College of Surgeons several specimens which admirably illustrate the relation to Gimbernat's ligament of the neck of a femoral sac. The accompanying drawings (Figs. 5 and 6) of one of these specimens show clearly that while Gimbernat's ligament lies opposite to the constricted neck of the sac, it is not the direct cause of the constriction. The neck is obviously smaller than the canal in which it lies, and the narrow constricting band completely encircles the neck, although it is best developed on the side of Gimbernat's ligament. The manner in which this constricting band protects the femoral vein from pressure is clearly shown.

I recently operated on a case in which the neck of the sac was constricted in an entirely different manner, and in which the cause of the constriction produced symptoms of a very unusual nature.

A man of 45 was sent into my wards with a diagnosis of renal colic. At long intervals he had had attacks of pain spreading from the right iliac fossa into the groin and accompanied by frequency and scalding on micturition. These attacks had increased in frequency and severity and were accompanied by the appearance of a swelling in the right groin. On examination he was found to have a small right femoral hernia, and the diagnosis was made of a femoral hernia containing a portion of the bladder wall. On exploring, however, no sign of the bladder could be found, but passing round the neck of the sac posteriorly was a dense round cord. Further examination showed that this was an obliterated hypogastric artery, with an abnormal origin from the external iliac. Traction on this cord drew down a part of the bladder wall and no doubt accounted for his symptoms, which have entirely disappeared since the operation, when the cord was divided and a radical cure of the hernia performed.

If in this case the hernia had been strangulated it is highly probable that the bladder would have been pulled down, and in such an event the division of Gimbernat's ligament as usually advised would involve a very real danger of injury to the bladder. It should be remembered that protrusions of the bladder are by no means uncommon complications of femoral hernia.

Conclusions.

I venture to think that the points to which I have referred are of practical importance in the diagnosis and treatment of a femoral hernia. They may be briefly summarized as follows:

1. In most cases the presence of a femoral hernia is characterized by the appearance of a soft, spherical swelling in the angle between Poupart's ligament and the tendon of the adductor longus.
2. The pubic spine is not so satisfactory as Poupart's ligament itself as a boundary between inguinal and femoral hernias.
3. Gimbernat's ligament is not directly concerned in the strangulation of a femoral hernia, and its division serves no useful purpose, and may be dangerous.

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EPITHELIOMA OF THE SKIN AFTER PROLONGED EXPOSURE TO CREOSOTE.

(With Special Plate.)

BY

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THE following case is an example of occupational cancer. It is an instance of epithelioma of the skin of the right hand in a worker exposed for many years to creosote. It will be noted that he had also a few warty growths on the left arm, one of which presented evidence pointing to possible early malignant changes.

The patient, who was 66 years of age at the time of his death, had worked for thirty-three years in a creosote factory; his work was to carry wood after creosoting, and he stated that he was "up to the eyes in creosote." A good many years ago (certainly fifteen

a small swelling (?) appeared at the back of his right hand, "like a corn." Every now and then it cracked open and healed up again. Eight years ago the growth had increased to the size of a two-shilling piece; subsequently it gradually spread until just before operation it was a large fungating ulcer covering the dorsum of the hand (see special plate).

A portion of the edge of the ulcer, removed and examined histologically previous to amputation, proved to be a squamous epithelioma.

A circular amputation above the elbow was performed by Mr. S. Raw, surgeon to Sunderland Royal Infirmary, to whom, and to Dr. Gilbertson of Sunderland, I am indebted for the clinical history. Seven or eight weeks later the patient died suddenly and a *post-mortem* examination was made.

Post-mortem Examination.

There were a few small warts on the left forearm. Beyond changes common to his age, the organs were healthy, save that there were small secondary epitheliomatous deposits in both lungs, in the liver, and in both kidneys. There were also two secondary deposits in the heart walls—one the size of a walnut in the left ventricular wall, involving its whole thickness and beginning to break down in the interior, and the other, the size of a pea, at the right border of the right ventricle.

The case is put on record because it appears fairly certain that creosote was the cause of the malignant growth and subsequent death of the worker. It will be noted that the internal organs showed numerous secondary deposits of cancer. The large deposit involving the wall of the heart projected into its interior. The glands in the right armpit were enlarged and invaded by malignant disease. The amputation wound was in excellent condition.

REINFLATIONS IN ARTIFICIAL PNEUMOTHORAX TREATMENT.*

BY

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THE importance of rest in the treatment of active tuberculosis is well recognized. Rest of the body and of the diseased part, followed by training of the body and rest of the diseased part until the lesion has healed, is now a common method of treatment. In pulmonary tuberculosis the lung can best be rested by artificial pneumothorax. When a lung is collapsed no special skill is required to put a needle into the pneumothorax cavity and inject gas. But it is only by experience that the amount of gas, and when it should be given, can be judged. A common mistake is to give a definite quantity of gas at definite intervals. Not only does the rate of absorption vary considerably in different individuals, but the optimum degree of collapse also varies. Moreover, the same patient may require more or less gas at refills as the treatment proceeds.

When starting pneumothorax treatment I give small frequent refills until the lung is well collapsed. I like to get a good collapse after about a fortnight and some five or six refills. I then find the degree of collapse which is best for the individual patient. Some require full collapse and others do much better when the collapse is less complete. We have next to discover how long the optimum degree of collapse will last without a refill; this is best done by means of *x* rays. If the patient does not do so well when the intrapleural pressure is higher than -2 cm. water, any further collapse of lung causing such symptoms as dyspnoea and loss of weight, it can be seen by *x* rays how much collapse of lung there is at this pressure, and by frequent screening when the lung begins to re-expand. If it is seven days the refills should be given weekly; if four days, then two refills a week are required. If, when the refill is due, the intrapleural pressure is -12 cm. water, then we know that the optimum pressures are between -2 and -12 cm., and the correct quantity of gas to give is that which will raise the pressure from -12 to -2 cm. By this means the lung is kept completely at rest; it is not collapsed beyond the optimum degree and the intrapleural pressure is never allowed to reach that stage at which re-expansion occurs. As time goes on it is usually found that the gas is absorbed less quickly and so longer intervals can be allowed between the refills. The object is always to keep the lung at rest. Sometimes

* On behalf of the Medical Research Council.

large refills (1,000 or even 1,500 c.cm.) are given at long intervals. In these cases the lung is completely collapsed and then allowed to re-expand before the next refill. I call these concertina cases, because the lung is alternately expanding and collapsing. As a rule in such cases the patient complains of dyspnoea and discomfort just after the refill; this is followed by a period of well-being, and this again by a period of increased symptoms before the next refill. Most of these patients lose weight; some, however, remain well, and this is usually because the disease is arrested and the treatment may safely be discontinued.

The difference between individuals is very considerable. I saw a patient recently who had been having 1,500 c.cm. of gas every five weeks. He had been having artificial pneumothorax treatment for over two and a half years, felt well, and was free from symptoms. The lung had completely re-expanded five weeks after the last refill; 600 c.cm. of air left a pressure of -6 cm. water, but it caused very good collapse. After two weeks there was not much re-expansion, but a week later the lung had expanded considerably. In this case to keep the lung at rest 500 or 600 c.cm. of gas should have been given every fortnight. However, as he kept so fit even when the lung was expanded it seemed that the disease was arrested, so I stopped the refills and he has remained quite well. On the other hand, absorption may be very slow. In one case there was practically no re-expansion of lung twelve weeks after the last refill and the lung had not completely re-expanded after six months.

X rays are of the very greatest value, but if not available an attempt must be made to ascertain the optimum degree of collapse from the signs and symptoms. If a patient says he feels ready for a refill it usually means that the interval between the refills has been left too long, for a refill should be given before the patient begins to feel the return of symptoms. Again, the temperature is often a guide. The chart shows a case in point.

R = Refill given.

The refill should have been given three days before, and when it was given too much gas was introduced and so a reaction occurred. Vital capacity is also of use in judging the optimum degree of collapse. After a refill the vital capacity is diminished, but if small refills are given the diminution should be very small. If the vital capacity is taken daily it will be found that at first it improves after a refill and then remains constant for a time. After this it will be found to diminish slightly, and the refill should be given before this diminution begins.

Adhesions may prevent a satisfactory collapse of lung, and may be so great that the treatment has to be abandoned. It may, however, be decided to continue the refills in the hope of stretching the adhesions and so making an efficient pneumothorax possible. In such cases very high pressures are not needed, for better results are obtained by giving frequent refills, leaving the intrapleural pressure constant and thus keeping a steady pull on the adhesions.

When fluid develops during pneumothorax treatment the gas is less quickly absorbed, so that the lung often remains collapsed for a long period without a refill. It is usually wise, however, to remove the fluid and replace it with gas, because if it is left adhesions are almost certain to form.

Sometimes, though only a partial pneumothorax is obtained owing to adherent pleura, it is sufficient to relieve the symptoms. Usually in such a case the pneumothorax cavity is gradually obliterated by contraction of the adhesions and increase of adherent pleura. In such cases the refills should be continued as long as possible, for the diseased lung becomes fibrotic and the patient often does very well. It is useless to continue the refills if there is so much adherent

pleura that an efficient pneumothorax cannot be obtained. It may happen during treatment that the disease begins to spread in the other lung, and in this case the refills should be stopped. The patient then usually improves, so that it may be said that the pneumothorax aggravated the disease. But cases are often to be met with in which the disease is spreading rapidly and steadily, and after an artificial pneumothorax has been induced there soon appears a spread on the other side. On stopping the refills, however, there is improvement on both sides, although the patient is kept on exactly the same treatment as before the artificial pneumothorax was started. It seems, therefore, that it is really the pneumothorax which checks the spread of the disease.

In conclusion, I should like to emphasize two points: (1) It should be remembered that the object of the treatment is to rest the lung, and that therefore the lung should be rested and not alternately collapsed and expanded. (2) It is not necessary to compress the lung fully; a very satisfactory collapse can be obtained with the intrapleural pressure still negative, and harm is often done by giving too large a refill.

TWO CASES OF INTESTINAL OBSTRUCTION.

BY

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I. *Strangulation through an Aperture in the Ligamentum Latum Uteri.*

THE unusual feature of the first case was the nature of the strangulating agent, and I can find no published report of a similar condition. Barnard¹ stated that herniae may occur into pouches in the broad ligament, and mentioned that a case of internal hernia of this type was exhibited in the museum of the London Hospital, but I have been unsuccessful in tracing it.

I have investigated the bodies of ten adult women, all probably parous, and in every case it was possible to stretch the round ligament forwards and to demonstrate a thin, apparently avascular fold of peritoneum joining it to the main part of the broad ligament. On no occasion did it require any real force to perforate this "meso-ligamentum teres" with my finger, and I can only suggest, as a possible cause of the condition described below, that, as the result of the recent pregnancy, all the structures connected with the uterus were in a relaxed state, and that in some manner a coil of gut had ruptured this meso-ligamentous fold, perhaps when passing into the inguinal sac. After reduction, the coil would be left lying under the round ligament, a condition favourable for an internal strangulation to occur at any moment.

Mrs. V., aged 35, was admitted to hospital with the following history. Fourteen days previously she had had a normal labour; the pregnancy and puerperium were also without abnormal incident. At 11 a.m. on the day of admission, while bathing her child, she was seized with sudden violent pain in the region of the navel. The pain was continuous and very acute. After two hours, vomiting began and recurred at intervals all the afternoon, but was at no time faeculent in character. The bowels were not opened during the day, and no flatus was passed after the onset of symptoms. She stated that a small hernia, never bigger than a pigeon's egg, had existed on her left side for three years; it had always disappeared spontaneously when she lay down. It was not present at the time of the examination, but from her description it was probably inguinal. With the exception of pleurisy two years previously the patient could recollect no previous illness.

On examination six hours later the patient was obviously extremely ill and suffering from severe collapse. Her temperature was 97.5° and the pulse rate 65 and very weak. She complained all the time of agonizing abdominal pain, and her face was cold and beaded with sweat. The abdomen was lax and no distension or visible peristalsis was noticed. Percussion demonstrated the presence of some fluid in the flanks, and on palpation slight tenderness and rigidity were discovered in the lower part of the left iliac fossa. No mass was to be felt. Per rectum the involuting uterus could be made out, but nothing abnormal was discovered.

The diagnosis was at first rather doubtful, and seemed to lie between an acute perforation and acute intestinal obstruction; but had peritonitis been present the lack of general rigidity at such an early period would have been difficult to explain. Acute intestinal obstruction seemed therefore to be more likely, due possibly to internal strangulation.