the Dominions and of the nationals of the Dominions to this country are one of the most important elements in the attainment of such knowledge and sympathy. This is true of all grades and classes in the nation and it is pre-eminently true of the medical profession. Whether here or in Australia or Canada the doctor's work is the same in principle, and everywhere it is equally vital to the production of strong and enduring nations. I point these remarks by reference to this particular hostel which, I understand, it is judged impossible to carry on. But it seems to me clear that the idea underlying it is one which must be an essential part of any such comprehensive solution of the problem of post-graduate medical education as we at the Ministry are earnestly desirous of securing. We need a hospital, we need the educational facilities, and we need an organisation and a place where men from here and the overseas countries can meet as friends and exchange ideas and can be put in the way of knowing exactly where they can obtain what they want, whether it is specific knowledge of one subject or process or whether it is general knowledge of a whole subdivision of the available knowledge. Therefore one says heartily of such a hostel as this "Resurgat," and the sooner it arises again the better we at the Ministry will be pleased. For it is never to be forgotten that important as is the obtaining of medical knowledge the interchange of ideas and the formation of personal relationships is just as important, and in arranging for the first we must never forget the second.

COMPRESSION OF THE SPINAL CORD.*

BY

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I WISH to discuss here the question of compression of the spinal cord. I shall first show you two typical cases of spinal compression which we have in the ward.

The first case is that of a nurse, Miss D., 26 years of age. Her history is as follows:—

A little over a year ago she had "shooting pains" down the right lumbar region and into the right thigh. At one time these pains were thought to be due to appendicitis, and at another to be due to gall-stones. She was radiographed in both those regions, but with negative results. Her appendix was then found to be infected and was removed, but its removal made no difference to the pains. Her teeth were all extracted by an enthusiastic pyorrheal specialist. The pain in the right loin still persisted, and about eight months ago she began to drag her right leg. It was then suspected that she might have something

more deeply seated, and her physician in Johannesburg, a personal friend of my own, sent her over to let us have a look at her. During the voyage she developed tingling in the left leg. When she came in here she was normal from the waist upwards, but showed well-marked sensory, motor, and reflex changes in the lower limbs. Her sensory symptoms consisted in anaesthesia to pin-pricks over the saddle area of the buttocks and in both lower limbs below the knees. This saddle area, as we know, corresponds to the distribution of the sacral roots, whilst the area below the knees is innervated by the lower lumbar roots. In addition she had weakness in the right lower limb, and, to a less extent, in the left, without any definite muscular wasting. Another important point is that she had absent knee-jerks in contrast with a double ankle-clonus, an important diagnostic point. There were bilateral extensor plantar responses. There was also some sphincter trouble—precipitancy, I think. The cerebro-spinal fluid, taken from the lumbar region, was characteristic. It was pigmented a yellow colour—xanthochromia—and contained a large excess of albumin and a few extra cells. In order to make certain, we compared it with the cistern fluid higher up. The fluid from the cistern was clear and colourless; it contained no excess of cells, and only a small trace of albumin. The lumbar fluid contained 620 times as much albumin as did the cistern.

On these facts there remained no doubt that she was suffering from spinal compression. More than this, it was evident that the lesion was at the level of the third lumbar segment, because her knee-jerks, corresponding to that particular segment, were absent. The presence of ankle-clonus and extensor plantar responses showed that the lesion was in the spinal cord and not in the cauda equina. To clinch the diagnosis, we injected lipiodol into the cistern, and this heavy oil was seen in the radiograms to be held up at the upper border of the twelfth thoracic vertebra, whereas normally it should have fallen down as far as the second sacral.

We then handed her over to my surgical colleague, Mr. G. T. Mullally. He exposed the spinal theca at the level of the twelfth thoracic vertebra and found a neoplasm outside the theca, compressing the spinal cord. This growth was dissected off the theca, the operation being done in two stages. She has made an uninterrupted surgical recovery. We know that the pressure has been relieved by the operation, because her sensory phenomena have largely cleared up; she has recovered power in her legs; moreover, her knee-jerks are now present with reinforcement, as I show you. Her ankle-jerks are brisk, and the plantar response is no longer typically extensor. A still more striking proof of relief of compression is that the lipiodol has now trickled down part of the way towards the sacral region, and has reached the lower lumbar vertebrae. Probably there are some adhesions around the area of compression preventing the heavy lipiodol from falling to the lowest part of the theca. The neoplasm turned out to be a fibrosarcoma arising from the pedicle of a thoracic vertebra.

Case 2 is that of Miss C., aged 38 years, who had been totally paraplegic for 15 years.

At the age of 14 she began to have tingling in the left hip. In the course of years she became gradually paralysed in both legs, especially the left. That was 15 years ago. Fourteen years ago she was operated on for ovarian

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tumour. That, of course, had no effect on her spinal symptoms. She has been totally paraplegic ever since, with complete incontinence of both sphincters.

She came to us three months ago with all the signs of a complete, or almost complete, transverse lesion of the spinal cord. Above the waist there was no abnormality; the upper limbs were normal. There was total anæsthesia to all forms of sensation up to an inch above the umbilicus—i.e., to the ninth thoracic segment. There was total spastic paraplegia of both lower limbs; the abdominal muscles were still able to contract, though feebly. The knee-jerks and ankle-jerks were brisk. There was no ankle-clonus, but the plantar reflexes were strongly extensor in type. The abdominal reflexes were absent.

We then proceeded to examine her cerebro-spinal fluid. As in the other case, we found it was straw-coloured. It underwent spontaneous coagulation, which meant there was a huge excess of albumin. So here was the same clinical picture as in the last case as regards spinal compression, only a more severe paralysis. Lipiodol in her case was found to be held up at the level of the eighth thoracic vertebra—that is, higher than in the first patient.

My surgical colleague, Mr. E. Rock Carling, operated upon this patient six weeks ago. There was no doubt as to where the precise obstruction was; we could see it in the radiograms. Mr. Carling found no tumour outside the theca, but when he opened the theca he met the lipiodol, and also found two bony plaques sticking inwards from behind, compressing the spinal cord. It was a bilateral osteoma, and had doubtless been there the whole of those 15 years.

What has been the result? Whereas previously she had been totally paraplegic and anæsthetic, she can now make feeble movements of flexion of the toes, which she had been unable to do for 15 years. She became able to do this a month after the operation. Her spinal cord, of course, has been permanently damaged, and this we cannot make good, but there is already some small return of function. The lipiodol is still arrested at the same place as seen in recent radiograms, so probably there are old adhesions present at the level of the compression, and these have not been removed, though the compression has been relieved.

Both these cases are typical examples of spinal compression.

SPINAL COMPRESSION: ITS MEANING AND CAUSES.

What do we mean by spinal compression? We mean a slowly progressing compression, such as by the tissue-cells of a tumour, abscess, or cicatrix, or some other force of a physiological magnitude. This must be clearly distinguished from acute traumatic disruption of the cord, which is due to a force of supra-physiological magnitude—e.g., a bullet wound, a sudden impaction of fractured vertebra, &c. Besides the difference in the intensity of the force, there is also a corresponding difference between compression and sudden disruption in the mode of onset of the symptoms. The symptoms of spinal compression come on slowly and are gradually progressive, whereas the symptoms of acute disruption reach their maximum at once, and any subsequent change in the patient's condition is for the better.

I do not propose to enter fully into the anatomy of the condition. I would remind you, however, that the arterial supply of the spinal cord is somewhat complex affair. Each segment has its own radicular arterial supply, and, in addition, there are two long re-inforcing arteries, one running down the whole length of the cord in front, the other running down its dorsal aspect. Therefore the arterial supply of the cord cannot be cut off by any mere local compression; there are arteries which enter above and below to prevent the cord from undergoing necrosis. On the other hand, the venous current is an upward affair, and if the spinal cord is compressed, the veins below get distended. Compression of the cord thus tends to produce venous congestion, but not arterial anæmia. The cord, as you know, is surrounded by the spinal theca, being separated from it by a cylinder of cerebro-spinal fluid, this fluid acting as a sort of water-jacket to lessen mechanical shock. It is further prevented from swinging to and fro by the spinal roots and the ligamentum denticulatum, which anchor it on either side.

What are the common causes of spinal compression? First, spinal compression may be due to vertebral caries, but remember that compression from this cause is rarely due to bony pressure; it is generally due to inflammatory thickening or to an actual tuberculous abscess inside the vertebral canal. The tuberculous bones themselves may be deformed, but they rarely compress the cord; it is the super-added tuberculous inflammation which compresses the cord, commonly from the front. Secondly, in rare cases there may be compression by bony pressure, as in cases of scoliosis. Last winter we had here several cases of severe scoliosis, none tuberculous, so severe that the spinal cord itself was compressed by the bones. That, however, is uncommon; some of the books say it never occurs, but it does. Osteo-arthritis is another rare cause of spinal compression. Finally, there may be compression by tumours. Some tumours are intrathecal—that is to say, between the theca and the spinal cord, in the cylinder of fluid. Various tumours may occur in this situation—e.g., neurofibromata of the roots, endotheliomata of the meninges, gummata, or hydatids—or there may even be very large varicose tumours, angiomas, inside the theca, and so on. Of course, we can only recognise any of these is present when we get inside.

Then we may have extrathecal pressure, of which both these patients are examples. We may have a primary tumour, such as a sarcoma or chondroma—we had one of those last winter—an aortic aneurysm in a middle-aged syphilitic case. Or the growth may be secondary: carcinoma or sarcoma, or secondary thyroidoma, or secondary lymphadenoma, or secondary hydatid. These various growths, whether primary or secondary, may arise from the roots, from the meninges, or from the vertebræ.

What are the effects of such pressure on the spinal cord? The roots, being fixed points, cannot

evade pressure ; they must either become stretched or compressed. For that reason root-pains are the earliest and most constant symptom which you expect in spinal compression. The tumour grows and catches the roots. Next it displaces the cylinder of cerebro-spinal fluid. That does not produce very much in the way of symptoms. Then it squeezes through the fluid cylinder and compresses the spinal cord itself. When it does so, it produces venous engorgement below the level of the lesion. This venous engorgement causes exudation of blood below the level of the lesion, and consequently there is pigmentation of the fluid, and in the fluid, too, there occurs an excess of albumin, so great in some cases as to cause spontaneous coagulation. This is commonly described as Froin's syndrome—xanthochromia and spontaneous coagulation, a condition which is pathognomonic of spinal compression. It was present in both of these cases. Lastly, as the tumour grows, the cord itself becomes flattened out, and then we get signs of disease of the long tracts which run through the cord. So the symptomatology of the condition is quite easily understood.

(To be concluded.)

INTESTINAL OBSTRUCTION FROM GALL-STONE.

"GALL-STONE ILEUS."

A PITFALL FOR THE PRACTITIONER.

BY

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(Concluded from p. 73.)

TREATMENT.

AT one time enormous doses of belladonna were given with the intention of relieving the intestinal spasm, and in the hope that natural cure might be thus brought about. One reads of cases where stones have been voided, and the treatment has been given the credit. I was amused at the account of a case in which the attendant claimed that he had cured a case in which the patient passed a stone 3" × 1½", and another in which, after abdominal manipulation, a stone the size of a ping-pong ball was voided naturally. I suspect that in all these cases treatment has been a coincidence, and that but for the grace of God the patients might have suffered oblivion.

The most charitable and practical way to look at the matter is to say that in these days probably surgical intervention with all its risks is much safer than the other plans that have been popular from time to time. The form which intervention should take may admit of some discussion in the late cases. Sometimes an enterostomy has been performed above the stone, with the idea that the obstruction having been relieved, the stone would

safely pass on and lead to spontaneous cure, leaving only the intestinal fistula to be dealt with by a plastic operation (Fig. 12). The relief of the obstruction in this way removes the only stimulus to effort on the part of the intestine and destroys the last chance of the stone being propelled along the bowel. In the cases in which I have seen this carried out, the result has been disappointing and the patients have died, either as the result of continued exhaustion, or from peritonitis, or from the rupture of an ulcer (see Case 2 in appendix). The experiences of the treatment of this type of obstruction have confirmed what has been learnt from other cases of obturation, and I have now come to look upon enterostomy as only an adjunct in the treatment of obstruction and never as a method by itself.

I would say, therefore, that if a diagnosis of gall-stone obstruction is made, the best chance for the patient lies in early laparotomy for the purpose of searching for and removing the cause of that obstruction.

The question may arise as to where the operation should be carried out. Generally speaking, I am strongly of the opinion that operations should be performed in properly equipped institutions ; but I do think that there are many emergencies which have a better chance if they are not disturbed in the way that such removal may imply. Cases, for instance, under consideration are always elderly and sometimes gravely ill, and I believe that their chances are sometimes better if they are dealt with in their own homes. I am always thankful to have learnt a great part of my emergency surgery in the small industrial houses of Tyneside, and cannot be too grateful to my doctor friends who have so ably assisted me and seconded my efforts in every way. Very often these patients are actually regurgitating and vomiting intestinal material, and this may be a very grave complication when the question of anæsthesia arises. Under these circumstances it is usually better to empty the stomach before an anæsthetic is administered, or, if that is not done, the stomach tube should certainly be passed as soon as the patient is unconscious.

The question of the anæsthesia is very important. In the early stages there can usually be no valid objection to carefully-administered general anæsthesia, but if for any reason the patient has to be operated upon in the stage when toxæmia has supervened, then general anæsthesia should certainly be avoided. Local infiltration of the abdominal wall will nearly always suffice, and should unexpected complications be met with, general anæsthesia can be administered. The operation is always simple. It consists in opening the abdomen in the middle line below the umbilicus. In most of my cases a couple of fingers introduced into the peritoneum have almost at once encountered the piece of intestine containing the stone, and when it has not been so, a short search has soon disclosed its whereabouts. It is best to follow the collapsed intestine, and it is well to