COMPRESSION OF THE SPINAL CORD.

BY

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CLINICAL SYMPTOMS OF SPINAL COMPRESSION.

What are the clinical symptoms of spinal compression? First, is there any deformity of the spine? Deformity is very common in caries, but rare in tumour. Neither of these patients had any deformity. Root-symptoms are, almost always, present from the start. If it is a posterior root which is compressed, there is root-anæsthesia at the level of the lesion. If an anterior root is compressed, there is root-atrophy of the muscles at the level of the lesion. There is also loss of reflexes at the level of the root affected. In the first of our two cases the patient had loss of the knee-jerks at the level of the lesion, the third lumbar.

Then there are tract symptoms, which may be sensory or motor. What are the sensory tract symptoms? If the compression is a unilateral affair, you get one set of symptoms; if it is a bilateral affair, you get another set. Suppose it is a unilateral compression of the cord, then you have the Brown-Séquard syndrome—that is to say, on the side of the lesion, pyramidal symptoms below the lesion, and on the opposite side loss of temperature and pain in the opposite leg. There is, in fact, a weak motor leg and a weak sensory leg.

If it is a bilateral lesion, what do you get? You get loss to temperature and pain on both sides from affection of the lateral columns, and if the posterior columns are also compressed—as they were in one of our cases—there is also loss of joint-sense and vibration-sense, the fibres for which run upwards in the posterior columns, and may be spared in an ordinary Brown-Séquard case. And if the whole transverse area of the spinal cord is affected, you also get loss of perception of cotton-wool touches. Cotton-wool touch sensations have a choice of two paths: they may take the narrow path in the posterior column of the same side, or the broad path in the lateral column of the opposite side. Therefore both those paths must be obstructed for there to be loss to perception of cotton-wool touches.

Remember, also, that the level of the anæsthesia in a completely transverse case is often lower than at first you might expect it to be. This is because of the obliquity with which the spino-thalamic tract crosses over from the posterior cornu to the opposite lateral column.

What are the pyramidal symptoms? If it is a unilateral case, there is motor paralysis below the

level of the lesion, with increased tendon reflexes, and an extensor plantar reflex on the affected side. If it is a bilateral lesion, both legs are the subject of spastic paralysis, with increased reflexes, double ankle-clonus, and extensor responses. If it is higher up, there will be paralysis of the abdominal muscles; if higher up still, at or above the upper thoracic region, there will be intercostal paralysis; and if higher still, in the upper cervical region, spastic paralysis of the upper limbs. It all depends on the level of the lesion.

What changes in the reflexes do you expect? The reflexes at the actual level of the lesion of the affected root are absent. In our first case the knee-jerks were absent at the level of the lesion, the third lumbar. Below the level of the lesion the deep reflexes are increased and the plantar reflexes are extensor in type. If the lesion is high enough up, the abdominal reflexes are also absent.

With regard to the cerebro-spinal fluid, as I have said, there may be xanthochromia and an excess of albumin which may be so great as to cause spontaneous coagulation of the fluid. Sometimes there is also an excess of lymphocytes in the fluid. There is another point about the cerebro-spinal fluid—viz., that the fluid during lumbar puncture no longer pulsates, as it does above the level of the lesion. If you put a needle into the cistern and attach a manometer, you will see the fluid pulsate at each beat of the heart. If you put it in below the level of the lesion, the fluid does not pulsate. Moreover, it is under decreased pressure. Again, if you inject air into the lumbar region, below the lesion, it will ascend to the lower level of the obstruction and stick there, so that it can be seen by X rays. Better still, if you inject lipiodol (which is an opaque iodised oil) into the cistern, and let it run down within the theca, the heavy oil will drop down to the level of the obstruction and stay there. It was so in both these cases. Or again, if you put lipiodol into the lumbar region and stand the patient on his head, it will go up to the level of obstruction and stick there, instead of running up into his brain. So you can employ lipiodol to determine the upper and lower levels of the lesion.

X rays also show us any bony deformity which may be present, as in caries, and, and in some cases of tumour of the bones. It will always show us the shadow of the lipiodol. In health the lipiodol should fall straight down to the second sacral segment and form a triangular pool there; but in spinal compression it will be held up at the level of the lesion.

These clinical symptoms are so easy nowadays that no one has any difficulty in localising spinal compression, and the exact level of that compression.

DIAGNOSIS AND PROGNOSIS.

Next as to the diagnosis. Your diagnosis rests upon the symptoms of a gradually progressive transverse lesion of the cord—first the roots, then

the unilateral symptoms, then the transverse symptoms. The transverse symptoms, generally preceded by root-pains, make you suspicious. If to these you add the characteristic condition of the cerebro-spinal fluid, the xanthochromia, and the increase of albumin, and if X rays show that lipiodol is abnormally held up, then the diagnosis is a certainty; it is one of the few certain things in medicine.

What is the prognosis? The prognosis of secondary neoplasms is uniformly unfavourable. The prognosis of caries and of primary neoplasms is much more hopeful. Severe scoliosis, if causing pressure, is also an operable condition. The ultimate prognosis depends on the escape or otherwise of the arteries at the level of the lesion, because if the local arteries are already thrombosed there is an area of permanent necrosis, which, of course, cannot be replaced. On the other hand, if the arteries are not yet thrombosed, and if it has only been a case of venous congestion, as in our first case, then the prospects for the patient are excellent when once the pressure has been removed.

TREATMENT.

What is the treatment? If the patient is lucky enough to have had a spirochaetal infection, he or she may get well, if given ordinary specific remedies—mercury, salvarsan, iodides, bismuth. But if the patient has a primary neoplasm, the only prospect of relief is by operation; in our first case the neoplasm has been removed, and improvement has already begun. If the neoplasm is a malignant one, operation may be supplemented by the application of deep X rays to what remains of the tumour. We had a woman here a little time ago with paraplegia from a malignant chondroma, and we relieved her spinal compression by laminectomy. Part of the tumour, however, was inoperable, so we followed up by applications of X rays, and she was walking about well when we last saw her.

With regard to the treatment of caries, we may open any abscess which may be present, but unless we can be sure that an abscess is present, it is better not to operate on the bones, because it is not the bones but the thickening of perithecal tissues which cause the pressure. Operation results in caries have proved disappointing; much better results have been got from prolonged rest and counter-extension. A patient with spinal caries may require to be laid up in bed for 6 or 12 months, but if the vertebræ are kept passively extended most of the patients get well, and the time is well spent.

What about secondary neoplasms? All you can do there is palliative treatment to relieve the pain. How can that pain be relieved in a secondary case? You can relieve it by morphia or heroin, which gives the patient relief from pain for the remainder of his illness. These are cases in which you are justified in giving morphia, whether a drug-habit ensues or not. No tribunal will quarrel

with you for giving morphia in full doses to a patient with malignant root-pains.

Is there any alternative to giving morphia? Yes, sometimes there is. If you are a neural surgeon you may divide the pain-fibres in the spinal cord above the level of the lesion. That is done by notching the spinal cord near its lateral surface where these fibres run up. If you go too deeply in, there is the risk that you may also notch the pyramidal fibres. This operation of chordotomy was devised, I believe, by Frazier of Philadelphia. But if it is ever my misfortune to have a secondary neoplasm of the spine, I hope someone will give me morphia or heroin, and keep me under it continuously until the end.

SOME CASES OF DIFFICULTY IN DIAGNOSIS

WHICH A CLINICAL PATHOLOGIST MAY ENCOUNTER.*

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I WISH to deal with those difficulties which you as practitioners present the pathologist when, as you often do, you send him a *single* specimen of the patient's blood or excreta to examine. As practitioners I am afraid you sometimes blame the pathologist when he cannot from a single specimen make a diagnosis for you. The following cases will illustrate some of the difficulties which you as practitioners and myself as a pathologist will encounter in dealing with that very common condition which we call

CONTINUED PYREXIA.

CASE 1 is that of a young woman, aged 24, who was admitted into the female wards of this hospital with the history that *suddenly* after completing a day at the wash tub she was seized with general pains and shivering. She rapidly became so ill that she was obliged to take to her bed, where she was attended by her doctor for 14 days prior to admission. When seen by us she presented simply those symptoms and physical signs associated with pyrexia. There was no diarrhoea or enlargement of the spleen, or any sign of "rose-coloured" spots. Typhoid or paratyphoid seemed the most likely diagnosis, but to my disappointment I was unable to find any evidence of agglutination to the typhoid or paratyphoid groups. The faeces examination was likewise negative, but on the third day of incubation a bacteriological examination of the blood showed the presence of bacilli which proved on investigation to be *B. typhosus*. The especially interesting point was the complete absence of any agglutination even at the end of the second week of the fever, and it will interest you to know that this patient did not show any evidence of agglutination until she was well convalescent at the end of the fourth week of the disease. Had it not been,

* A Lecture delivered to the Post-graduates of the Fellowship of Medicine at the Westminster Hospital Medical School, September, 1926.