adherent pleura. If the parietal and visceral pleurae are separated, as they are in cases of pneumothorax, one would expect effusion to form if pleurisy developed. In almost every case of spontaneous pneumothorax (where, of course, the disease has spread to the pleura) effusion develops. The cytology of the fluid is that of a tuberculous exudate, and although at first tubercle bacilli are not usually found, in long-standing effusions they can almost always be demonstrated. In none of my cases has any other organism been found. If my opinion is correct we would expect to find that in cases of non-tuberculous disease artificial pneumothorax would not be complicated by pleural effusion, and this is actually the case. In one of my cases of abscess of the lung a small pool of fluid appeared for a short time, but in none of my other 54 non-tuberculous cases of artificial pneumothorax did any fluid form—a percentage of less than 2, as against 41.4 for the tuberculous cases.

In spontaneous pneumothorax effusion is usually present, being absent in only 4 of my 22 cases, and these 4 all did well. As a rule, the fluid in spontaneous pneumothorax becomes thick tuberculous pus, the lung never re-expands, and the outlook is very serious; 13 of my 18 cases in which fluid formed are dead, and only 2 can be said to have done really well. I think thoracoplasty should be considered early in many cases of spontaneous pneumothorax, for unless the pneumothorax cavity can be obliterated by re-expansion of the lung it will sooner or later become infected, and the outlook is almost hopeless.

The treatment of pleural effusion complicating artificial pneumothorax depends on many factors, and each case must be taken on its merits. If the fluid is left it will usually cause obliteration of the pneumothorax cavity by pleural adherence and fibrosis starting at the bottom of the pleural cavity and working upwards. If this result is desired, therefore, the fluid should be left, but the patient must be kept under careful observation lest too much fluid develops and causes displacement of the mediastinum. Before this happens a sufficient quantity of the fluid must be aspirated. Many cases in which artificial pneumothorax treatment is allowed to terminate in this way do extremely well. As the lung is drawn out a satisfactory fibrosis develops over the diseased areas and the healthy parts of the lung begin to function once more. If, however, it is thought better to prevent obliteration of the pneumothorax cavity and to continue the refills it is very important to keep the pleural cavity dry by aspirating the fluid at each refill. Usually after one or two aspirations the fluid does not re-form. When fluid forms, a lung which was previously partially collapsed will become flattened out, and as the fluid accumulates the pressure rises, but it should never be allowed to rise sufficiently to displace the mediastinum. Sometimes the first indication of the presence of fluid is an unusually high intrapleural pressure. In one of my cases a pressure which was usually negative before a refill was found to be +5+10, and after giving 100 c.cm. of air it rose to +10+15. The pressure may, however, be negative in spite of the fluid, but a small refill will render it positive. For example, in one of my cases with fluid the pressure before refill was -10-6, but it became +17+19 after only 100 c.cm. of air had been given.

In replacing fluid the quantity of gas given must be much smaller than that of the fluid removed; roughly speaking, about one-quarter the quantity is required. In one case 225 c.cm. of fluid was removed and 200 c.cm. of air given, and the pressure was altered from -8-0 to 0+6. In another case where the initial pressure was -17-9, after removing 600 c.cm. of fluid it became -22-13, and after another 200 c.cm. had been removed it was -28-15. On adding 200 c.cm. of air the pressure at once rose to -16-9, another 200 c.cm. made it -10-6, and a third 200 c.cm. sent the pressure to -4-0. In one more case a pressure of +7+14 became -7+4 after removing 1,700 c.cm. of pus. It is not wise to allow the intrapleural pressure to alter too suddenly, and in replacing fluid with gas I think it is best to use two needles, the gas being introduced through one and the fluid flowing out through the other; by this method the pressure can be raised or lowered or kept constant at will. If it is wished to encourage the lung to re-expand the fluid should be removed without adding any gas, or if this is not thought wise

oxygen should be used, as this is absorbed more quickly than air.

If the fluid is purulent the case is very much more serious. The pus should be aspirated and the intrathoracic pressure kept as low as possible in order to help the lung to re-expand. If the lung does re-expand the outlook is hopeful, but if x rays show that there is no sign of re-expansion in the lung then we must face the fact that the patient will probably have a permanent pyopneumothorax and be liable to any of the many complications of that condition. In these cases I have washed out the cavity with saline, normal or hypotonic. I have injected 2 per cent. formalin in glycerin and other disinfectants into the pleural cavity, and in cases where the pus is thick I have injected some modifying fluid and rendered the pus more liquid and easier to aspirate, but the end is the same. If, therefore, the fluid is purulent and the lung fails to re-expand I think the best chance for the patient is to perform thoracoplasty as soon as possible. Before the operation, however, it is necessary to get the patient as fit as possible by washing out the pleural cavity and allowing him a period of rest in bed and good feeding. In most cases a preliminary operation for evulsion of the phrenic nerve is desirable. It is hardly necessary to add that active disease of the other lung is a contraindication to thoracoplasty, but in this case the outlook is almost hopeless. If, however, the better lung shows no sign of active disease I feel strongly that in these cases thoracoplasty offers the best chance to the patient.

A CASE OF CHRONIC OBSTRUCTIVE JAUNDICE:

FOCAL NECROSES IN THE LIVER: MYELOID TRANSFORMATION OF THE SPLEEN.

BY

F. PARKES WEBER, M.A., M.D., F.R.C.P.,
LONDON.

I HAVE recently encountered a case of chronic obstructive jaundice which presents some points worthy of comment. I will relate only the main points of the patient's last illness.

A man, aged 62, had chronic obstructive jaundice (complete obstruction) owing to primary adenocarcinoma of the hepatic duct at its junction with the cystic duct. He died on March 11th, 1926, after having had jaundice for only about one month. He had had occasional haematemesis, but at the *post-mortem* examination Dr. E. Bock found no lesion in the oesophagus, stomach, or upper bowel to account for it. The adenocarcinomatous growth was small and macroscopically scar-like. No metastases were seen. The liver weighed 2,300 grams (70½ oz.), and the intrahepatic bile ducts were distended with colourless bile. On microscopic examination sections of the liver were found to contain numerous foci, recalling by their small size and their irregular distribution the focal necroses of infectious diseases (enteric fever, etc.). They were distributed in various parts of the hepatic acini and contained necrotic liver cells and bile pigment; in many of them there were also quantities of large phagocytic cells (macrophages), and in some of them there were also collections of polymorphonuclear leucocytes, as if suppuration were threatening.

There had been no fever during life, and an infective origin for the focal necroses can hardly be maintained. It seems to me that these necroses are most probably a minor variety of what Horst Oertel¹ called "multiple non-inflammatory necrosis of the liver with jaundice," an example of which I described with a coloured illustration in 1909.² One possibility is that they are merely capillary "cholérhagies" due to overdistension of bile capillaries (apoplexia biliaris hepatis), but biliary focal necroses of a peculiar kind in the liver have been found in the absence of obstructive jaundice.³

Another point of interest was the enlargement of the spleen, which I have not yet mentioned; it had been a puzzling feature of the case during life. This splenomegaly had much diminished (as splenomegaly generally does) after haematemesis, but at the necropsy the spleen still weighed 770 grams (27 oz.); it was evenly enlarged, of

rather firm consistence, and red on section. Some of this enlargement might have been due to congestion, but some of it was certainly due to myeloid transformation. On microscopical examination the splenic pulp, not the lymphatic follicular tissue, was enlarged. In Giemsa-stained sections the pulp was found to contain great numbers of eosinophil cells and erythroblasts and a certain number of myelocytes; a few megakaryocytes were also found (in other sections). A blood count at the end of February, 1926, had given 3,416,000 erythrocytes and 5,450 white cells to the cubic millimetre of blood (of the white cells 89 per cent. were polymorphonuclear leucocytes, 5 per cent. were lymphocytes, 4 per cent. were monocytes, and 2 per cent. were eosinophils), and in a blood film taken on March 10th (the day before death) a few myelocytes were seen.

Since myeloid transformation (metaplasia) was first discovered by Dominici it has probably been more often observed in the spleen than in any other viscus. O. Naegeli, who gives an excellent review of the subject,⁴ says that though Itami failed to produce myeloid metaplasia by experimental bleeding, Skornjakoff succeeded in obtaining this vital reaction by allowing sufficient intervals for recovery between the bleedings. Myeloid transformation of the spleen may (though of course it does not always) occur as a vital reaction to anaemias and cachexias of various kinds and various infective and toxic agents,⁵ including even mercury perchloride.⁶ It has likewise been observed as a compensatory reaction in cases in which the bone marrow has been reduced by osteo-sclerosis.

In conclusion, I must thank Dr. O. Bode (house-physician) for help in the clinical examination of the case, and Dr. E. Bock, and likewise Dr. J. W. McNee, for help in the pathological and histological examination.

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THREE CASES OF OBSTRUCTION AT THE VESICAL OUTLET AFTER PROSTATECTOMY.

BY

W. K. IRWIN, M.D., F.R.C.S.E.,

SURGEON TO OUT-PATIENTS, ST. PAUL'S HOSPITAL FOR GENITO-URINARY DISEASES.

A GREAT deal is written about the satisfactory results of prostatectomy, but comparatively little about the unsatisfactory cases. Yet it is by no means uncommon for obstruction at the vesical outlet to recur after a short interval, however carefully and apparently successfully the operation has been performed, and when there has been nothing to suggest malignancy. The following typical cases from my own practice may be of interest.

CASE I.

In October, 1922, a man, aged 62, was sent to me complaining of difficult and frequent micturition of twelve months' duration. He stated that he was passing urine ten or a dozen times a day, and two or three times at night, the first few hours of recumbency being untroubled. His eye and knee reflexes were normal, and nothing unusual could be palpated in the renal and vesical areas. His urine was free from blood and from any sign of infection, but the residue left in the bladder after micturition amounted to $4\frac{1}{2}$ ounces. On rectal examination the prostate was of uniform consistency, though not so elastic as usual, and the right lobe was definitely enlarged.

I did the ordinary (Freyer) suprapubic prostatectomy, but

experienced some difficulty in enucleating the enlargement, which was more fibrous than usual. I was particularly careful to divide any shelf existing between the prostatic and vesical cavities, and no abnormal symptoms showed themselves during convalescence. The suprapubic wound was completely closed and the patient was passing water by the twenty-second day. He stated that he had to strain more or less to get the urine away, but bougies and catheters passed readily into the bladder.

Five months later he returned complaining of great difficulty and considerable pollakiuria, so much so that he asked for something further to be done. Instruments would not pass owing to definite obstruction at the internal meatus.

I excised the scar tissue, opened the bladder suprapubically and freed it anteriorly, passed a metal bougie by the urethra into the prostatic cavity, put the patient in the Trendelenburg position, and inserted a Thomson-Walker retractor. I found that the internal meatus was greatly contracted, so, while my assistant pushed up the metal bougie, I cut away the intervening tissue with scissors until the instrument entered the bladder. The bougie was then removed, and I completely dissected away the partition between the vesical and prostatic cavities, leaving a large internal meatus. I then passed a big coude catheter along the urethra, fastened it into the bladder and closed the vesical and abdominal wounds around a small suprapubic tube, which was removed three days later.

The bladder was irrigated with boric lotion and the catheter changed daily for a week, the urethra being irrigated as the instrument was withdrawn. On the eighth, ninth, and tenth days I passed a catheter in order to irrigate the bladder, but did not tie it in. On the eleventh day, when the urethra had had three days' rest, I again fastened a catheter in and continued to do so till the seventeenth day, when the wound was firmly healed and the patient passing urine more freely than for years previously.

I have seen him from time to time since, and introduced urethral instruments with a view to ascertaining whether there was any contraction at the bladder neck. Large coude catheters pass without meeting any obstruction, and micturition is free.

CASE II.

In November, 1924, I was asked to see a man, aged 65, suffering from retention, who gave a history of slight difficulty for about a year and great difficulty for two or three months. By rectal examination both prostatic lobes were moderately enlarged but of uniform consistency. I passed a catheter and emptied the bladder gradually.

I performed a prostatectomy by the Freyer method as in the former case, the enlargement being hard, fibrous, and difficult to remove. Great care was taken to divide any shelf between the prostatic and vesical cavities, and the patient convalesced normally for some time, passing urine by the urethra at stool on the eleventh day. Three days later, and many times afterwards, he micturated independently of defaecation.

On the twenty-second day I found that I could not pass a catheter into the bladder, the point being arrested in the prostatic cavity. Only a small quantity of fluid could be introduced and that very slowly. I tried again on many subsequent occasions, always with the same result. Five weeks after the operation, when I was out of town, the suprapubic wound closed and the patient developed retention. The medical man who was called to see him passed a catheter. He thought the instrument was in the bladder when it was really only in the prostatic cavity, a mistake easily made in these cases. As he could not draw off any urine, he came to the conclusion that he had to deal with a case of suppression, and actually applied the treatment for that condition. After a short interval, however, the suprapubic wound burst open again and discharged a large quantity of urine.

I explored the vesical neck in the way already described, but after a thorough examination failed to find any opening. I completely removed the partition between the prostatic and vesical cavities, inserted a catheter into the bladder by the urethra, and treated the vesical and abdominal wounds as described above. The technique of the after-treatment was the same as in the former case. The patient made a rapid and uninterrupted recovery and has since passed urine without any difficulty.

CASE III.

A man, aged 50, who had been leading a healthy country life, consulted me in April, 1925. He looked very fit, but complained of slight difficulty for four or five years and great difficulty for a month. He was passing small quantities of urine every hour both by day and by night. He found it very hard to begin the act, and the stream was a mere dribble. There was no haematuria, no sign of infection, and no history of gonorrhoea. The eye and knee reflexes were normal. On rectal examination the right lobe of the prostate was definitely enlarged, the left little, if at all. I passed a catheter, after meeting some obstruction in the prostatic region and drew off 6 ounces of residual urine.

I performed prostatectomy by the open method, as recommended by Sir John Thomson-Walker, and again found great difficulty