. 2.

Section of liver shows a pattern of pale areas of regenerating liver lying in a background of a nutmeg pattern of chronic venous congestion.

Photographs by Mr. D. McA. Mehaffey.

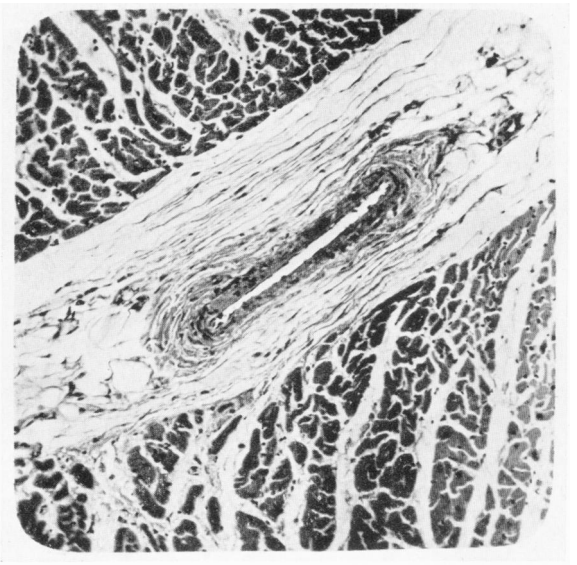

Fig. 3.—Histology of Myocardium.

Paravascular fibrillar fibrous scars lying in relation to a branch of the coronary artery indicates a healing Aschoff node.

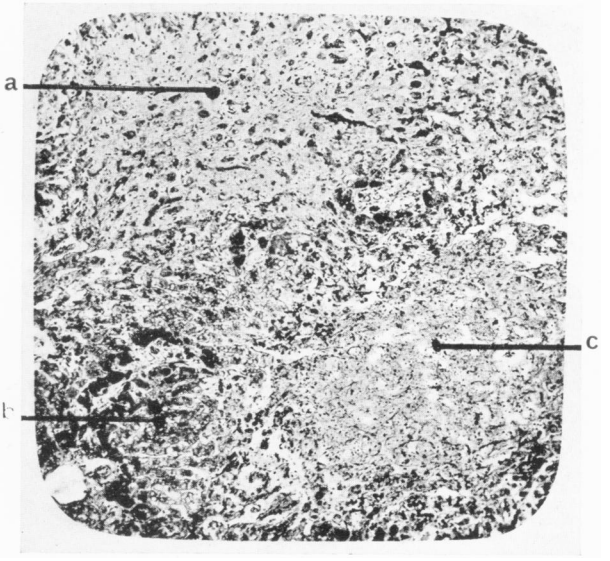

Fig. 4.—Histology of Liver.

(a) Marked fibrosis with evidence of liver regeneration.
(b) Normal liver tissue.
(c) Blood lake in centre of lobule.

Photographs by Mr. D. McA. Mehaffey.

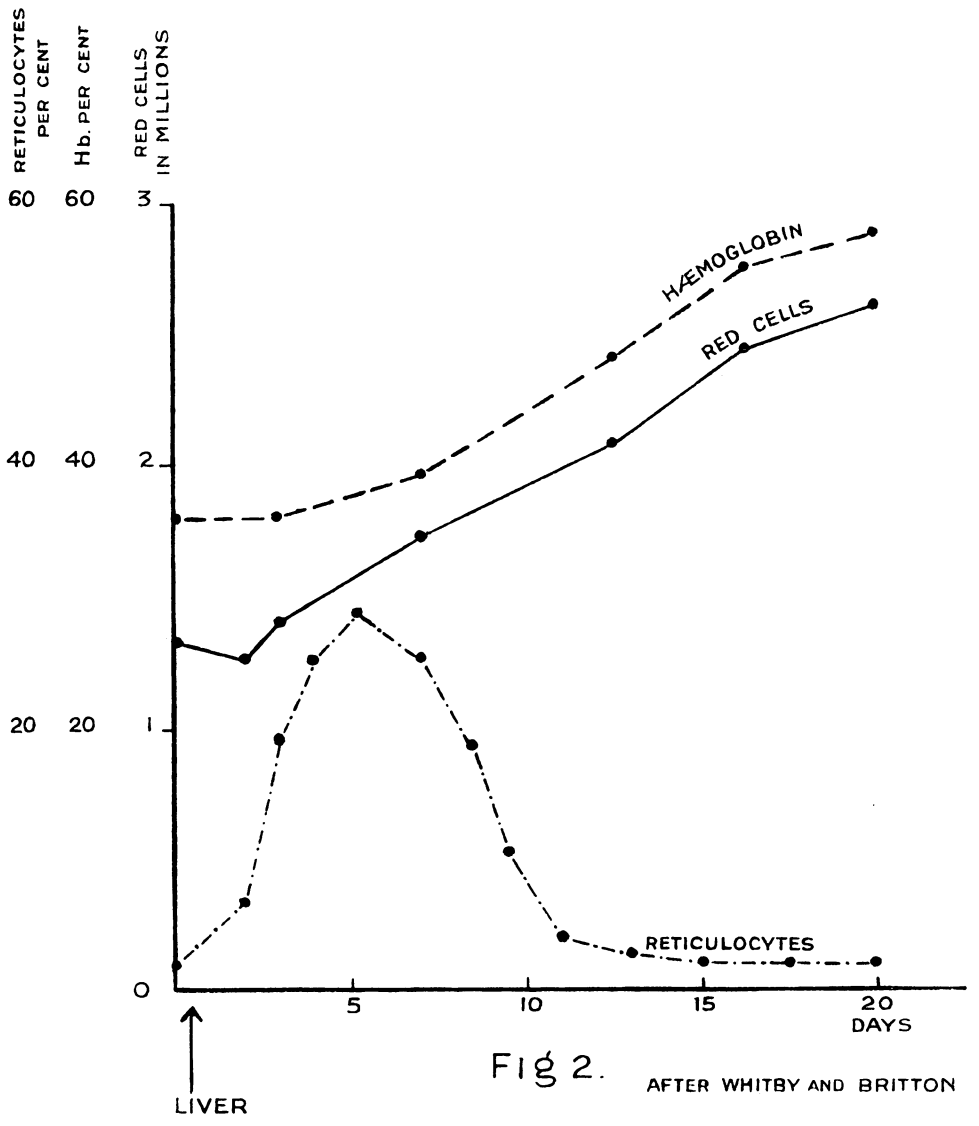

Fig 2. AFTER WHITBY AND BRITTON

and it became obvious then that the disease was progressing towards a fatal termination.

She was still receiving intramuscular liver extract on alternate days, and on 7th January a small transfusion of 200 cc. of blood was given. This produced a temporary feeling of well-being, but no lasting improvement, and the red-cell count fell from 2,320,000 before the transfusion to 1,600,000 four days later.

On 16th January she suddenly developed a sore throat and pyrexia of 103°F., and a throat-swab produced a pure culture of streptococcus hæmolyticus. Perhaps with the aid of this streptococcal infection her spleen now became just palpable.

She had always been melancholic, and two days after her temperature rose she began to exhibit severe mental symptoms of the nature of a confusional psychosis, for which it was necessary to give paraldehyde per rectum. She began to look very old and very ill, and on the sixth day of the pyrexia she passed into a stupor and died, two and a half months after admission to hospital.

As the relatives objected, no autopsy was performed, but a sample of bone marrow was obtained post-mortem which gave a positive Perl's prussian blue reaction for iron.

Discussion.

A case of macrocytic hæmolytic anæmia is described which seems identical with the acquired form of acholuric jaundice as recorded by Widal, Abrami, and Brulé in 1908. The syndrome differs from the congenital form of acholuric jaundice in many ways which this case well exemplifies. There is no familial history, it is more severe, and mortality-rate is greater. Frequently the spleen is smaller and spherocytosis is generally less or absent. (In this case microcytic hyperchromic cells were seen which might have been spherocytes, but their mean corpuscular average thickness was not investigated.) The diameter of the red cells may be increased so markedly that the blood picture closely resembles that found in pernicious anæmia (Kremer and Mason, 1936).

With regard to the close resemblance which this case at its inception bore to pernicious anæmia, the following points made by Lescher and Osborn (1939) are worth noting :—

"In pernicious anæmia, during the first few days of adequate treatment, there is an increase in the reticulocyte count. The extent of the reticulocyte response has been shown by Minot, Cohn, Murphy, and Lawson (1928) to be proportional to the depression in the number of erythrocytes. This increase, however, is only temporary, the number of reticulocytes falling within a few days, when the number of erythrocytes starts to rise (fig. 2). A persistently raised reticulocyte count and low red-cell count are incompatible with pernicious anæmia. . . . Finally, the therapeutic test of giving a potent liver extract in adequate amounts over a suitable period failed to cause any improvement."

Other finer hæmatological points against the diagnosis of Addisonian anæmia in this case were :—

1. There was no ovalocytosis of the macrocytes so characteristic of pernicious anæmia.
2. There was no leucopenia.
3. The presence of myelocytes indicated some degree of bone-marrow irritation, and there was a shift to the left in the polymorphonuclears. There were, however, no macropolycytes, so that this was not part of a dissociation effect such as is occasionally found in pernicious anæmia.
4. Platelets were small, plentiful, and in clumps, and not large and single, as in pernicious anæmia.

Thompson (1936) and Whipple (1937) recommend that hæmolytic anæmia be divided into two major groups, "typical" and "atypical." The typical group consists of familial acholuric jaundice which has definite diagnostic criteria — a chronic disease of long duration and of relative mildness with acute exacerbations; chronic variable jaundice, anæmia with evidence of blood regeneration, spherocytosis, and splenomegaly. The characteristic feature of the typical hæmolytic anæmias is the presence of spherical microcytes.

The atypical group includes acquired acholuric jaundice, and is heterogeneous except for the uniform absence of spherocytosis. In some cases the cause is known (infections, chemicals, etc), in others none can be found during life or at post-mortem.

This classification is unsatisfactory, but is useful in the absence of exact knowledge of the mechanism of hæmolysis in idiopathic hæmolytic anæmias—whether it is the result of congenital and acquired defects in the red cell or due to the presence or advent of hæmolysins.

I wish to acknowledge my indebtedness to Dr. S. I. Turkington for permission to publish this case, which was under his care, and to thank Professor W. W. D. Thomson and Professor J. H. Biggart for their encouragement and help in preparing this paper.

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GENITO-URINARY INFECTIONS

A fourth edition of the booklet on DAGENAN (M & B 693) in the treatment of genito-urinary infections has recently been sent to us.

This drug is now widely employed in gonorrhœa. In the course of a discussion concerning the treatment of venereal diseases in the B.E.F., it was stated that "The treatment of gonorrhœa with M & B 693 has shown that in all but very rare instances complete cure is achieved in three weeks." The possibility of treating cases at the front brought forward the opinion that "The proved efficiency of M & B 693 in the treatment of gonorrhœa renders the evacuation of such cases to the base quite unnecessary."

The latest publication on DAGENAN contains a note on the treatment of gonorrhœa in general practice. A copy will be sent, we understand, to any medical practitioner on request to Messrs. Pharmaceutical Specialities (May & Baker), Ltd., Dagenham, England.

THE STUDY OF FORENSIC MEDICINE

THE appearance of the second edition of "Recent Advances in Forensic Medicine" once more reminds us of the lack of proper attention to this subject in Northern Ireland. Neither the Northern Ireland Government nor the Belfast City Council seems to appreciate the enormous importance of this subject to the community. The University, on its part, depends on the Professor of Pathology to give a few lectures on the subject, but for efficient teaching, theory and practice go hand in hand, and as this is essentially a practical subject our students are deprived of the practical experience so important in their future work; the Government on its part, when an important case turns up, is obliged to send the "exhibits," "clues," etc., to Edinburgh or other cross-channel town for help and advice, as was done in a recent murder case. The loss of time, which is an important factor in these difficult cases, is inestimable, and the sooner the present unsatisfactory arrangement is changed the better.

The University from time to time has considered the desirability of creating a Professorship in Forensic Medicine, but so far, mainly due to lack of funds, nothing has been done. It seems strange to us that a working arrangement has not been made between the University, the Government, and the City Council, to the benefit of all three, whereby a Professorship of Forensic Medicine could be created, with the threefold duties of giving proper and efficient instruction in this important subject to medical students, acting as advisor in all medico-legal cases in Northern Ireland, and working as medico-legal expert for the City of Belfast.

If the responsible persons concerned in the University, the City Council, and the Northern Government could be induced to read this "Recent Advances," we feel sure that a change of attitude would be the result, from the present apathy shown in the matter.

This book deals with a number of important aspects of forensic medicine, where detailed knowledge has greatly extended in the last few years: for example, the value of ultra-violet and infra-red rays in medico-legal investigations is described, and it is clear that highly specialised training is required for the proper use of these delicate methods of crime detection. This is only one branch of medico-legal work which requires specialised study and training: others equally important are the examination of hairs, of blood and its analysis, of stains and smears in cases of alleged rape, the laboratory methods employed in the investigation of alleged "drunks" by blood-alcohol estimation, the study of blood groups for bastardy, the investigation of food and other forms of poisoning, etc.

Forensic medicine spreads a wide net for specialised knowledge, and it cannot be too strongly stressed that it is not, as is commonly supposed, confined to the muddy waters of an occasional murder.

RECENT ADVANCES IN FORENSIC MEDICINE. Second Edition. By Sydney Smith, M.D., F.R.C.P., and John Glaister, M.D., D.Sc., J.P. London: J. & A. Churchill. Pp. 264 + viii. Illustrated. Price 15/- net.

THE CARE OF CRIPPLES

NORTHERN IRELAND COMMITTEE FORMED OF THE CENTRAL COUNCIL FOR THE CARE OF CRIPPLES

It is not generally known that a Central Council for the Care of Cripples exists in London, with the Rt. Hon. The Earl of Dudley as chairman, and R. C. Elmslie, Esq., O.B.E., M.S., F.R.C.S., chairman of the Executive Committee.

The object of the Council is to organise a national scheme to deal with the cripple problem as a whole throughout the United Kingdom, and local committees have been established in various centres in England and Scotland since 1919.

A representative of the Central Council has been in Northern Ireland for some months, and a Northern Ireland Committee in connection with this important work has been formed.

A preliminary meeting was held in the University on Monday, 11th March, 1940, under the chairmanship of the Vice-Chancellor of Queen's, Mr. D. Lindsay Keir, with representatives of the Government, the Belfast City Council, the Cripples' Institutes, the Belfast Hospitals, and other interested bodies, from which the Northern Ireland Committee was formed. An Executive Committee was also formed, consisting of the Vice-Chancellor of Queen's University (chairman), Mr. H. P. Hall, Mr. M. J. Lavery, Mr. S. T. Irwin, Mr. Archibald Irwin, and Mr. W. A. Cochrane, with Miss Seeds (the local representative of the Central Council) as secretary.

The formation of this local committee brings Belfast into line with Scotland, where two centres are already at work, one in Glasgow and one in Edinburgh. The work of the local committee will cover the counties of Antrim, Down, Armagh, Fermanagh, Tyrone, and Londonderry, and the County Boroughs of Belfast and Londonderry.

The committee is faced with a number of difficult problems in organising the care of potential and actual cripples in Northern Ireland. It is not merely a question of seeking out and treating children suffering from tuberculous disease of bones and joints; it includes the treatment of congenital deformities, the deformities following such diseases as infantile paralysis, and men and women crippled as the result of accidents in our shipyards, our mills, and our factories, and on our public highways, all of which require treatment by surgeons with highly specialised knowledge if they are to be brought back to a physical condition to enable them to return to productive employment.

The first problem is to devise machinery for bringing potential patients into contact with specialist orthopædic surgeons in the early stages of disease, which if not given prompt and efficient treatment will produce cripples unable to carry on wage-earning work *under normal conditions*. The training of actual cripples will

be, it is true, a branch of the work which is already being done so ably by the Cripples' Institute on the Donegall Road. It is the intention of the committee to seek the co-operation of the Institute in continuing their work.

To solve this first problem it has been suggested that one means of approach might be the appointment of nurses specially trained in orthopædic work, to seek out suitable early cases and report them for treatment at special centres. Such appointments appear to be unnecessary, for it is the experience of general hospitals, when specialised clinics are created, that patients seem to "find themselves," and in a short time the clinics are overflowing, and accommodation has to be extended. Another suggestion is a system of notification on the same lines as certain diseases are notified to the M.O.H. of Belfast and of other centres. School medical officers and dispensary doctors throughout the province would be important links in establishing contact between patients and surgeons.

The second great problem is to find accommodation for treating potential cripples, especially those suffering from tuberculous conditions which require prolonged institutional treatment. The general hospitals do not cater for patients suffering from such conditions as tuberculous spines, hips, and other joints. In addition, they are not for the most part even situated in localities in any way suitable for modern methods of open-air treatment; and with long waiting-lists of acute and other short-term patients they cannot allocate beds for tuberculous and long-term patients, the majority of whom cannot be treated as out-patients. The great difficulty of the committee will be to find institutional accommodation for such patients, and the co-operation of Graymount, Whiteabbey, and the county infirmaries would be essential for any scheme possible under existing conditions, unless means can be found to build a suitable central institution in a healthy, sunny locality where these unfortunate sufferers can be sent for the prolonged rest and treatment essential if they are not to become permanent cripples.

Long-term residence of children in institutions raises the ancillory problem of educational facilities. Boys and girls require mental as well as physical treatment, and arrangements must be made for their education, so that when they return to their homes they will be no less equipped mentally for the struggle of life than their brothers and sisters.

Clinics for out-patients can be created in connection with the existing hospitals and infirmaries, and useful work can be performed there in treating certain types of patient, in providing after-care for patients who have completed a course of hospital treatment, and in selecting and recommending patients for admission to the institutions available.

If the county infirmaries and other institutions agree to co-operate in the scheme, the appointment of a specialist orthopædic surgeon would be essential. Such a surgeon would visit the infirmaries and clinics throughout the Six Counties, advising and, where desired, giving specialised treatment; such a surgeon would also act as consultant in cases of orthopædic conditions both resident in the institution and in the out-patient clinics which might be established in connection with them.

The establishment of the Northern Ireland Committee marks an epoch in the history of the fight against disease in this country. It can serve a useful purpose, and with the £26,000 made available by Lord Nuffield for it to carry on its work, much good can ensue. But until a central institution for long-term patients is established in our midst, only a fringe of the problem can be tackled. It is hoped that some public-spirited philanthropist will arise and make such a sum of money available as would make possible the building of an orthopædic institution in some suitable locality in Northern Ireland.

THE ROYAL MEDICAL BENEVOLENT FUND SOCIETY OF IRELAND

9 College Gardens,
Belfast.

The Editor, Ulster Medical Journal.

Sir,

I can make no pretence about it—this is a begging letter, and in a double sense. First, I beg the hospitality of your columns, and here I feel sure of my welcome because you have been generous in the past. Secondly, I beg the charity of your readers, and here I am perhaps a little less confident.

I am happy to say that many of those who read this letter are already old friends of the charity for which I beg, and to them I send on its behalf my warmest thanks.

To them and many others I have recently sent the annual report of the Royal Medical Benevolent Fund Society of Ireland. The following figures have been abstracted from it :—

	Subscribers	Total Amount
Belfast and Antrim ...	164 ...	*£193 13 11
South Armagh ...	12 ...	10 15 6
Down	18 ...	15 5 6
Fermanagh	23 ...	*36 2 6
Derry	34 ...	31 6 6
Tyrone	37 ...	18 13 6
	124	£112 3 6
		193 13 11
		£305 17 5

*These sums include special donations and collections at medical meetings.

The establishment of the Northern Ireland Committee marks an epoch in the history of the fight against disease in this country. It can serve a useful purpose, and with the £26,000 made available by Lord Nuffield for it to carry on its work, much good can ensue. But until a central institution for long-term patients is established in our midst, only a fringe of the problem can be tackled. It is hoped that some public-spirited philanthropist will arise and make such a sum of money available as would make possible the building of an orthopædic institution in some suitable locality in Northern Ireland.

THE ROYAL MEDICAL BENEVOLENT FUND SOCIETY OF IRELAND

9 College Gardens,
Belfast.

The Editor, Ulster Medical Journal.

Sir,

I can make no pretence about it—this is a begging letter, and in a double sense. First, I beg the hospitality of your columns, and here I feel sure of my welcome because you have been generous in the past. Secondly, I beg the charity of your readers, and here I am perhaps a little less confident.

I am happy to say that many of those who read this letter are already old friends of the charity for which I beg, and to them I send on its behalf my warmest thanks.

To them and many others I have recently sent the annual report of the Royal Medical Benevolent Fund Society of Ireland. The following figures have been abstracted from it :—

	Subscribers	Total Amount
Belfast and Antrim ...	164 ...	*£193 13 11
South Armagh ...	12 ...	10 15 6
Down	18 ...	15 5 6
Fermanagh	23 ...	*36 2 6
Derry	34 ...	31 6 6
Tyrone	37 ...	18 13 6
	124	£112 3 6
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The Medical Directory shows that there are 1,246 doctors in Ulster (547 live in Belfast). Of these, the names of more than 950 do *not* appear on the list of subscribers.

Suppose that each of these 1,246 doctors earns £500 per annum, their total income would be £623,000. As you have seen, Northern Ireland gives £305 a year—less than half a farthing in the pound.

Even allowing for the undisputed fact that this total of 1,246 includes the recently qualified, and some ladies who have deserted medicine for matrimony, there are too many who have hitherto failed to appreciate how helpful they could be.

The annual report gives well-deserved praise to County Fermanagh and its indomitable secretary, Dr. Leonard Kidd. Every practitioner in Fermanagh gives a guinea a year, and in addition a table collection of £9. 17s. 6d. was given. If only we would *all* follow the flag of Fermanagh. A guinea a year from every man and every woman who is earning his or her living in medicine in Northern Ireland! What a lead it would give to the whole country and even to our brethren across the Channel, and how much more worthy of the old Ulster traditions of heart and hand. Best of all, what a difference it would make to the poor ladies and the children for whom the Fund exists.

Take the case of Belfast and County Antrim, which as honorary secretary I know best. Last year we gave £193 odd, and the grants came to £232, a deficit of £40. This sum of £232 was spread over more than twenty persons, since dependants must be included. Surely Belfast and County Antrim doctors can do better than this for the widows and orphans of their brothers.

Some of those whose names appear on this year's list of subscribers have been lost to us for ever. May I suggest that their successors maintain their subscriptions "in memoriam"?

When last I addressed you on this subject, Sir, the war clouds gathered, and I pointed out that war makes penury harder and a pound buys less.

Can we not make life more bearable for those who suffer?

I am, Sir,

Yours faithfully,

ROBERT MARSHALL.

REVIEW

ILLUSTRATIONS OF BANDAGING AND FIRST-AID. By Lois Oakes, S.R.N., D.N. 1939. Edinburgh: E. & S. Livingstone. Pp. 246 + vii. Price 6/- net.

The publication of books on bandaging and first-aid is legion, but when a really good one appears it is more than welcome. Miss Oakes' book is of this category, and it appears to the writer of this note to be the most practical book on bandaging he has so far seen. In contains 290 photographs, showing the various kinds of bandages and their application. The book will undoubtedly have the extensive circulation which it deserves.

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Acne Vulgaris

By REGINALD HALL, M.B., B.CH.

From the Skin Diseases Department, Royal Victoria Hospital, Belfast

THE object of this paper is to set out shortly some of the etiological factors in acne vulgaris, and to indicate the bearing which they have on treatment.

It is, unfortunately, often regarded as a condition about which nothing is known, and to which treatment makes very little difference; and patients are too frequently told that they must just wait until it disappears.

Acne is so common as to need no description. Its onset is, as a rule, synchronous with the onset of puberty, and if untreated, it remains with remissions until 25 to 30 years of age, then possibly to be followed by rosacea.

Within recent years it has become recognised by most dermatologists that acne is merely a phase in what has been called "the seborrhœic diathesis." A good deal of prominence has been given to this recently, but it may be helpful to recall in an abridged form the description of it given by Barber :—

"The skin as a whole appears thickened, coarse, and dirty, particularly in dark-complexioned persons; the horny layer is improperly formed and hypertrophied, the pilosebaceous follicles are enlarged and patulous, and either exude visible droplets of oily sebum, or are filled with solid plugs—the so-called seborrhœic cocoons—containing countless acne bacilli; there is often visible hyperidrosis in certain situations; the scalp after puberty is greasy, covered with adherent dandruff which swarms with the pityrosporon and the staphylococcus albus, and the hair is dank and lifeless. To this picture must be added the infective complications—the comedones, pustules, and nodules of acne, the various forms of seborrhœic dermatitis, the superficial pustules due to the staphylococcus albus, and the deeper folliculitis of sycosis and boils due to the staphylococcus aureus. In middle-life, rosacea occurs, which sooner or later becomes complicated by a superadded infection of the flushed area of the face with seborrhœic organisms. Gingivitis and dental caries are often seen, and chronic naso-pharyngeal catarrh with frequent exacerbations is almost invariably present in severe cases of the seborrhœic state.

"True flatulence, as distinct from ærophagy, is common, due apparently to the fermentation of carbohydrate, since it is usually relieved by the exclusion of soft carbohydrate foods from the dietary.

"The urine of seborrhœics has been studied by several observers. The alkali tolerance is, as a rule, markedly raised, so that very large doses of alkali are required to render the urine alkaline."

It will be appreciated, therefore, from this description, which holds for practically all cases of marked acne, that there is a general constitutional disorder underlying the condition; and the recognition and adjustment of this should be as much a part of treatment as the local applications to the "spots."

Apart from this, perhaps the most striking feature of acne is its relation to

puberty, because it does not usually occur during the pre-adolescent period, in eunuchoid states, or in senility. At puberty, along with the various other changes which occur, the sebaceous glands hypertrophy and their secretion increases; occlusion of the orifices of the ducts readily takes place, and so the typical essential lesion of acne occurs. Such lesions are almost entirely limited to those situations where the sebaceous glands are naturally largest, e.g., face, shoulders, centre of chest and back. They do not occur on the limbs where the glands are small, except in those cases where the orifices of the glands have been subjected to constant irritation and occlusion by substances such as mineral oils, e.g., in "oil acne." They never occur on the palms or soles, where the sebaceous glands are absent, though sweat-glands occur here in profusion. They do not occur on the scalp, nor in follicles which contain a well developed hair; and in this connection Sharlit has put forward the interesting theory that as the lesions only occur in pilo-sebaceous follicles where the hair-growth is vestigal, such follicles, when stimulated by the hormones of puberty, attempt to produce hair, but succeed in producing only hyperkeratosis of the orifices which occludes them. Variations in degree of acne are explained by Bloch as being merely due to the follicular apparatus of the skin differing individually in its sensitivity to the hormone.

Although much research work has taken place, both in this country and in America, into the hormonal aspect of acne, little advance has been made; and no uniform results are yet obtainable by hormone therapy.

That the pilo-sebaceous follicles in the majority of cases are very sensitive to bromides and iodides is commonly shown by the frequent occurrence of anceiform lesions following the administration of these drugs; and on being given to patients with acne, even in minute doses, exacerbation of the lesions almost invariably occurs within a few days. The importance of this observation on treatment will be clear when it is remembered that some articles of diet contain these substances in amounts sufficient to aggravate existing lesions, e.g., some varieties of table-salt are iodized; and white bread may have been treated with an "improver" such as potassium bromate, which in the process of baking is converted into a bromide.

It has been shown that carbohydrate in large quantity has a hydrating effect on the skin which encourages infective processes, and certainly the majority of cases of acne take a high carbohydrate diet, which is a factor in maintaining the seborrhœic state, and encourages the formation of pustules and cystic lesions.

As regards the bacteriological aspect of acne, it may be recalled that the normal flora of the skin consists of :—

(1) The pityrosporon of Malassez—a yeast-like organism.

(2) The acne bacillus—a diphtheroid.

(3) The staphylococcus epidermidis albus.

The first of these normally inhabits the stratum corneum, while the other two are to be found in the pilo-sebaceous follicles. On suitable soil, such as is presented by the skin of the seborrhœic, these organisms multiply markedly in their natural

situations, and are to be found in great profusion in various lesions, e.g., the pityrosporon is present in the greasy scales of dandruff and seborrhœic dermatitis, acne baccili occur in the lesions of acne vulgaris, and the staphylococcus epidermidis albus is found in association with the other two. As to their true significance, however, there has been much controversy. Experimentally it has not been possible to produce the lesions with which they are generally associated; and theoretically, therefore, it might be expected that vaccine therapy would not be of much value. In practice this has been found by most observers to be true.

TREATMENT.

While local measures are still the most important part of treatment, it may be appreciated from the foregoing that the best results are only to be obtained when general constitutional abnormalities are also corrected.

(1) GENERAL MANAGEMENT : Chronic constipation is often an associated condition, and the bowels should be regulated by mild laxatives. Fresh air and exercise are important, as sedentary occupations tend to make the condition worse. It is a common observation, for example, that in the weeks prior to examinations when little exercise is being taken, new lesions tend to form, and existing lesions become more marked. Menstrual abnormalities should be corrected.

(2) DIET : Carbohydrate should be restricted, especially white bread, cakes, pastries, chocolate, and cane sugar, all of which tend to promote fermentative processes in the intestine. Pig-fat should be avoided. Iodized table-salt has been shown to be a serious offender.

The diet should consist chiefly of raw and cooked fruits and vegetables, meat, fish, eggs, milk, and butter and crisp farinaceous foods. All starchy foods should be thoroughly masticated, and an adequate intake of vitamins should be ensured.

(3) FOCAL SEPSIS : As this is often present in teeth, tonsils, or sinuses, suitable corrective measures should be adopted.

(4) DRUGS : (a) *By mouth*—owing to the marked acidity of the urine, a simple alkaline mixture should be given. Cystic cases are often improved by liq. arsenicalis m. iii, t.i.d., which may be given for about one month.

(b) *By injection*—in frankly pustular cases, staphylococcus vaccine, either stock or autogenous, or toxoid, occasionally helps; but the ordinary case of simple acne is rarely benefited by acne vaccine.

(c) *Locally*—the object of local applications is to cause mild exfoliation and reduction in the amount of sebaceous secretion. For this reason, the basis of most local applications is sulphur. It is antiseptic, has some action in reducing the secretion of sebaceous glands, and in the presence of alkali in the tissue juices, it forms a soluble sulphur alkali which dissolves keratin. It may be applied either in the form of a lotion, or as a paste.

Lotion :—

Zn. Sulph.		
Pot. Sulphurat	... ...	aa dr. i. s.s.
Aq. Rosæ	... ...	ad oz. iii.

This solution should be freshly prepared frequently, and should be applied to the affected parts with cotton-wool as often as desired.

Paste :—

This should consist of sulphur and resorcin in such strength as to cause mild exfoliation. As a rule, pastes are much more effective than lotions, but may cause some discomfort if too concentrated. They may be made up as follows :

	Weak	Medium	Strong
Resorcin ...	dr. ½ ...	dr. i ...	dr. ii
Sulph. ppt. ...	dr. ½ ...	dr. i ...	dr. ii
Zn. Oxid. ...	dr. iii ...	dr. ii ...	—
Lanolin ...	dr. i ...	dr. i ...	dr. i
Vaselin ...	dr. iii ...	dr. iii ...	dr. iii

Patients should wash the affected areas thoroughly with warm water and sulphur soap at night, remove as many blackheads as possible with a blackhead extractor, then apply the paste, and wash it off the next morning. During the day the lotion may be applied.

After four or five days of such treatment there is usually a good deal of erythema, and the skin is dry and exfoliating. Cold cream may now be applied, and when the erythema has disappeared, a good deal of improvement should be seen. The treatment may be repeated.

When the skin has become clear the lotion is all that is required locally to keep the condition in subjection.

(5) PHYSIO-THERAPY : Exfoliation may also be obtained by erythema doses of ultra-violet light, and this method is useful when there are many lesions involving the trunk.

Small doses of X-rays are of great value in reducing the sebaceous secretion, but are not as a rule used before the age of twenty, because when used alone the condition is liable to recur, and frequent X-ray therapy is fraught with danger.

Large cystic lesions may require incision, but cauterising with pure carbolic after incision, as is sometimes done, is unnecessary.

Associated dandruff should be energetically treated. For this the following methods are usually successful :—

Severe cases will require an ointment—

Sulph. ppt.	gr. x
Ac. Salicyl	gr. xv
Paraff. Moll. alb	dr. ii
Ol. Cocois Nucifer	ad oz. i
Perfum.	q.s.

This should be thoroughly applied to the scalp each night.

For mild cases, a lotion such as the following may be found sufficient :—

Resorcin Monoacet.	...	...	...	gr. xv
Ac. Salicyl.	...	...	...	gr. xv
Hydrarg. Perchlor.	...	...	...	gr. $\frac{1}{6}$
Aq. Coloniensis	...	...	...	q.s.
Spt. Vini. Indust.	...	...		ad oz. i

If this is found too drying, Ol. ric. m.v.—x may be added.

The lotion and pomade should not be used together on account of the reaction which occurs between the mercury and sulphur.

A shampoo may be used once or twice weekly :—

Ol. Cad.	...	...	...	dr. i
Thymol.	...	...	...	dr. $\frac{1}{2}$
Sap. Moll. Vir.	...	...	...	oz. iii
Spt. Vini. Indust.	...	...	...	ad oz. iv

Most cases of acne will improve considerably if the above measures are thoroughly carried out; and in any event, the grossly disfiguring nodular type may be prevented. It is true that, generally speaking, acne tends to disappear in the third decade, but by this time much permanent damage may have been done, and the psychological effect on young girls must be not inconsiderable. It is, therefore, well worth while encouraging them to co-operate in the treatment, so much of which depends on their own efforts.

REVEIW

PATHOLOGICAL HISTOLOGY. By R. F. Ogilvie, M.D., F.R.C.P. With foreword by A. Murray Drennan, M.D., F.R.C.P., Professor of Pathology, Edinburgh University, and 220 Photomicrographs in colour by T. C. Dodds. F.R.P.S., F.I.B.P. Pp. 332+x. Price 27/6.

This volume, designed as a companion book to the standard textbook of pathology, fills an important gap in student textbooks. In its arrangement it follows the usual one of the student textbook, and divides the subject into general pathology and special diseases of the various anatomical systems. Whilst on the whole we object to too sharp a division between the study of gross pathology in relation to the patient's disease and the study of microscopical pathology, the present textbook, if utilised as the author desires it to be in conjunction with the students' routine pathology, will certainly serve its purpose. The wealth of illustrations, especially where these appear in the same colour-scheme as the prepared section, will render microscopical work much easier and should serve as a useful means of revision both for the graduate and under-graduate. Many of the illustrations are perfect, and the main objection which can be raised is that sometimes the selection of the microscopical field and the power of magnification could have been improved. Thus without the accompanying legend, figs. 58, 116, and 124 are relatively unspecific. It also seems unnecessary, when more common lesions are omitted, to figure a malignant hæmangio-endothelioma, and many pathologists will disagree with the important rôle assigned to the Kupffer cell as the origin of this type of rare tumour. In the section on tuberculosis, no differentiation is made between the "hard" and "soft" miliary tubercle, while some of the more common ovarian tumours could have been illustrated with advantage.

However, these are minor criticisms, and the author is to be congratulated on this production. Its appearance opens up a new field in medical illustration, and it will serve for many years as an important guide to students.

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Polyarteritis Nodosa

By JAMES McMURRAY, M.B., B CH.

Institute of Pathology, Queen's University, Belfast

POLYARTERITIS NODOSA, as its name implies, is an inflammatory lesion involving the whole three coats of the arterial system in a nodal manner. The causation of this is unknown, but its peculiar affinity for widespread involvement of the smaller arteries results in a multiplicity of clinical signs and symptoms, and leads to confusion and difficulty in diagnosis. The more severely affected organ dominates the clinical picture, and the other inexplained signs and symptoms vague in character are perhaps labelled "rheumatism," etc. Thus it would seem that some cases are missed. Sometimes the vessels involved are so small that the condition can only be recognised after careful microscopical examination of affected tissues. This further increases the difficulty of clinical recognition of the disease, and would suggest a possible cause for its relative rarity. Exemplification of these facts is seen in the following case, which was recognised only on histological examination post-mortem.

The patient was a female aged 67 years of age.

She was admitted to Royal Victoria Hospital with the history, that above five years previously she began to have an increased frequency of micturition and was informed that she had kidney trouble. Subsequent to this the frequency of micturition did not clear up.

About eight months before her admission she received vaccine injections for rheumatoid arthritis.

For the past four months she complained of breathlessness on exertion and slight swelling of her ankles; and spots before her eyes for about three months.

On clinical examination in the hospital she was found to be an elderly, frail, emaciated woman with dry skin and typical rheumatoid changes of the joints of her fingers; her ankles were œdematous and eyelids puffy. Her artery-wall felt thickened, heart was slightly enlarged on percussion, blood-pressure 200/110. Some fluid was detected at the bases of both lungs. Temperature 99°. Pulse 100.

Wassermann reaction was negative: blood urea was 34 mgm. per cent.: a catheter specimen of urine contained albumen, microscopically showed blood-cells, hyaline casts, and gave a pure growth of B. coli. The specific gravity range of the urine was=6 a.m. 1018; 9.30 a.m. 1012; 10 a.m. 1012; 11 a.m. 1012. An electro-cardiogram showed a flat T wave in leads i, ii, and iii; P wave inverted in lead ii, P wave prominent in lead iii; a "W" type of S wave in lead iii—such findings suggested myocardial change.

Pulse gradually became weaker and patient died.

During her stay in hospital, temperature fluctuated between 97 and 100: her pulse 88—114, and she received citrates and Mist. Sod. et Gent.

Post-mortem examination.—The naked-eye findings were:—

Some œdema of feet and ankles.

The pericardial sac contained about 3 oz. of straw-coloured fluid, and numerous greyish-white flecks were seen on the pericardium.

Both pleural sacs contained a small amount of free fluid. The right sac showed a few fine adhesions.

The peritoneal sac showed long-standing adhesions between the ileum, both sides of bladder and the uterus, and the pelvic colon. This formed a fibrotic mass and caused constriction of both ureters.

The heart showed slight hypertrophy of the left ventricle. The right auricle, including its appendage, appeared healthy. Tricuspid valve was somewhat fibrotic. The right ventricular endocardium showed a diffuse greyish streaking, the myocardium was thin. The pulmonary valve and artery appeared normal. There was a patent foramen ovale, which was guarded by a valve. The left auricle appeared slightly hypertrophied and its endocardium thickened. The mitral valve showed some thickening and fatty change. There was hypertrophy of the left ventricular muscle, the endocardium was greyish in patches. The aortic valve was the seat of fatty change and was competent to water-pressure. The aorta showed early intimal fatty change. The coronary arteries were patent and free from pathological change at their ostia.

The left lung was air-containing and greyish-pink. On section it was congested. There was a small calcified sub-pleural nodule on the anterior and upper portion of the lower lobe.

The right lung was also congested, especially in the lower lobe, where it was slightly œdematous. The bronchi were congested.

The liver was small and showed capsular thickening. On section, its substance was reddish-brown in colour and engorged with blood. There was a small yellowish nodule in the right lobe, just beneath the capsule, containing necrotic material surrounded by a fibrous wall and about the size of a large pea.

The gall-bladder was filled with yellowish bile and its mucous membrane was intact. The bile-ducts were patent.

The stomach, duodenum, and greater portion of the small intestine appeared healthy and contained bile. The lower ileum at three places was involved in dense adhesions and attached thereby to a fibrotic mass in the left side of the pelvis in the region of the left ovary. As far as the pelvic colon and the large bowel showed no lesion, but the pelvic colon was surrounded by the mass of fibrous tissue described above. The lumen of this part of bowel contained purulent fæcal debris, and showed at one part an annular ulcer with hard thickened edges and was suggestive of an annular scirrhus carcinoma. The appendix lay free in the peritoneal sac. The involvement of the colon by adhesions suggested an old diverticulitis with a chronic colonic ulcer which became malignant.

The left ovary was involved in this mass of fibrous tissue and was not recognised. The left fallopian tube was fibrotic. The uterus was normal in size, its cavity contained polyp which protruded through the cervix into the vagina by a hæmorrhagic bulbous end. The vagina was smooth and atrophic. The right fallopian tube was normal in size. The right ovary was the seat of a small thick-walled cyst.

The kidneys presented similar appearances. Externally they appeared somewhat larger than normal and felt soft and boggy. The capsule showed patchy adhesions and was stripped with difficulty, and left a purplish-red irregular surface with numerous pinhead yellowish-grey spots. On section it was seen that the renal cortical substance was much diminished, was reddish coloured, and had scattered throughout its substance numerous yellow-grey spots, pinhead in size. The medulla, also greatly diminished in amount, was congested. The smaller calyces contained purulent urine. The renal pelvis was grossly dilated in major and minor calyces and showed submucosal hæmorrhages. Both ureters were grossly dilated—about two inches in circumference as far as the fibrotic mass in the pelvis at the base of the bladder. At this point a probe could be passed through the vesical opening of the right but not through that of the left.

The urinary bladder was distended with purulent urine. Its mucosa was hæmorrhagic and gangrenous, being coated with a thick greyish-green purulent necrotic exudate.

Iliac and para-oartic lymph-glands show slight enlargement.

The spleen was small. On section its pulp was red and trabeculæ easily seen.

The pancreas was normal in size and in naked-eye appearance on section. Its ducts were patent.

Both suprarenals appeared normal on microscopical examination, except that the left was adherent to the kidney.

The aorta was the seat of atheromatous plaques, some of which have become calcified, with resultant reduction in the elasticity of the vessel wall.

Oesophagus, larynx, and trachea showed no gross lesion.

Thyroid presented the normal appearances externally and on section.

The histological findings were as follows:—

In the heart the small arteries and arterioles were involved in an inflammatory process of fairly recent origin. The medium and larger sized arterial branches of the coronaries appeared normal. In some of the smaller arteries there was an eccentric thickening of the intima by a hyaline fibrous tissue, the media of the vessel at this point was infiltrated by polymorphonuclear neutrophils, lymphocytes, and plasma cells. In some areas the lumen of the vessel had been obliterated and had been recanalised, in others it was a mere eccentric slit. The adventitia was infiltrated with inflammatory cells and showed an increase in fibrous tissue. The myocardium and endocardium appeared normal. There was an increase in interstitial fibrous tissue, chiefly perivascular and focal in distribution.

The lungs showed slight thickening of alveolar walls, suggestive of some chronic venous congestion consistent with a slowly failing heart. The arteries and arterioles in the sections examined presented normal appearances.

The liver was the seat of an infarct of fairly recent origin, in which the gross hepatic architecture could be distinguished. There was a marked increase in the fibrous stroma around some of the portal tracts, and the hepatic arteries in these showed intimal thickening by hyaline fibrous tissue and inflammatory infiltration of their media and adventitia, with patchy destruction of the muscle.

The spleen showed thickening of the sinusoidal walls consistent with chronic venous congestion. The vessels presented no foci of necrotic or of inflammatory change.

Pancreatic parenchyma was normal; its stroma had a patchy increase in fibrous tissue. Some of the small arteries showed areas of necrosis with fibrinous exudate replacing the normal cellular muscle of the media. In others the intima was thickened by a subendothelial proliferation of fibrous tissue, and their adventitia was infiltrated by inflammatory cells, most of which were lymphocytes and plasma cells.

The renal tissue was the seat of active inflammatory change of a diffuse character. The pelvic mucosa was infiltrated with polymorphonuclear leucocytes. There was an increase in the interstitial fibrous tissue of the whole organ, which was somewhat obscured by the infiltration of the stroma by polymorphonuclear leucocytes. This invasion by inflammatory cells was best marked in the region of the pelvis of the kidney and around a few glomerular units in the cortex. There were no irregular scars that would fit in with fibrous tissue reaction in long-standing pyelonephritis. The glomeruli showed, in some areas, evidence of recent infarction, and appeared necrotic with loss of the structural detail of their capillary tuft. There was no evidence of old inflammatory change in the glomeruli. The afferent arterioles in the sections examined appeared normal, but the smaller arteries showed various degrees of damage. The media in some was necrotic; in others it was fibrotic, and the walls of all vessels affected were infiltrated by inflammatory cells. The acellular necrotic material of the media on further investigation had the staining qualities of fibrin. This pink-staining hyaline material was most marked in the subendothelial zone of the vessel wall, and the cellular infiltration greatest in the adventitia and outer media. One vessel cut obliquely showed in one portion an acute necrosis with fibrinous exudate of the media and a cellular infiltration of the adventitia, whereas the rest of the vessel appeared absolutely normal. The vascular lesions also showed evidence of healing by the presence of a loose fibrous tissue thickening in the sub-intimal zone and a fibrous replacement of the muscle of the media. the subendothelial proliferation was related to medial destruction, and resulted in the production of an eccentric lumen. The large arteries appeared normal. The renal tubules contained casts, some of pus cells, others of hyaline albuminous material. The tubular epithelium was intact. The capsule of the kidney was within normal limits in structure and thickness.

The adrenal tissue was the seat of several small fibrotic areas representing old healed infarctions, but showed no evidence of recent necrosis of its parenchyma. The arterioles and small arteries were in some areas the seat of a fibrotic replacement of the media, over which areas the intima was grossly thickened by vascularised fibrous tissue. The internal elastic lamina was in this portion of the wall flatter than usual and was splitting. The remaining media showed fibrous scars as evidence of a previous infiltration with inflammatory cells. The adventitial fibrous tissue was thicker than normal. In some vessels the change was more acute in the form of a necrosis of the media with exudation of fibrin.

The colon near the ulcer and constriction showed an extension of glandular acini from its mucosa through its wall. The muscle-coat was infiltrated with small acini made up of hyperchromatic cells and supported by a flimsy stroma containing weak-walled blood-vessels. In the fibrous tissue around the acini several arterioles showed inflammatory involvement. There was an infiltration of the adventitia and outer media by a few polymorphonuclear leucocytes, many plasma cells and lymphocytes. The media itself had lost its sharp cellular pattern and was hyaline in appearance. The endothelium was more cellular than normal and the lumen greatly reduced. A small diverticulum was present, and this appeared healthy.

The aortic intima appeared within normal limits. The media showed some foci of infiltrations around the vasa vasorum near the adventitia. The small vessels in the adventitia showed marked involvement. The adventitial coats were infiltrated with lymphocytes, plasma cells, and some polymorphs. This had in some vessels involved the media, and all that apparently remained of the muscle-coat was a hyaline acellular band. The intima in most vessels also showed a proliferation which had reduced the lumen to practically a slit. The appearance was suggestive of syphilitic aortitis, but for the extent of arteritis in the adventitial vessels there is very little medial change—infiltration or scarring—which is characteristic of the syphilitic involvement. Besides this the Wassermann reaction was negative.

The thyroid presented normal histological appearances.

The urinary bladder had a necrotic mucosa and a muscle-wall slightly necrotic in a zone immediately beneath the mucous membrane. The smaller arteries in some cases showed cellular infiltration of their walls and intimal proliferation similar to that described in the other organs.

The uterine endometrium was the seat of systic change. The small arteries in the wall of the uterus showed cellular infiltration by mononuclear cells and plasma cells and intimal hyperplasia.

The ovarian tissue is atrophic. There was marked cellular infiltration of the walls of some of the smaller arteries and thickening of their intima.

Reviewing these findings, it was concluded that the following was the sequence of events in this case :—

That this old woman who died aged 67 years had developed rheumatoid arthritis (many years ago), which slowly progressed. About five years before her death a cystitis developed, probably following a seepage of B. coli through the bladder-wall from the localised pelvic peritonitis, which developed as a spread of infection from a diverticulitis of the pelvic colon. This pelvic peritonitis, which was maximum around the colon, on healing led to the constriction of ureters and partial urinary obstruction with the production of hydronephrosis. The proximity of pelvic inflammation and the interference with the physiological flow of urine into the bladder allowed the bladder infection to extend upwards into the pelves of the kidneys and produce a pyelitis. Increasing urinary stasis permitted an extension of the inflammatory condition into the renal tissue. These factors, along with the polyarteritis nodosa, were responsible for a considerable reduction in the amount of renal secretory tissue, and so, as renal reserve decreased, the blood-pressure increased. Sustained hypertension could only be maintained by a hypertrophied

heart; this compensated for renal insufficiency over a period, but the extent of its adaption was limited, and so dyspnœa on exertion and œdema of the ankles appeared as omens of a failing myocardium four months before she died. In the meantime the ulceration of the colonic mucosa near the diverticulum had persisted, and later undergone malignant change, giving an adeno-carcinoma of the annular scirrhus type. The date of onset of the polyarteritis could not be definitely stated beyond the fact that some of the lesions seen in the heart were old and fibrotic and similar to those in the kidney, where the interference with urinary secretion had been present for some considerable time. The subsequent spread of the arteritis occurred at different times, some of the nodes having been recently formed, as manifested by their necrotic and exudative character. These vascular lesions had therefore been responsible in part for the renal insufficiency, for the hypertension, and for the myocardial change, directly and indirectly. Nevertheless there was no clinical picture to suggest the diagnosis of polyarteritis nodosa, the presence of the long-standing pyelitis having been regarded as the predominating lesion and sufficient to account for the renal damage. The extreme difficulty of diagnosing clinically a case of polyarteritis nodosa is thus obvious, and it is also quite evident that many such cases must die unrecognised and remain so. That perhaps accounts for the rarity of the condition as a clinical entity.

The main features of the polyarteritis in this case were—the vessels affected were small and only recognised microscopically, the type of the lesions varied from a healed obliterated vessel which was undergoing recanalisation to a very recent acute necrosis of the subendothelial media with fibrinous exudation and acute infiltration of the vessel wall. The majority of the vessels affected have undergone healing, a few have been occluded, and resulted in small areas of infarction as seen in the liver and adrenal.

There have been no gross hæmorrhages from rupture of a vessel through the necrotic patch in the media, and in no instances was there any evidence of aneurysmal dilatation of the artery involved.

This condition was described by Kussmaul and Maier in 1866, and apart from a more detailed histological knowledge of the lesions, very little new has been added to their work. The various features of the condition have been discussed more recently by Arkin (1930) and by Harris (1939) and by Fitz (1939).

The main characteristics of it are the variation in the extent and number of organs and tissues affected by the arterial lesions, the resultant multiplicity of signs and symptoms, and so the difficulty in clinical diagnosis. Up to June, 1938, out of the 101 cases reported, only 26 had been clinically recognised as such. This difficulty probably also accounts for the relative rarity of the condition. Various etiological factors have been postulated, but none as yet definitely proven; amongst these are treponema pallidum, streptococci, virus, parasitic invasion, and allergy. All that can be said is that this is a specific infectious disease caused by some agent which has an elective affinity for arteries.

The changes in the vessels can be divided according to Arkin into four stages—(1) alterative degenerative stage, in which there is a necrosis of media with

exudation of fibrin. (2) The acute inflammatory stage, in which the vessel is infiltrated with inflammatory cells and a fibrinous exudate occurs in the media and subendothelial zone. This may lead in some cases to intimal damage severe enough to precipitate thrombosis and infarction. Sometimes aneurysmal dilatation and rupture occurs with extensive hæmorrhage. (3) Granulation tissue stage, when the exudate and necrotic muscle is removed and replaced by fibrous tissue, and any thrombus in the vessel is organised and recanalised. (4) Healed or scar tissue stage, where the vessel is represented by a mass of fibrous tissue in which irregular scattered concentric fibrils of elastic tissue may be seen with special stains, or in some less severe cases an eccentric functioning lumen may remain with evidence of localised medial fibrosis and adjacent intimal thickening. The condition may clear up and undergo healing after a slight attack, or may progress to a fatal end in a first or second attack from hæmorrhage or renal insufficiency.

This case shows features of the first and second stages in the kidney; the remaining lesions are mainly those described under stages 3 and 4.

The organs most commonly involved in this disease are the kidneys, heart, liver, muscles, peripheral nerves, and gastro-intestinal tract. Clinically one finds commonly a septic type of temperature, polyneuritis, vague pains, hæmaturia, and perhaps by a careful examination nodules in subcutaneous arteries.

In this case histologically arterial involvement was found in the heart, kidneys, adrenal, liver, pancreas, urinary bladder-wall, parametrium right ovary, and bowel-wall. No microscopical vascular lesions were seen in the lungs, spleen, or thyroid. The brain was not examined. In spite of the fact that these organs were involved, there was no clinical sign or symptom which could not have been the result of the cystitis and pyelitis with subsequent hypertension and cardiac failure, as diagnosed in hospital. Besides the presence of the urinary infection, hydronephrosis, and carcinoma of colon, there is the microscopic character of the arterial lesions, which in itself rendered clinical recognition of the complete vascular lesion impossible unless by a means of a biopsy. The involvement of the cardiovascular system may produce symptoms and signs simulating practically any common clinical condition. Involvement of gall-bladder may precipitate a syndrome surgically interpreted as an acute cholecystitis, or a submucosal vessel in the gut may rupture and produce an intestinal hæmorrhage, or a cerebral vessel may be so damaged as to produce a fatal hæmorrhage. Renal damage may result in hæmaturia and later insufficient urinary excretion. It is thus apparent that a patient with this disease may have symptoms resembling any common disease and be treated as such, or the symptoms and signs may be so varied and indefinite as to suggest double pathology. Thus in the absence of complicating conditions, it is exceedingly difficult to make a correct diagnosis, and in presence of such lesions as a urinary infection it is well nigh impossible. The absolute proof of correct diagnosis of a case of periarteritis nodosa lies in the histological confirmation. The taking of a biopsy, however, may leave a wound which takes months to heal.

Mussmaul and maier, in describing this clinical picture, said that it became the more perplexing the longer is was observed, and was one with a prognosis likely to be evident before the diagnosis could be established.

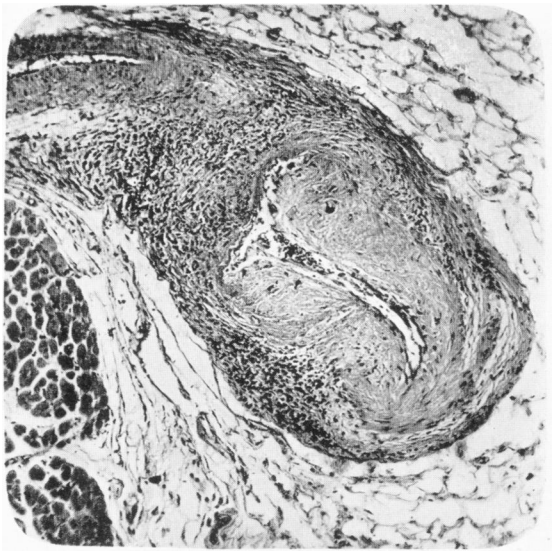

Fig. 1—Heart and coronary vessels.

There is an infiltration around the vessel and in the wall of the vessel. In the upper part, the media acellular and necrotia, a small strip of normal media is seen at the lower end of the vessel. The intima shows eccentric thickening by loosely arranged fibrous tissue. The condition is passing into the granulation stage.

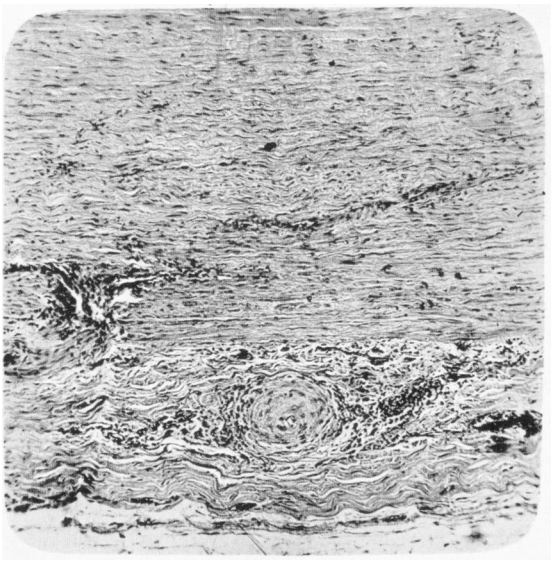

Fig. 2—Aorta.

There is an infiltration around the vasa vasorum and a marked reduction in the size of the lumen in one, which is due to the proliferation of the subendothelium. There is a slight infiltration around the unaffected vessels. The adventitia is not thickened, and there are no radiating scars, points which are against a long-standing syphilic aortitis. However, its resemblance to a recent syphilitic aortitis is striking.

Photographs by Mr. D. McA. Mehaffey.

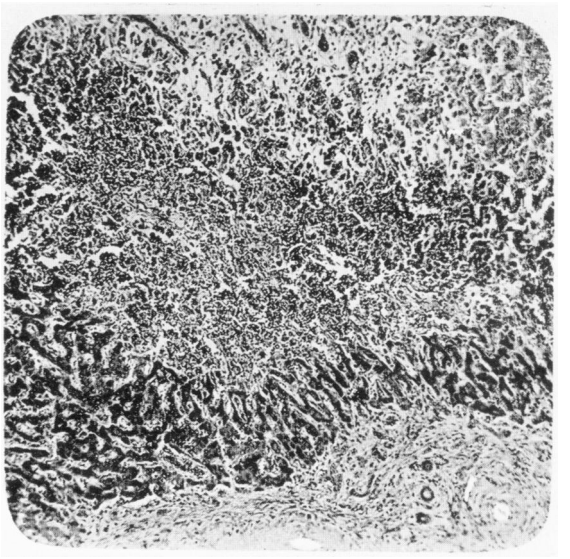

Fig. 3—Liver.

There is a liver necrosis, a zone of surviving liver tissue, and a portal tract showing an increased amount of fibrous tissue, a cellular infiltration and a hepatic artery showing a periarteritis.

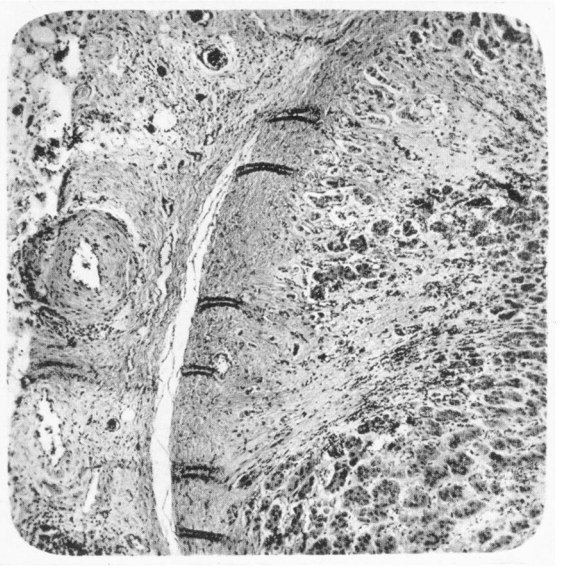

Fig. 4—Adrenal infarcts.

This is a healed small infarct of the cortical zone of the adrenal. There is a capsular vessel showing slight cellular infiltration of its adventitia and thickened intima.

Photographs by Mr. D. McA. Mehaffey.

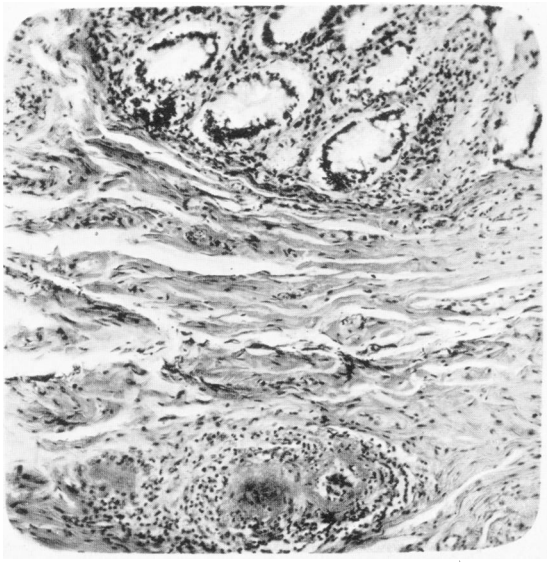

Fig. 5—The large intestine from tumour area.

Adeno-carcinomatous acini. Two small arteries, one completely obliterated by necrosis and exudate in its wall, the other partially occluded and less severely affected. There is an infiltration by inflammatory cells of the outer medial and adventitial coats.

Photographs by Mr. D. McA. Mehaffey.

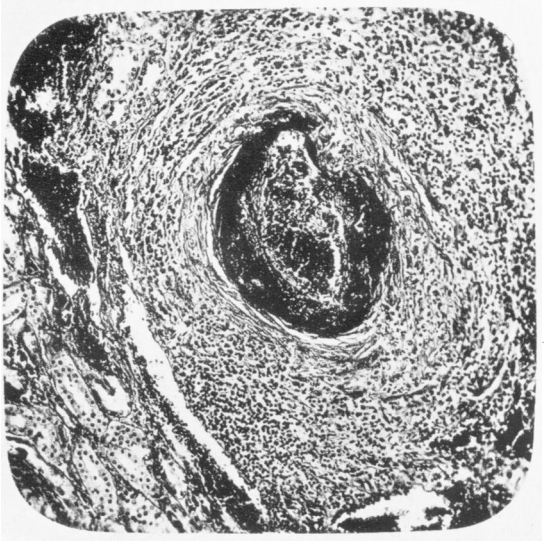

Fig. 6—Kidney.

This small artery shows acute inflammatory infiltration of the adventitia, media, and intima, with damage to the endothelial cells and a thrombosis as a result.

Photographs by Mr. D. McA. Mehaffey.

Studies from the Institute of Pathology

CASE VIII—A2512.

A Case of Mitral and Tricuspid Stenosis.

THE patient is a male, aged 24, whose family history reveals no relevant findings.

Previous Illnesses.

1. Eight years ago—meniscectomy following a knee injury.
2. Three years ago—attack of acute rheumatic fever.
3. Readmitted to Royal Victoria Hospital on five subsequent occasions with breathlessness on exertion, palpitation, and sense of fullness in head and epigastrium. These symptoms usually set in following a "cold" or overstrain.

History of present complaint.—The patient noticed a gradually increasing dyspnœa on exertion, with palpitation. Three days before admission the patient spat up some blood and complained of a pain in the right side of the chest. Since then he has noticed :—

1. Marked breathlessness even at rest.
2. Anorexia and insomnia.
3. Marked diminution of urinary output.
4. Swelling of the feet and ankles.
5. Feeling of heaviness below the right costal margin.
6. Headache.
7. Cough and spit, which is blood-stained.

The appetite was poor and bowels constipated, but moved readily with medicine. The amount of urine passed had diminished, and the urine was of a high colour. Sleep was poor.

On Examination.

The patient is a strongly-built male, showing some cyanosis of cheeks and ears. The skin is a lemon-yellow colour, which, with the cyanotic cheeks, has produced a very ashen-green colour of the facies. He is markedly orthopnœic. Gross œdema is present over the ankles and legs, and also over the sacrum. There is marked congestion of the external jugular veins, with venous pulsation to two and a half inches above the clavicle. Multiple small white punctate scars of an old infected chicken-pox rash are present over the body.

Alimentary system.—Tongue is heavily coated. Abdomen is distended, but shows bulging on both flanks—"burst tyre" effect. Shifting dullness and dullness in the flanks and both iliac fossæ can be elicited.

There is tenderness in the right costal margin, with an enlarged liver extending to the level of the umbilicus.

Cardio-vascular system.—Pulse-rate 104. Rhythm is regular, volume and tension are fair, and artery-wall is not palpable.

Blood-pressure 120/85.

Heart.—Marked generalised pulsation is present over the præcordium, with a palpable thrill at the apex. The apex beat is palpable in the fifth intercostal space five inches from the mid-line. D.A.C.D. = $\frac{iii}{1/5''}$

The heart-sounds are regular, but are almost completely obscured by a double "see-saw" type of murmur at the apex. The systolic element is rough and is conducted towards the posterior axillary fold. The diastolic element is softer and not conducted. This murmur can be heard all over the præcordium. In the pulmonary area the first sound is rumbling, and the second loud and slapping. No abnormal sounds can be heard in the aortic area or at Erb's point. In the tricuspid area the same "see-saw" murmur is present as occurred at the mitral area.

Respiratory system.—The chest expansion is poor, especially at the right base. Over the right base also, the lung resonance is impaired and the vocal fremitus and vocal resonance diminished. The breath-sounds are clear, except at the right base. Crepitations are present over both bases, but are considerably coarser on the right side.

W/R.—Negative.

Urine.—Acid. S.G. 1030. Trace of albumen. Urates + +.

X-ray chest.—There is considerable general enlargement of the heart, with a prominent pulmonary conus and dilated right auricle. Congestion is present at both lung-bases.

Electrocardiograph.—S_1 prominent. P. ii and iii prominent and bifid. Right ventricular preponderance.

Blood urea.—27 mgm. per cent. after admission. 43 mgm. per cent. shortly before death.

POST-MORTEM.

The body is that of a well-built young male, showing generalised icterus. The eyes and ears are cyanosed. Nail-beds are also blue, but there is no finger-clubbing. Gross œdema of lower limbs is present. Rigor mortis is commencing.

Serous cavities.—The peritoneal sac contains an excess of clear yellow serous fluid. The great omentum and appendices epiploicæ are a brick-red colour due to intense congestion. Both pleural sacs contain an excess serous transudate, which is most marked on the right side, while the pericardial sac contains about two ounces of a clear yellow fluid.

Heart.—The heart is grossly enlarged and globular in shape. The right auricle and great veins are dilated, and tense with blood. The tricuspid valve is the seat of a moderate stenosis, and the valve-cusps are shortened and thickened but show no recent vegetations. The right ventricle is enlarged and the wall hypertrophied. The endocardium of the left auricle is thickened and opaque, and there are calcified mural vegetations present in the posterior auricular wall. The mitral valve is grossly stenosed, and the valve-cusps hard and calcified. The left ventricle is not enlarged and the wall not hypertrophied. There are no changes in the aortic valve.

Left lung.—The lung feels heavy. The pleural surface is smooth. Small, hard nodules can be palpated throughout the lung substance. The bronchial mucosa is

intensely congested. On section the lung cuts firmly, and the changes of "brown induration" are well seen. On pressure a small amount of blood-stained fluid exudes, but there is no gross œdema. Scattered over the cut surface are a number of small hæmorrhagic infarcts of typical appearance.

Right lung.—The appearances are similar to the left, and the lung contains numerous infarcts, which are larger than those found in the left lung.

Liver.—This is enlarged and weighs four pounds. The surface is finely granular and presents some yellow-grey areas lying in a congested background. The liver cuts with some little difficulty. On section the nutmeg appearance of chronic venous congestion is well seen. Superimposed on the background is a distinct pattern of circumscribed yellow areas clearly marked off from the general background. Fibrosis is evident in relation to these areas and throughout the right lobe of the liver. The radicles of the portal vein and bile-duct appear normal. The gall-bladder contains no stones, and there is no obstruction to the biliary flow.

Spleen.—This is not much enlarged and weighs six ounces. The consistency is firm and the capsule smooth. On section it cuts firmly and the malpighian bodies stand out clearly, lying in a congested pulp.

Pancreas.—This is a little enlarged, and on section the acinar tissue appears normal. Scattered through the stroma of the gland and in nearby tissues are some yellow opaque areas of fat necrosis about the size of a pinhead.

Left kidney.—This kidney is enlarged and weighs eight ounces. The capsule strips readily, leaving a smooth surface. On section the organ is congested. The cortex is of normal width and the vascular pattern plain. The medullary striæ are prominent. There are no lesions in calyces, pelvis, or ureter.

Right kidney.—There is a double ureter to this kidney, connected to a double pelvis. The urine in the lower pelvis is turbid, but the mucosa shows no inflammatory changes. The appearance of this kidney otherwise does not materially differ from that of the left.

Urinary bladder.—The bladder appears healthy.

Alimentary tract.—Oesophagus: the mucosa is congested. Stomach: the mucosa is dark red in colour and of a velvety consistency. The surface is covered by a mucous secretion. There is no evidence of ulceration.

The rest of the alimentary tract shows some mild congestion of the mucosa, but no other lesions.

Adrenals.—These are of normal size, and the cortex is of average width and lipoid content. The medulla is grey in colour.

Thyreoid.—The thyreoid is a normal light brown colloid-containing gland.

Trachea.—The mucosa is intensely congested.

Aorta.—Shows no naked-eye changes.

Microscopical Examination.

Heart.—The mitral valve is thickened, fibrosed, calcified, and vascularised. There is general diffuse hypertrophy of the muscle fibres throughout the heart. Paravascular fibrous scars with a characteristic fibrillar appearance are scattered throughout the stroma of the heart.

Aorta.—None of the coats of the aorta shows any specific changes.

Lungs.—The typical microscopical changes of C.V.C.* are present, with thickened fibrosed alveolar walls, prominent alveolar wall capillaries, and alveoli packed with red blood-cells, and hæmosiderin containing macrophages (heart-failure cells). Throughout the lung the smaller branches of the pulmonary artery show evidence of a thickened intima, which is produced by a proliferation of the subendothelial connective tissue.

Superimposed on this basis of chronic passive congestion and diseased vessels there are numerous scattered hæmorrhagic infarcts of normal histological appearance, many of which are of some duration and show necrotic changes in the centre.

Liver.—The pathological changes in the liver vary greatly from one area to the next. There is marked chronic passive congestion present, with a blood-lake in the central part of the liver lobule in some areas. Some of the lobules show fibrosis and reticular collapse in the mid-zone of the lobules, while areas of liver-cell hyperplasia are present with bile-duct proliferation. The whole makes up a composite picture of chronic passive congestion, liver-cell damage, liver regeneration, and fibrotic replacement. Such appearances are typical of a long-standing chronic passive congestion, with periods of recovery, leading to a "cardiac cirrhosis" of the liver.

Kidneys.—The kidneys, apart from the marked venous congestion, are remarkably healthy and show no microscopical changes beyond a little albuminous fluid in Bowman's capsule and in some of the tubules.

Urinary bladder.—The mucous membrane is desquamated and the submucosa fibrosed with prominent blood-vessels.

Spleen.—The malpighian bodies are clearly marked off from a congested pulp, in which sinusoids are dilated with blood and show extensive fibrosis of their walls.

Pancreas.—There is a mild degree of post-mortem autolysis in the acinar and islet tissue.

Adrenals.—The cortex shows some loss of the cortical lipoid with a marked increase of the fibrous stroma. Focal areas of round-celled infiltration are present in the medulla.

Other organs.—The other organs showed no significant microscopical changes.

Anatomical diagnosis.

Mitral stenosis : calcification of cusps.
Hypertrophy of right ventricle : tricuspid stenosis.
Healed rheumatic carditis.
Gross C.V.C. of liver, lungs, spleen, and kidneys.
Cardiac cirrhosis.
Infarcts in lungs.
Pulmonary endarteritis fibrosa.

*Chronic venous congestion.

Commentary.

Congestive heart failure is, unfortunately, quite a common clinical condition, and it is usually the end result of an unsuccessful battle waged between the heart muscle and some mechanical difficulty to the normal blood flow. This difficulty to the blood flow often takes the form of some valvular damage to the heart, either a valvular insufficiency or stenosis. Thus, when such a patient dies he will show pathological changes, due to two causes :—

1. Changes produced by the mechanical insufficiency in the circulation.
2. Changes caused by the etiological agent which has damaged or is damaging the heart-valves.

The changes produced by either of these factors will vary in degree and extent, and it is unusual for both to be equally prominent in any one case.

The clinical course followed in this case is in many ways typical of that group of cases of congestive heart failure, in which the etiological factor is of rheumatic origin. However, certain abnormal clinical features presented interesting problems which could best be solved in a clinico-pathological correlation.

The patient lived a normal, healthy life until he was twenty-one, when he developed an attack of acute rheumatic fever, which caused the initial cardiac damage. Subsequent recrudescences of the rheumatic infection, induced by intercurrent infections which were mainly of the upper respiratory tract, produced further cardiac damage. The brunt of the rheumatic infection fell on the myocardium and on the mitral valve. During the recrudescences of the infection the myocardium suffered further insults, while the mitral valve cusps became thickened, fibrosed, shortened, and at last calcified. Stenosis of an extreme degree resulted.

Evidence of involvement of the tricuspid valve with stenosis was present, but the changes were less marked and less advanced than in the mitral valve and were probably of not so long standing. During some of these later relapses, signs of congestive heart failure became evident and indicated the extremely low state of the myocardial reserve. Under medical treatment the patient's clinical condition improved, and on each occasion after treatment the patient was discharged from hospital much improved, but he had only to return each time intercurrent infection or overstrain again reduced the cardiac reserve to zero.

The rheumatic nature of the lesion described in the clinical history was borne out by the pathological examination. Stigmata of previous rheumatic carditis were present in the shape of typical valvular and myocardial lesions. Histological examination revealed the presence of para-vascular fibrillar fibrous scars scattered throughout the myocardium, which resembled in all detail the descriptions given by Gross of healed Aschoff nodes. No recent Aschoff nodes were seen, so that active rheumatic infection was absent at the time of the patient's death, which must have been due to mechanical causes dictated by an inefficient valvular apparatus and a myocardial reserve strained to the uttermost.

The case, in its final state, was therefore an example of combined mitral and

tricuspid stenosis. Such multiple valvular lesions present many interesting problems, both in diagnosis and hæmodynamics. This case was no exception.

The ante-mortem diagnosis of acquired tricuspid stenosis has been rarely made. Of the two hundred and fifty cases recorded in the literature, an ante-mortem diagnosis has only been made in thirty-one. There are many reasons, apart from the rarity of the condition, which account for this.

The condition is rarely found alone, and the commonest condition with which it is associated is mitral stenosis. This greatly complicates the detection of tricuspid stenosis, as the signs of mitral stenosis dominate the picture and obscure those produced by the tricuspid valve.

Relative tricuspid insufficiency, which is said to accompany frequently long-standing mitral stenosis, also presents considerable difficulty.

However, from the pathological physiology the following clinical manifestations have been deduced, which indicate the presence of a tricuspid stenosis :—

1. Dyspnœa on exertion, out of proportion to that found in mitral stenosis.
2. Cyanosis, often of extreme degree.
3. Distention of the cervical and brachial veins, with marked presystolic pulsation due to right auricular contraction.
4. Enlargement of the liver, with presystolic pulsation due to the same cause.
5. A diastolic or presystolic murmur heard best at the ziphoid, which usually differs in quality but not in timing from the murmur at the apex.
6. Enlargement of the heart to the right, with dilatation and hypertrophy of the right auricle.
7. The signs dependent on auricular activity will disappear during auricular fibrillation, auricular arrest, etc.

A diagnosis of organic tricuspid stenosis is justified if a patient with a chronic rheumatic heart and mitral stenosis presents the above signs. One must first exclude mitral stenosis and adhesive pericarditis, and mitral stenosis and patent foramen ovale, which are rare conditions presenting similar signs.

Pathologically, tricuspid stenosis presents no new features. The valve undergoes the same changes as the mitral and ends in healing. The right auricle is usually distended and its wall hypertrophied, and in one case reported the right auricle actually held 2,500 cc. of water. The right auricular enlargement is evident on both clinical and X-ray examination by enlargement of the heart to the right. A grossly enlarged heart on X-ray examination is not a common finding in pure mitral stenosis.

The changes found in the liver in this case were both unusual and interesting. They present the pathological features of that condition known as "cardiac cirrhosis," which has been much stressed in the French literature, but has received scant attention here. The condition was first described by Becqueruel in 1840, who suggested that C.V.C. from cardiac failure could produce hepatic cirrhosis—cirrhose cardiaque. Later it was produced experimentally by mechanically obstructing the inferior vena cava. Roussy pointed out in 1853 that when C.V.C. was prolonged, the picture of C.V.C. of the liver became complicated by regeneration of hepatic

tissue to form hyperplastic nodules and associated proliferation of connective tissue in two sites : (a) around the central vein, (b) around hypertrophic islands. Such a liver, on naked-eye examination, appears irregularly granular on the surface and shows on section pale yellow areas separated clearly from a background of a congested and cirrhosed liver.

There are no pathognomonic clinical signs which indicate the presence of cardiac cirrhosis, and jaundice, which occurs in ten per cent. of all cases of congestive heart failure, is not a constant clinical finding.

The only suggestive evidence is purely of an inferential nature and is given by the clinical history. This is the presence of severe recurrent episodes of congesitve heart-failure over a long period, with intervals of improvement in cardiac function, when the liver recovers from the venous stasis and regeneration takes place. However, even with such favourable conditions, cardiac cirrhosis does not always result.

The pathogenesis of the condition is readily explained. The degeneration of the liver cells resulting from the C.V.C. varies in extent and in severity in different lobules. The damage to the liver epithelial cells occurs during the phase of cardiac failure. Those lobules which have only been partially destroyed are repaired during periods of improvement in the cardiac function. When entire lobules are destroyed the change is irreversible, the supporting reticulum collapses, and fibrous tissue proliferation replaces the area by a fibrous scar.

The naked-eye and microscopical lesions in the liver of this case all tend to confirm that the liver is the seat of an advanced cardiac cirrhosis.

The lungs showed the changes of C.V.C. as the result of the long-standing mitral stenosis. This was clearly manifested by the "brown induration" of the lungs and the fibrosis of the alveolar walls, with the presence of red blood-cells and "heart-failure cells" in the lung alveoli. A small but persistent hæmoptysis was clinical evidence of this C.V.C. of the lungs.

The final illness was ushered in by pain in the chest, cough, and hæmoptysis. Clinical and post-mortem examination confirmed the presence of lung infarcts. Careful search was therefore made in the right auricle and auricular appendage for the presence of any thrombus which might have become partially dislodged, to impact in one of the branches of the pulmonary artery to produce the infarcts in the lungs. None, however, was found. Although the possibility of a thrombus having become completely dislodged without leaving any trace was considered, it was thought that in all probability the thrombus was locally formed in the lung-vessels. The presence of long-standing pulmonary hypertension was known to have been existent, and reactive changes with intimal hyperplasia was evident in the pulmonary vessels. Secondary degenerative changes occurring in the intima of such vessels could readily be the nidus for local thrombosis, especially with a reduction in the normal blood-flow induced by heart failure. This local thrombus formation, with subsequent lung infarction, might explain many cases of lung infarcts occurring in mitral stenosis, where the presence of a source for embolism is non-existent.

The inadequacy of the general systemic circulation resulted in anoxæmia, reduced

capillary blood-flow, raised venous pressure, and, because of the liver damage, some reduction in the plasma proteins is probable. Because of these factors peripheral œdema was marked, and generalised anasarca present. Portal hypertension, the result of the heart failure and some obstruction to the portal circulation in the liver, was shown by ascites, marked congestion of the mesenteric vessels, and a congested firm spleen. The spleen showed the typical changes of long-standing C.V.C. with fibrosis of the splenic sinusoidal walls.

The congestion of the kidneys, the pre-renal deviation of fluid to œdematous tissues, and the inadequate renal blood-flow were all reflected on examination of the urine. Oliguria was present, with the passage of small amounts of a high S.G. urine containing albumen and urates. The kidneys responded well to the use of mercurial diuretics and the renal excretion markedly improved, with a concomitant reduction in the œdema. At this time the kidneys showed no difficulty in ridding the body of unwanted waste products, and the blood urea was normal. However, as the cardiac condition deteriorated and the rate of blood-flow fell, the dependence of the kidney on the general state of the cardio-vascular system became evident. The output, therefore, diminished in spite of diuretics, and the blood urea rose shortly before death.

Death occurred after a long illness in an unavailing fight between the heart and an inadequately adjusted circulatory system which ended in marked right heart failure, showing gross œdema, C.V.C. of all organs, and fluid in all the serous sacs.

SUMMARY.

1. A case of rheumatic heart disease showing mitral and tricuspid stenosis is presented.
2. Points in the ante-mortem diagnosis of acquired tricuspid stenosis are made.
3. The liver showed the changes of "cardiac cirrhosis" which is discussed.

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REVIEWS

ELEMENTARY PATHOLOGICAL HISTOLOGY. By W. G. Bernard, F.R.C.P. (Lond.). Second Edition. 1940. London: H. K. Lewis & Co., Ltd. Pp. 75. Plates 181, Coloured 8. Price 10/-.

If every medical student could be induced to purchase this book, and keep it by his side on the bench when examining sections, he would quickly gain a sound basic knowledge of pathological changes involved in diseased tissues, instead of, as so often happens, a facility in merely "spotting sections." The book consists mainly of 181 micrographs of typical pathological conditions (eight of which are in colour), with a concise but accurate account of the changes from the normal in each condition. These short descriptions attempt to interest the student to try and resolve his own particular section into the various cells, particles, and substances of which it is composed, and thus to learn to analyse the changes represented from the normal, and to add them into a coherent account of what has happened to the tissue. It is only by such means that a true appreciation of pathology can be obtained, and it is only on such a true appreciation that a sound knowledge of medicine can be built. It is therefore a pleasure to strongly commend this book to the attention not only of undergraduates but to those who, while being beyond that stage, remain students in the true sense.

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