discoverable source of reflex irritation—such as errors of refraction, phimosis, intestinal worms, nasal troubles, dentition, etc.—should be removed or corrected. The bowels must be kept gently open by occasional doses of mild alteratives. There is no specific for the disease, but better results are got from arsenic than from any other drug. It ought to be administered from the earliest development of symptoms, in moderate doses at first, but judiciously increased and pushed up to the limit of toleration. For a child of 8 years old, Fowler's solution may be commenced in 3-minim doses, and gradually increased until within a week 10 minims are given three times a day. Children bear arsenic well, and can frequently stand large doses without inconvenience. Brilliant results have been claimed for heroic doses of from 15 to 25 minims, given from the onset of symptoms, three times a day after meals, in a large tumblerful of alkaline water. It is doubtful whether the ordinary and safer dosage of from 3 minims gradually increased to 10 is not attended, in most instances, with perfectly satisfactory results, without the disadvantages of the need for constant medical supervision. When the stomach is intolerant of the drug, Eulenberg has advocated its administration hypodermically, but in children the emotional upset which results from the prick of the needle constitutes a serious drawback. Cacodylate of soda is extensively used in France instead of liquor arsenicalis in the treatment of chorea. It may be given either hypodermically or in the form of a pill in much larger doses than other preparations of arsenic, but the garlic odour it imparts to the patient's breath is a strong reason against its administration. Chapuis has shown experimentally that arsenic is less toxic when combined with butter than when given in solution. A known quantity of arsenious acid, according to the dose to be administered, is taken and added to chloride of sodium in the proportion of 0.1 gram of the sodium salt to 0.005 gram of arsenious acid. This mixture is triturated with 10 grams of fresh butter, and given, spread on bread, after a meal. In all cases where arsenic is being administered over a prolonged period in large doses, a keen eye should be kept open for the occurrence of pigmentation of the skin or of peripheral neuritis, either of which may be regarded as evidence of saturation.

In stray cases arsenic is not tolerated; under such circumstances sulphate of zinc is sometimes found a good substitute, and may be given in doses of two or three grains, gradually increased to 5 or 6 grains to a child of 8 or 10 years. Antipyrin is another drug which has been recommended as a substitute for arsenic, but, to be of service, it must be administered in bold doses. McCall Anderson was in the habit of prescribing from 10 to 20 grains every six or eight hours for a child of 8 or 10. Ergot of rye is also credited with good results, but, like antipyrin, must be administered freely—a drachm of the liquid extract may be given to a child of 8 three or four times a day. Trousseau set great store on strychnine as a curative drug in chorea, and ordered its administration in gradually increasing doses until its physiological effects were produced. In the later stages of the disease, there can be no doubt about its value.

Besides arsenic, other drugs may be called for, according to the indications in each case. Thus, anaemia is frequently an important accompaniment and demands the administration of iron for its relief; nutrition may be much impaired, and will be benefited by the addition to the daily dietary of maltine and cod liver oil; if rheumatic symptoms are pronounced, the salicylates must be given in suitable doses; excessive restlessness demands the exhibition of bromides; and so on. If restlessness is accompanied by a dry state of the skin, diaphoretics are often very soothing. It is under such circumstances that antimony is specially useful. When exhaustion threatens, stimulants are necessary, and should be given in quantities to suit the requirements and age of the patient.

In all severe cases something should be done to control the muscular movements until the more slowly acting drugs above referred to have time to exercise their power. The movements may be so incessant as to threaten the patient's life from exhaustion, and under these circumstances more good will be got from hydrate of chloral than from any other drug. Gairdner declared that, when pushed sufficiently far, it has an almost absolute power of sus-

pending or controlling the muscular disorder. It may be given either alone or combined with bromide of potassium. If, for any reason, chloral is inadmissible, sulphonal or trional may be tried. Ringer recommends conium juice in doses of from one to several drachms, but its effects are not permanent, and in such large quantities it is apt to upset the child's digestion. In desperate cases it may be necessary to resort to the careful administration of morphine hypodermically, or to the inhalation of chloroform at short intervals. Cold douches or icebags to the spine, or the application along the course of the vertebral column of the spray of ether or of chloride of methyl, are local measures which are sometimes worthy of trial in extreme cases. Where the movements are not too violent, a warm bath or hot-pack may be used with benefit. Mechanical restraint often affords the patient comfortable relief: it is best accomplished by bandaging with flannel the upper limbs to the trunk and the lower limbs to one another, subsequently fastening a blanket or sheet tightly to the sides of the cot over the child's chest and abdomen. The use of a water bed lessens the risk of skin abrasions and bed-sores. In the later stages of the paretic variety of the disease, massage and faradization are valuable additions to medicinal treatment.

During convalescence, tonics, a generous dietary, carefully graduated gymnastic exercises, and change of air are all serviceable. The child should not be allowed to undertake mental work until complete recovery is established.

In the maniacal form of the disease met with in adults, treatment must be conducted on general principles, always bearing in mind that the condition is one likely to become serious, and therefore demanding, from the beginning of the illness, skilful and energetic management.

In all choreic cases the parents or friends of the patient should be warned of the liability to recurrence, and instructed that it is essential to put the child to bed and seek advice on the first indication of a renewal of the symptoms. In order to minimize the risk of relapse the general health must be maintained at the highest possible standard. The child should be warmly clad; a dry sunny climate should be chosen when possible, so that rheumatic ailments may be escaped; and during the cold months of the year cod liver oil should be administered perseveringly. Education must be carefully adjusted to the capacity of the child, and all forms of competition for distinction stringently forbidden. The daily life should be so arranged that a certain proportion of every fine day is spent in the open air, and all forms of excessive fatigue or mental excitement must be guarded against. Healthy children should be protected against the risk of fright and not allowed to associate with any child suffering from chorea.

A Clinical Lecture

ON

THREE CASES OF PARAPLEGIA.

DUE TO ACUTE ANTERIOR POLIOMYELITIS, TOXIC POLY-NEURITIS, AND TRAUMATIC MYELITIS RESPECTIVELY.

BY

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I WISH in this lecture to deal with cases which well illustrate the features of three important forms of paraplegia.

The first case which I shall take is one of acute anterior poliomyelitis in a young adult:

A. B., aged 20, a farm labourer, had always enjoyed good health, and came of a healthy family, with the exception that one sister had suffered from tuberculous glands. On September 25th he had a slight headache on going to bed, being in his usual good health all day, and on the 26th the headache was more severe, and he had constant "darting" pains in the calves of both legs. He went to work on the 26th and 27th, but on the latter day could only hobble along on account of pain. On the 28th he could just stand and walk across the room with great difficulty on account of pain and increasing weakness in the legs, and on the 30th he found he could not stand nor use his legs at all. There was also pain in the lumbar spine, but never very

intense. Incontinence of urine began on the 30th; the patient was conscious of the passage of urine, but unable to prevent it. Defaecation was not affected. The pain continued for a week and then stopped. There was never any girdle pain, numbness, or paraesthesia; the patient said he could always feel quite well over his legs; lately they have become "ticklish." After the first few days there was no spread of paralysis, but the muscles of the legs wasted rapidly.

The patient was admitted to the Bristol General Hospital on December 11th, 1907. His aspect was healthy, and he was well nourished, the pulse 100, temperature 99°, bowels constipated. He had many carious teeth.

Examination showed that the thoracic and abdominal organs were normal. The bladder was much distended, reaching higher than midway between the pubes and umbilicus. The urine was alkaline, ammoniacal, and deposited phosphates. The musculature generally was small; the muscles of the arms and of the trunk were normal. The paralysis was confined to the lower extremities. In them it was complete, except that he could flex and extend to a slight degree all the toes of the left foot. These toes were in a normal position, those of the right foot were flexed. From contraction of the flexor muscles the lower limbs were flexed at the hip and knee; the legs could only be passively extended to an angle of 60 degrees with the hips. Great pain was induced by attempts to passively straighten the hips and knees; otherwise he had no pain. There was great wasting of all the muscles of the thighs, legs, and feet. The feet and legs were blue and cold. Electrical reactions; faradic current.

Right leg: Reaction lost in quadriceps extensor, hamstring muscles, peronei, tibialis anticus, calf muscles, accessorius, extensors of toes and small muscles of foot.

Left leg: Reaction lost in same muscles, except that with a strong current a reaction occurred in hamstring muscles, long extensors of toes, and small muscles of foot.

Constant current: In a few muscles no reaction, but in most the muscles reacted to a strong current with a slow, wave-like twitch, and more readily to the anode than to the cathode.

Reflexes: Cremasteric and abdominal present. Right plantar absent; left doubtful. Knee-jerks and ankle-jerks absent. Sensation unaffected to touch, pain, temperature, and pressure. He was ordered the constant current and massage to the legs with gentle passive movements; bladder to be washed out twice daily, and urotropin given internally.

In six weeks the condition was improved in so far that the lower extremities had been got into a straight position and the secondary contraction of the flexors corrected; the pain on passive movement had now disappeared; he had recovered control over his bladder, and the catheter was no longer required; the urine was acid and normal; he could slightly abduct his hips and draw up the left thigh. As further progress was likely to be very slow and probably recovery would be very imperfect, and as he could be looked after at home, he was allowed to go out.

The first point to which I wish to call attention is the age of the patient. It is now well recognized that acute anterior poliomyelitis is an affection which not infrequently occurs in young adults between the ages of 20 and 30, and occasionally in older persons. The term "infantile paralysis" as a synonym for "anterior poliomyelitis" is therefore a misnomer, and should be dropped. The second point is the distribution of the paralysis. In this case it was paraplegic; but, as is generally the case in anterior poliomyelitis, all the muscles of both lower extremities were not paralysed. The muscles which flex and extend the toes of the left foot had escaped. This apparently capricious escape of certain muscles or group of muscles of a paralysed limb is an important diagnostic point. The affected extremities in anterior poliomyelitis are nearly always remarkably blue and cold to the touch; this symptom, in the absence of any affection of sensation, is also of practical importance in the diagnosis of the disease.

Another interesting feature of the case is the presence of an extensor plantar reflex in the left foot; this developed after he had been in the hospital a short time; the muscles moving the toes of this foot were left intact, and so gave the opportunity of determining the presence of this reflex. An extensor plantar reflex is evidence in this case of involvement of the pyramidal tract. Dr. Farquhar Buzzard, in noticing the presence of this sign, observes that it is explained by the fact that in the acute stage of the disease the white columns of the cord, particularly the antero-lateral, show a marked swelling of the axis cylinders and medullary sheaths. Without going more fully into the pathology of acute poliomyelitis, I may add that Dr. Buzzard¹ comes to the conclusion that "the essential lesion is an inflammation of the interstitial tissues of the central nervous system, due to the presence of micro-organisms or their toxins probably in the blood (but possibly in the lymph) circulating within the system, and the fact that the grey matter suffers more than the

white is explained by the greater vascularity of the former and the resistant character of the component parts of the latter."

A further point of interest is the character of the pain from which the patient suffered. He was at ease when lying still, but any attempt at passive movements of the joints produced so great pain that he could not bear them to be moved. This is the characteristic feature of the pain met with in acute poliomyelitis after the first few days of onset. It varies greatly in different cases. In this patient it had been unusually severe, and in consequence the limbs had been allowed to become contracted in flexion. The treatment carried out was massage with very gentle passive movement, gradually getting the limbs each day more into the position of extension. After about a week the limbs were in full extension, and the pains at the same time gradually disappeared. There is no doubt that allowing the limbs to remain in a position of flexion from contraction keeps up the pain and adds greatly to the patient's sufferings.

Lastly, another important point, which is common to nearly all cases of paraplegia of spinal origin, was the condition of the bladder. From the history one would have inferred that he had paralytic incontinence, and might have jumped to the not improbable inference that it was due to lesion of the centre for micturition. But on examination we found that the bladder was greatly over-distended, and that he was in reality suffering from an overflow incontinence. Careful catheterization for a short time resulted in the gradual recovery of his power over the bladder, adding immeasurably to his comfort, and also relieving him from the dangerous consequences to the kidneys which follow from a constantly over-distended bladder.

The case I shall consider next is also one of paraplegia, coming on acutely, with muscular wasting, and without any loss of sensation, but with a very different pathogeny. It is one which comes under the clinical category of 'ascending paralysis.'

Under this term are included cases in which, with a certain similarity of clinical symptoms, the underlying pathological changes are quite distinct. Cases of ascending paralysis fall into three groups: (1) Landry's paralysis or acute ascending paralysis, in which formerly no lesion was discoverable in the cord by the methods of investigation then available, but in which examination of some recent cases by Nissl's method has shown a chromatolysis of the cells of the anterior cornu; (2) acute ascending myelitis; and (3) multiple neuritis, or acute toxic polyneuritis.

The following case belongs to the last category, and well illustrates the features of this group, which runs a more favourable course than the other two varieties, and therefore the opportunities for pathological investigation of it are not very numerous.

The patient, a man aged 42, had always enjoyed good health, except for an attack of pleurisy eighteen months previously. He was a teetotaler and had never had syphilis; he used both tobacco and tea very freely. Five weeks before admission he was taken with numbness, stiffness, and difficulty of movement in the legs, with some shooting pains. The "numbness" rapidly spread up to his waist, and in a few days he lost all power in his legs. This was followed some two weeks before admission by loss of voice; the day before admission spasm of the jaw came on whilst eating. There was constipation, some frequency in micturition, but no incontinence or retention. On admission, June 29th, 1906, the patient looked healthy. Pulse 108, respirations 28; temperature normal. Lungs normal, except for deficient entry of air into the bases. Heart normal. Abdomen normal. Liver not enlarged. Stomach somewhat dilated. Urine acid, specific gravity 1020. No albumen or sugar.

The patient could sit up in bed, and could just raise his knees, but not his feet, off the bed; there was also slight power of abduction or adduction of the thighs and flexion of the feet. These were the only possible movements.

He could move the arms and hands well, but there was some inco-ordination of movement. The muscles generally were flabby, relaxed, but not wasted. No loss of the muscular sense in either arms or legs. Sensation to light touch, pressure, pain, and temperature normal all over the limbs and trunk. Hyperidrosis of feet.

Reflexes.—Deep; absent in the legs; present in the arms. Superficial plantar absent; abdominal, cremasteric, and gluteal present. No difficulty in swallowing. Face and eyes unaffected. The left vocal cord almost completely paralysed. Voice weak.

July 6th. The paralysis had extended; in the feet and legs it was now absolute; the muscles of the left calf were wasting more rapidly than the right. There was commencing loss of power in the forearms and hands. He could not completely

close the right eye. There was now complete aphonia, both cords being paralysed and lying in the cadaveric position. Mentally he was dull, irritable, and restless.

July 9th. Temperature normal; respirations 32. The diaphragm was paralysed, and he had loss of power in both orbiculares oculorum. Double foot-drop.

On July 18th he had a bad attack of dyspnoea, in which he nearly died. He suffered much from accumulation of mucus in the trachea, which he had great difficulty in expelling; there was partial consolidation of base of right lung, and dilatation of the right side of the heart. Cough toneless; complete aphonia; diaphragm paralysed. All the muscles of the lower limbs were paralysed and much wasted and tender to pressure; this was especially so in the calf muscles; the only movement left was that of abduction at the hips. On testing electrically, R.D. was present in all the leg muscles and in both quadriceps extensors; reaction to a strong faradic current was entirely lost in dorsiflexors of feet. There was now also much loss of power in forearms and hands; extensors were more affected than flexors, so that there was partial wrist-drop. No R.D. in these muscles. Ataxy of movement was very marked. The nerve trunks in both legs and forearms, especially in the former, were very tender to pressure.

On July 27th. In addition to previous symptoms, there was now some wasting of the muscles of the thenar eminences, with partial R.D. in them, and of muscles of forearm. The rectus abdominis muscle wasted, and he could not raise himself in bed. There was occasional incontinence of urine. Sensation unaffected in all parts.

During August there was a partial improvement. There was considerable recovery of the movement of the diaphragm, and respiration became easy; rate, 20 per minute. The paralysis of the orbiculares oculorum and vocal cords passed off; he also regained the power of adduction of the thighs, and some increase of power in the forearms. In addition to the wasting of the muscles of the lower extremities—now extreme—and of the small muscles of the hands in less degree and most marked on the left side, there was now also apparent some wasting of the trapezii, and of the scapular muscles, but not of the deltoid.

During September his condition remained much the same until towards the end of the month, when his general strength began to fail, without any increase in the paralytic symptoms. There was frequent vomiting, so that he retained very little food; the pulse became feeble and rapid; oedema of the legs and hypostatic congestion of the bases of both lungs appeared; he was quietly delirious at night, and died on October 10th, three and a half months after the onset of the illness.

The post-mortem examination was made on October 11th. Stated in brief, the body was emaciated; hypostatic congestion of the lungs; the post-arytenoid muscles small, a little wasted and yellowish on the left side; trachea and bronchi contained frothy mucus. Heart: right side dilated and contained clot, heart muscle soft and uncontracted. Abdomen: peritoneum normal; no ascites; liver and spleen normal; gall bladder contained five large gall stones; kidneys, chronic congestion. Skull cap: meninges, brain healthy in all parts; cerebro-spinal fluid normal in quantity; the spinal cord and spinal meninges looked healthy, and the former was of normal consistence. Sections were made of the medulla oblongata, of the cord at short intervals throughout its length, of the anterior and posterior tibial, peroneal, a nerve from the left forearm, both recurrent laryngeal, left vagus, and left phrenic nerves, of the heart muscle, and of the diaphragm, peronei, gastrocnemius and intercostal muscles. Sections of cord and nerves were stained by Weigert's, Van Gieson's and Marchi's methods, and the cord also by Nissl's method; the muscles by Marchi, Van Gieson, and haematoxylin and eosin.

In the medulla and cord no inflammatory changes were recognizable (Van Gieson's stain); the central canal of the cord showed in all sections proliferation of the lining epithelium, choking the lumen and extending widely into the surrounding commissure. Sections stained by Weigert were normal; the fine plexus of fibrils in the grey matter of cord was present.

By Marchi's method a few very fine black dots were found scattered over the cord, chiefly in the posterior columns, in many sections; these are described by Dr. F. Buzzard² as present in the cord in many septic or toxæmic cases.

Nissl's stain showed well marked and extensive changes, consisting in chromatolysis of the anterior horn cells; these changes were found in the cervical region, at the level of the seventh and eighth cervical and first dorsal roots, and in the lumbar and sacral regions. The cells showed various stages of degeneration, from a disappearance of Nissl bodies at central parts of cell body and loss of the normal contour, through the second degree of degeneration—that is, disappearance of granules and processes with lateral position of nucleus, vacuolation, etc., to a few instances in which there was complete degeneration, loss of nucleus, and the cell had almost disappeared. In the great majority of cells the changes were of the first or second degree.

Although the paralysis was too widespread to warrant any accurate localization of nerve-cell groups in relation to muscles, in view of the predominant affection of peripheral and extensor muscles the following points are of interest.

1. The anterior mesial column of cells (and small post-mesial group in cervical region), supplying muscles of trunks, were healthy throughout.
2. Clarke's column normal.
3. Antero-lateral group only moderately affected, most in sacral region.
4. Post-lateral group showed throughout cord chief changes, which in this column were both extensive in range and advanced

in degree; in the sacral region the cells were almost universally more or less degenerated.

5. Post-posterior lateral group (cervical region) normal.

6. Central group showed advanced disease in lumbar region almost as severe as in posterior lateral group; slight changes in upper part of sacral region. No changes were found in nuclei of hypoglossal, spinal accessory, vagus, ascending root of fifth or eighth nerves, or in the olive.

No degenerated fibres could be seen in anterior or posterior roots by Marchi's or Weigert's method, nor in several roots of the cauda equina which were examined.

Peripheral Nerves.—These showed a very advanced degree of neuritis, but Marchi's stain showed no recently degenerated fibres. There was parenchymatous degeneration of the nerve fibres, followed by a secondary interstitial inflammation of endoneurium and connective tissue. There was a moderate degree of endarteritis and marked endophlebitis, with the lumen obliterated by thrombus, of the vasa nervorum. In the left recurrent laryngeal nerve there was probably some increase of the interstitial connective tissue; the vagus and phrenic were normal.

Muscles.—The cardiac muscle was normal; in the diaphragm there were patches of parenchymatous degeneration of muscle fibres, in which the striations had disappeared and the fibres contained fine fatty granules. In the gastrocnemius, peronei, and a forearm muscle (extensor) there was considerable degeneration of muscle fibres, with some increase of interstitial tissue.

Thus in this case there was a rapid loss of power in the legs, spreading upwards from the periphery, and followed speedily by wasting of the muscles, shortly afterwards affecting the diaphragm, laryngeal muscles, and orbiculares oculorum, and, as in the first case of acute poliomyelitis, the paralysis was unaccompanied by any loss of sensation. Later the muscles of hands and forearm were similarly, but not so severely, affected. The clinical features which enabled the case to be recognized as one of polyn neuritis were the distribution of the paralysis, which was symmetrical, more severe at the periphery of the limbs, and affecting the extensor muscles earlier and more severely than the flexors. The affected muscles gave a well-marked reaction of degeneration to electrical currents. Other points are the tenderness of the nerve-trunks and muscles, the predominant affection of the diaphragm, whilst the other trunk-muscles escaped—a not unusual feature of these cases—and the absence of any affection of the sphincters. Although there was no objective loss of sensation the patient complained much of numbness, "pins-and-needles," and shooting pains in the legs. In all these features this second case differs in a marked way from the first one of anterior poliomyelitis.

With regard to the pathology, the nature of the poison which caused this extensive and severe paralysis could not be determined. It was not lead or alcohol, nor the diphtheritic toxin. Whatever it was, it exerted a selective action on the lower motor neuron, picking out the terminations of the motor nerves, and only slightly affecting the sensory ones. But its action was not confined to the peripheral nerves, but was shown in the morbid changes in the cells of the anterior horn. Similar changes, more or less intense, are well known to occur in the neuritis produced by diphtheria and alcohol. In these cases the poison, being carried by the blood, is able to act upon the whole of the lower motor neuron. Both cell and nerve are exposed to its action, and it may be supposed that the peripheral parts of the nerve, being furthest from the trophic centre, should suffer most severely.

The history of the cases comprised under the clinical term of "ascending paralysis" is interesting. First described when multiple neuritis was not known, they were all ascribed to acute disease of the spinal cord. Later, since, by the histological methods then available, no changes could be found in the cord, and cases of polyn neuritis, such as our present one, came to be recognized and described, some authors claimed that all cases of ascending paralysis were due to neuritis. The use of Nissl's method, however, demonstrated changes in the anterior horn cells previously irre recognizable. If the microscopic investigation of this case had been confined on the one hand to the cord, or on the other to the peripheral nerves, it is obvious that the case might have been considered to be due solely to the cord changes or to neuritis respectively.

The third case on which I wish to speak to you is one of another kind of paraplegia, also without loss of sensation, but due to a localized lesion, a patch of softening in the cord, which was the result of a trivial accident, and led to the patient's death six months afterwards.

G. N., a labourer, aged 45, sent to me by Dr. Mackay of Devizes, had always enjoyed good health, and had lived a temperate, steady life. Whilst walking home on December 26th, 1906, he slipped and fell on his back. He got up and walked home without difficulty, but, on account of pain in the back, had to stay in bed three or four days. He then returned to work for a week, but had to give up on account of pains in the back and around the abdomen and some numbness of the hands. When examined on February 27th, 1907, his condition was as follows:

A well-nourished and muscular man, complaining of pain in the back, and a girdle pain around the abdomen about the level of the umbilicus, with difficulty in walking. There was no obvious defect in gait, except occasionally the movements of the legs appeared slightly ataxic. Romberg's symptom was present. There was no loss of motor power in either arms or legs; grip of hands good; no muscular wasting. Slight blunting of sensibility to touch, pain, and temperature over the fingers of both hands. No sensory affection elsewhere. No tenderness over spines; no deformity of spine. Cranial nerves normal. Superficial and deep reflexes all present; knee-jerks slightly exaggerated; doubtful ankle clonus; plantar reflex flexor and very lively on both sides. The thoracic and abdominal organs and the urine were normal.

When seen again on March 13th his gait had become more ataxic, and walking was difficult. There was marked static ataxia. The loss of co-ordination of the movements of the legs was most evident when he was carrying out movements of the legs when lying on his back. There was defective sense of position of the legs. Electrical reactions normal. Micturition and defaecation normally performed.

At the beginning of April the condition was briefly as follows: inability to walk or to stand unless supported on both sides;

inco-ordination of movement and defective muscular sense in legs; when he tried to stand his legs seemed "all abroad." Increased muscular tone in legs, so that there was some rigidity on passive movement. A distinct band of hyperaesthesia (see figure) passed round the abdomen from 2 in. below the salient angle of the lower ribs to just below the umbilicus, about two to three inches in breadth, and extending from umbilicus midway to pubes. This remained constant during the further progress of the case, although its exact limits varied slightly on different days, and corresponded fairly to the area of the eleventh and part of the tenth dorsal roots. Knee and ankle jerks exaggerated. No decided ankle clonus; plantar reflex flexor at this time. Some irritability of the bladder. He felt

pain around the abdomen corresponding to hyperaesthetic zone, and along the course of the sciatic nerves. Arms and trunk unaffected, except for slightly defective sensation over the fingers as mentioned above.

In May cremasteric reflex had disappeared; tender spots were found over the twelfth dorsal spine; tips of tenth ribs and the backs of thighs and legs were hyperaesthetic. One month later, in June, the condition was decidedly worse. He could not stand or walk, and complained of much pain in the spine, hips, and groin. The band of hyperaesthesia around the abdomen was more extensive, reaching from the umbilicus to Poupart's ligament. There was slight defect of sensation (to all forms) over the fingers and toes, and also to hot and cold test tubes over the lower extremities; otherwise sensation normal.

All movements of the legs were ataxic, and the inco-ordination was increased when his eyes were shut. There was also now decided rigidity of both legs.

Reflexes.—Abdominal and cremasteric absent; plantar extensor on both sides. Knee-jerks and ankle-jerks increased; ankle clonus. He had now difficulty in emptying his bladder, acquiring the use of a catheter, and towards the end of his life lost control over the rectal sphincter. There was some general tenderness over the spinal column, but no persistent tenderness over any one spine.

He gradually became cachectic, and died somewhat suddenly in July.

At the post-mortem examination the following changes were found:

Lungs.—Hypostatic congestion of the lungs.

Heart.—Cavities dilated, muscle soft and flabby, and showed well-marked fatty degeneration.

Liver.—Enlarged, soft, and fatty.

Kidneys.—Congested throughout both cortex and medulla.

There was no other evidence of disease of the viscera.

On removing the cord, it was found that there was no fracture of the spine, but that the body of the ninth dorsal vertebrae had slipped forward, and its anterior lower edge projected about a quarter of an inch over the tenth. This was the only evidence of injury.

The meninges were healthy. A patch of softening could be felt in the cord extending for about half an inch at the level of the eleventh dorsal roots. The cord was hardened, and sections stained by Van Gieson's, Weigert's, and Marchi's methods. These showed a patch of softening at the level of the eleventh

and twelfth dorsal roots. The Marchi sections were most successful in showing the degenerations that had occurred.

Above the lesion numerous degenerated fibres were found in the ventral and dorsal cerebellar tracts and around the margin of the cord; also in the posterior median and external columns, but fewer in number. These latter could be traced in small numbers to the nuclei of the posterior columns in the medulla oblongata. At the level of the lesion, for about two segments, degenerated fibres appeared scattered throughout the spinal white matter. Below it they were found in large numbers around the margin of the cord, in the pyramidal tracts, in the posterior column, at first forming a comma-shaped tract in the central part of it, and then in the septo-marginal tract below. Weigert preparations showed the same lesions, but not so well, as the degeneration of the tracts was nowhere complete, many healthy fibres remaining in all of them.

The foregoing statement of the chief features of the case requires to be supplemented. The diagnosis was at first not at all easy. The man was not intelligent, and his general demeanour was such as to lead one to think that he was making the most of his troubles. Then the accident was a slight one; it seemed unlikely that very serious damage to the cord would be produced by a simple fall upon the back.

Although laid up for a few days immediately after the accident, there was a considerable interval in which he was able to get about before the symptoms became really severe. Such an interval of freedom from symptoms after an accident is often present in cases of traumatic neurosis. Again, when first seen there was no sign pointing to organic disease of the cord with absolute certainty, unless a slight degree of inco-ordination of some of the movements of the legs could be considered as such. The general aspect of the case was, therefore, at this time against any serious injury to the cord. The numbness of the fingers also afforded an anomalous symptom, not likely to be produced by the accident or in connexion with the disability in the legs, and therefore again rather in favour of some functional disturbance. This symptom was never satisfactorily accounted for, and the post-mortem examination threw no light upon it. The first sign that showed positively that there had been damage to the cord was the appearance about three months after the accident of the band of hyperaesthesia corresponding to the distribution of the tenth to eleventh dorsal nerve roots. This sign afterwards remained constantly present until the end of the case, and its persistence and distribution showed that it could only be due to a lesion of the cord at this level. Later on other signs appeared which indicated clearly that severe damage to the cord had been done; these showed that from the seat of lesion degeneration of the descending motor tracts, causing increased deep reflexes, extensor plantar reflex, and of the ascending tracts to the cerebellum, causing marked inco-ordination of the movement of the legs, was taking place.

But had it not been for the presence of this "focal sign" there might for some time longer have been doubt as to whether an actual lesion of the cord had occurred. Again, although in various "functional" nervous disease, traumatic or otherwise, areas of hyperaesthesia may be found, they do not correspond to the distribution of the spinal areas of the nerve roots.

The case thus shows the extreme importance of "focal" signs in diseases of the cord, first, in the general diagnosis from functional affections, and secondly, in the localization of the level of the lesion.

A further point worthy of remark is that in this case, in which there was partial damage done to the cord at the level of the tenth dorsal segment, the interference with voluntary movement and with co-ordination was marked, whilst throughout, except for the hyperaesthetic band corresponding to the level of origin of the damaged roots, sensation was practically unaffected until just before the end of the patient's life, and then only slightly impaired. This predominance of motor over sensory effects is not unusual in cases of transverse myelitis or, to speak more accurately, in patches of softening of the cord, when the lesion is a partial one, from whatever cause the disease may arise.

REFERENCES.

- ¹ Brain, Part cxvii, vol. xxx, 1907. ² Loc. cit.

UNDER the will of the late Mr. George Stubbley, of Carlton, Yorkshire, who died on April 14th, Batley Cottage Hospital and Wakefield Clayton Hospital each receive a sum of £250.