over chest. The patient was under observation in the medical wards for six weeks, and was treated with potass. iodid., rest, and ordinary diet. The pain continued severe, and there was some weakness of the right shoulder. The other symptoms remained the same, and the patient was transferred to Mr. Heath.

Operation.—On December 7th, 1887, Mr. Heath tied the subclavian artery in its third part. There was no difficulty in the operation beyond some venous bleeding, which was arrested by ligature. The vessel was tied with carbolised silk, and the wound closed with drainage. The common carotid was then tied just above the omo-hyoid.

After-History.—The wounds healed by first intention. There were no unfavourable symptoms after the operation, except that the cough was troublesome. On January 16th, 1888, the swelling at the right sterno-clavicular articulation was much smaller, and the pulsation less marked. The cough was more troublesome, and it was suggested that extension of the deeper part of the aneurysm had occurred. This continued until patient left the hospital on January 27th, 1888. The breathing was much better, and the difficulty in swallowing had nearly disappeared.

It was subsequently ascertained that the patient had died at Acton on May 1st, 1888, from bursting of the aneurysm internally, and that no *post-mortem* examination was made, hence it is impossible to make certain of the diagnosis.

In 1879 Mr. Barwell had the opportunity of following my suggestion of applying simultaneous ligatures to the carotid and subclavian arteries in a case of aortic aneurysm diagnosed as such, and his patient survived fifteen months. In 1880 his example was followed by Lediard, whose patient survived for eight and a-half months, and by Wyeth, whose patient lived a year. It should be noted that in all three cases Mr. Barwell's ox-aorta ligature was employed.

Returning now to Dr. Cockle's proposal to tie the left carotid in cases of aneurysm of the arch of the aorta, I first performed that operation in 1872, and the patient was greatly relieved, and lived over four and a-half years.¹ Mr. Holmes and Mr. Barwell had each a successful case of the kind, but Mr. Holmes's proved, after twelve years, to have been a case of malformation of the heart.² In 1874 I again performed the operation on a man, aged 44, who had undergone a variety of treatment, including iodide of potassium, venesection, and compression of the left carotid before he came under my care for supposed aneurysm of the aorta, which was making progress. The right radial pulse was not to be felt, and the arm had been swollen. I tied the left carotid with catgut on January 21st, but the patient died two hours afterwards from failure of the respiration. At the *post-mortem* examination a large innominate aneurysm was found, and the right subclavian artery was impervious up to the sac, death having resulted from want of blood supply to the brain.

In October, 1878, I tied the left carotid for the third time in a man, aged 44, with an aneurysm of the arch, chloroform being given for the skin incision only, and even then producing great cyanosis. Immediately on the ligature being tied the patient became pale, his heart weakened, and he became profoundly unconscious, the left pupil being widely dilated. After bleeding to 3x and the administration of brandy and beef-tea by oesophagus tube and enemata, the patient recovered consciousness, but never properly rallied, and died twenty hours after the operation. The *post-mortem* examination showed an aneurysm of the size of an orange from the junction of the ascending and transverse portions of the arch, both of which were considerably dilated and pressed on the trachea.

In 1885 I tied the left carotid for the fourth time in a man of 43 who had noticed a pulsating tumour of the episternal notch and left side of the chest for nine months, which was increasing in size. Cocaine was the anæsthetic used, 15 minims of a 20 per cent. solution being injected in the line of incision fifteen minutes before the operation. The artery was tied with carbolised silk. In the evening he was found to be aphasic, and the following morning there were facial paralysis and palsy of right arm. The wound healed and the aneurysm became more solid, and did not pulsate so forcibly as before the operation.

The power of speech gradually improved, and on the fifty-eighth day was reported good, though there was a tendency to reduplicate the first syllables of some words; the aneurysm remained *in statu quo*. He was transferred to the medical wards on the sixtieth day, and remained there two months, and was then discharged at his own request in much the same condition. I have ascertained that this patient died at Birkenhead in June, 1886, the certified cause of death being "thoracic aneurysm."

Dr. Elwin Harris has recorded in the *Clinical Soc. Trans.*, 1891, a case of aortic aneurysm occurring under his care in

the St. George's-in-the-East Infirmary, in which I tied the left carotid under cocaine only in March, 1890. The patient lived two months, and at the *post-mortem* examination the aneurysm was found to be filled with clot, mostly laminated.

Lastly, in November, 1890, I had a sixth case, thanks to the kindness of my friend, Dr. Robinson, of the Mile End Infirmary. The patient was a woman, aged 61, who had had symptoms of aneurysm of the aorta for three years, for which she had been treated in the London Hospital. In July, 1890, when admitted to the infirmary there was marked pulsation in the suprasternal notch, with a *bruit*. She was unable to lie down, and suffered from an irritating cough. After four months' treatment by rest and iodide of potassium without improvement, the operation was performed without an anæsthetic. Three days after it was noted that respiration, which had been distinctly noisy, was quite quiet. Six days after the operation she was able to sleep for seven hours.

In January, 1891, she complained of a return of pain in the right shoulder. In February, the tumour pulsating above the sternum was found to have undergone diminution, and all her symptoms becoming ameliorated she took her discharge on August 8th, 1891.

On September 3rd she was admitted into the London Hospital with a pulsating tumour at the inner end of the right clavicle, which could be felt just above it. About the middle of November she began to complain of great pain shooting through the sternum and between the shoulders, and died suddenly on November 29th, more than a year after my operation.

The *post-mortem* examination showed the first part of the arch to be uniformly dilated. From the right superior aspect of the transverse arch (between the innominate and left carotid) was a large aneurysmal sac of the size of an orange, with an opening into the aorta of the size of half-a-crown. The sac was adherent to the trachea, and almost completely filled with laminated clot.

It will thus be seen that of six cases in which I have tied the left carotid, as suggested by Dr. Cockle, the first was by far the most successful. In the second case the diagnosis was incorrect, the aneurysm proving to be innominate, and the proper vessel to have tied would have been the right carotid. In the third case the embarrassment of breathing caused by the anæsthetic was so great that I abandoned its use in my subsequent operations, and strongly advise the employment of cocaine in all future operations on the large vessels of the neck. The results, taken as a whole, are perhaps not very encouraging, but it must be borne in mind that in dealing with a practically incurable disease, a prolongation of life for even a few months may be worth attempting.

REFERENCES.

¹ On "Aneurysm," p. 61. ² *Lancet*, December 2nd, 1865. ³ *Ibid.*, January 5th, 1867. ⁴ *Lancet*, April 10th, 1869. ⁵ *System of Surgery*, vol. iii. ⁶ *Clin. Soc. Trans.*, vols. v, vi, x. ⁷ *Ibid.*, 1883.

REMARKS ON THE DISTRIBUTION OF CERTAIN SENSORY SPINAL ROOTS,

FOUNDED ON A CASE OF SPINAL CARIES.*

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So much attention has been given within recent years to the question of the sensory distribution of spinal nerve roots that it seems desirable to record individual cases which bear upon this point. I am greatly indebted to Professor Ogston for having had an opportunity of observing and recording the following case, in which it was possible, mainly by the help of the recent work of Head, Thorburn, and others, to arrive during life at an approximately accurate diagnosis of the site and extent of the lesion, as confirmed at the necropsy.

J. F., aged 42, a printer, was admitted into the Aberdeen Royal Infirmary under the care of Professor Ogston on April 30th, 1896. He complained that his legs had become weak, so that walking was difficult, and gave him a sensation of "pins and needles" up the legs. The following is a summary of notes taken while J. F. was in the infirmary:—
The family history showed nothing worthy of note, except that one brother was lame—attributed to an accident in childhood (tuberculous?).

* Read before the Aberdeen, Banff and Kincardine Branch of the British Medical Association.

Previous History of Patient.—At the age of 2, he had measles (?), which caused temporary loss of power in the legs. Ten or eleven years before admission, he was under treatment for a weak back—"spinal caries," he says. He wore a plaster of paris jacket for a month, and was then able to resume work, but since that time his back has been bent and he has suffered almost continuously from a feeling of constriction and pain in the lower region of the spine and round the loins, especially on the left side. This pain disappeared in bed, and was most intense at the moment when he got out of bed, gradually wearing off after he had walked a little. Eight years ago he had an abscess over the left trochanter major, and a cicatrix remains. For a few years he has been subject to bronchitis and attacks of "shortness of breath." Within the last year, when washing his face he had felt a staggering feeling and could scarcely stand erect. He drank heavily about twenty years ago, but has been temperate for many years. No history of specific disease can be made out.

History of Present Illness.—On April 8th, 1896 (that is, three weeks before admission) he felt a "sleepy" sensation in his left foot and noticed that his left leg trailed behind him in walking. A week later, he noted that he could not move the toes of his left foot and that his left leg occasionally "started" at night. A week later still, his right foot began to be affected with a sleepy sensation and, by this time, the numb feeling in the left foot had gradually spread up the leg to the thigh.

Condition on Admission.—(April 30th, 1896.) The patient is short of stature and poorly nourished, weighing 7 st. 8 lbs. Examination of the spine shows a well marked angular kyphotic curvature about the mid-dorsal region, while the normal lumbar and cervical curves are exaggerated. There is also a certain amount of scoliosis. The skin over the kyphosis is red and soft, but there is no note of pain or tenderness there. There is no loss of power in the arms and little, if any, in the right leg. There is defective power of movement at all joints of the left leg, especially at the ankle, where the range and power of movement are very limited. The left ankle is held rigid. Objectively, sensation to touch seems good everywhere, except about the toes of the left foot. There is some inaccuracy in distinguishing heat from cold, especially on the feet. Subjectively, there is a numb and cold feeling in the feet, which perspire freely. The knee-jerks and plantar reflexes are both exaggerated. There is no affection of the sphincters; bowels are constipated. The muscles of the leg show fair nutrition, but are distinctly wasted all over, from the gluteal region downwards. There is no special wasting of any muscle or group of muscles. The heart and lungs are normal, and the urine shows nothing abnormal (sp. gr. 1021, acid, light straw colour, no albumen or sugar).

After-History.—May 11th. The numb feeling is gradually extending up the right leg, which has now become weak and twitches occasionally. May 23rd. The patient was transferred to the medical side of the infirmary, under Professor Finlay. He cannot move either leg voluntarily. Both legs are often drawn up involuntarily, and he cannot straighten them. Sensation is impaired on both legs, especially the right. He feels a touch, but it appears quite different from a touch on the arms. He cannot definitely distinguish between heat and cold on the legs, especially the right. Subjectively, he has "pins and needles" in the legs. The plantar reflexes and knee-jerks are much exaggerated, especially on the left side. There is marked ankle clonus and knee clonus, especially left. The patient's condition got continuously worse. The spastic condition of the legs increased: the legs became markedly rigid, the knee-jerks tremendously exaggerated, and the ankle clonus and knee clonus very readily elicited, especially on the left side. Muscular spasms of the legs were frequent during the night. There was absolute paraplegia until August 28th, when a slight power of movement of the toes of the left foot was noted. The numb feeling in the legs spread up as far as the pelvis, and complete loss of sensation of all forms gradually developed on the legs as far up as the groins. On June 30th, it was noted that all the muscles of both legs reacted to both faradism and galvanism, and that sometimes the reaction was rather excessive, for example, in the adductors of the right thigh. From the end of June onwards, there was complete retention of urine, which necessitated continuous catheterisation. Only once was there incontinence of urine. The bowels remained very constipated. He continued to complain of pain in the lower region of the spine, but there was no note of pain or tenderness in the region of the kyphosis. The temperature remained rather subnormal, ranging usually between 97° and 98.6° F., with an occasional evening temperature of 99° to 100° F.

He returned to the care of Professor Ogston, who performed the operation of laminectomy on September 14th, 1896. The spines of the fifth, sixth, and seventh dorsal vertebrae were nipped off with bonepliers, and the corresponding neural arches were removed. The fifth and sixth neural arches, especially the sixth, were seen to press on the spinal cord and make a visible constriction; the sixth was grown to the dura mater by bone. It was not necessary to open the dura. No pus was found. The wound of operation healed by first intention.

History Subsequent to Operation.—Improvement in the patient's condition was observed as early as two days after the operation, for on September 16th it was noted that some sensation was present in the legs, especially the right one. He felt manual pressure applied to either leg, but was not certain which limb had been handled. He could feel the stab of a pin applied to any part of either leg, and there was considerable recovery of the power of distinguishing heat and cold. On September 18th (that is, four days after the operation) he was able to empty his bladder partially without a catheter. On September 25th he completely emptied his bladder without the aid of a catheter, having been unable to do so for over sixteen weeks. Sensation had improved on the legs, and he had re-acquired some power of the coarser movements of the right leg, while the left was in the same condition as before operation.

CONDITION OF THE NERVOUS SYSTEM THREE WEEKS AFTER OPERATION.

I was asked by Professor Ogston to examine the patient. The following is a synopsis of the notes made on October 5th, 1896. The spine cannot be examined owing to the weakness remaining after the operation. Both legs are markedly rigid, especially the left. The right leg shows strong power of flexion and extension at the hip and knee, while dorsal flexion of the ankle is weak, and is effected mainly by the tibialis anticus and extensor longus hallucis, so that on an attempt to flex the ankle the sole

of the foot looks inwards. The left leg shows movements at the hip and knee fairly strong, but weaker than on the right side; there is practically no power of dorsal flexion of the ankle, and very slight power of flexion and extension of the toes.

Sensation.—On the front of the trunk, at a level about 2 inches beneath the umbilicus, there is a fairly sharp line, separating the upper part of the abdomen where tactile sensation is perfect from the lower part where the sensation of touch is absent. On the legs, especially the left, the sensation of touch is very indefinite all over; it is very deficient (absent?) on the front and inner sides of the thighs, on the dorsum of both feet, and on the left sole. There is no defect of tactile sensation on the genital organs. Localisation of touch is markedly defective on the legs, a touch at one part being often referred to quite a distant region on the same or even the opposite leg. The sensation of pain seems normal on the legs, pain being felt as such everywhere; localisation is, however, slightly defective. The temperature sense is very defective on the legs; hot is often called cold, while cold as a rule is appreciated aright, but is sometimes called hot, especially about the feet. The muscular sense is also very defective; there is no appreciation of passive movement of the toes of either foot, and very little appreciation of movements at the ankles and knees.

Reflexes.—The knee-jerks are tremendously exaggerated, and bot

Vertical lines denote marked defect (or absence) of tactile sensibility. Horizontal lines denote defect of tactile sensibility. (b = 2 inches).

ankle clonus and rectus clonus are very readily obtained on either side. No cremasteric reflex can be elicited on either side. The plantar reflexes are much increased. No abdominal reflex can be obtained below the level of the umbilicus, while above that level the abdominal and epigastric reflexes are readily elicited on the right side, but are absent on the left side. There are no bladder troubles, but marked constipation is present. Nothing abnormal can be detected about the arms or in the region of the cranial nerves, except fine tremor of the lips and tongue, probably of alcoholic origin. The urine is acid and normal in other respects.

AFTER-HISTORY.

October 7th.—There is some return of tactile sensation on the dorsum of both feet and on the sole of the left foot, and there is slight appreciation of passive movements of the toes of the left foot.

From this time till he left the infirmary on January 20th, 1897, there was a gradual falling off in the patient's condition. His legs regained no further power and became more and more rigid, with more frequent twitching and drawing up of the legs, which caused very severe pain over the abdomen and in the lumbar region. Practically no objective change in sensation was noted, as compared with the condition on October 5th and 7th. He complained greatly of pain in the spine, in the region of the lumbodorsal junction, where there was distinct tenderness on pressure, but no irregularity; while no tenderness could be elicited by pressure over the seat of operation. The pain in the spine would often shoot round the pelvis, especially on the left side, about the level of the iliac crest. There was marked fibrillary tremor of the abdominal muscles on both sides, as high up as the umbilicus. From October 31st onwards, he suffered from cystitis, and the catheter revealed a considerable quantity of strongly ammoniacal residual urine. This condition improved under treatment, but recurred again and again; and even when the urine became acid, there was a quantity of residual urine. About January 1st,

1897, he became affected mentally; he had delusions, and sometimes got very excitable and noisy, imagining that he was being drugged, etc. After leaving the infirmary on January 20th, 1897, his general condition remained the same, and his chief complaint was the difficulty of micturition, which required the almost constant use of a catheter. He came under the care of the medical officers of the Aberdeen Dispensary, and finally of the Parish Council, and died on August 19th, 1897, two days after the commencement of an attack of acute pleurisy.

From the examination of the patient on October 5th, 1896, the following conclusions were arrived at:—

A.—THE NATURE OF THE LESION.

The whole clinical course of the illness pointed to an extramedullary lesion rather than to a primary myelitis or growth in the cord itself. The following points were in favour of a lesion causing compression of the cord from without:—(1) the feeling of constriction and pain round the abdomen for years before the onset of the paraplegia, these root symptoms being usually a late and not very prominent symptom in myelitis and growths within the cord; (2) the rather gradual onset of symptoms in the legs, beginning in one leg and spreading along it, and then similarly affecting the other leg; (3) the rapid recovery—up to a certain point—of sensation, motion, and vesical function after the operation of laminectomy.

As to the cause of the compression, there seemed little doubt that caries of the spine was the lesion. Although no very distinct tuberculous family history could be made out,¹ there were the following points in the personal history of the patient:—(1) Ten or eleven years before, he had been treated for weakness of the back, which, according to his own account, had been diagnosed as “spinal caries,” and since that time he had the angular curvature of the spine at the mid-dorsal region; (2) eight years before, he had an abscess over the left trochanter major, probably tuberculous, the scar of which still remained at the time of examination. (3) Although he had suffered from root pains for many years, they had never reached the intensity so common in cases of growths in the vertebrae; further, although these pains disappeared in bed and appeared when he rose, they gradually wore off after he had walked a little. There was thus not the relation between movement and intensity of the pains so constantly seen in vertebral growths. The pains, too, were confined to a definite limited area in the lumbo-dorsal region of the cord, thus making the diagnosis of pachymeningitis improbable.

The following facts were rather against the diagnosis of caries:—(1) The first symptoms, namely, weakness of the back, appeared about the age of 40. Gowers,² however, has known caries of the spine commence at 50, and says it has been met with as late as 70. (2) The symptoms appeared first in one leg and then in the other. In caries of the spine, “both legs usually suffer together” (Gowers), but this rule is not invariable. Gowers³ mentions a case, verified *post mortem*, in which the first symptom in the legs was “pins and needles” about the right knee; in three days the right leg became almost powerless; two weeks later, there was a similar sensation in the left knee, and, a week later still, in the left foot. During the next three weeks the left leg became powerless, so that at the end of six weeks from the onset both legs were motionless. This case has many points in common with the case of J. F., who first felt a sleepy sensation in the left foot, which gradually spread up the leg. One week later the left leg became weak; one week later still there was the same sleepy sensation of the right foot, and the weakness and numbness in the legs gradually increased, so that at the end of six weeks from the onset there was complete paraplegia and numbness of both legs up to the groins.

B.—THE SITE AND EXTENT OF THE LESION.

Granted that caries of the spine was the probable lesion, it was more difficult to decide on the site. The most natural conclusion was that the well-marked angular curvature at the mid-dorsal region marked the site, and this seemed to be confirmed by the condition found by Dr. Ogston on performing laminectomy there, namely, a visible constriction of the cord opposite the fifth and sixth dorsal neural arches.

Against this view, however, were these facts:—(1) The position of the long-continued pain in the lower part of the spine, about the lumbo-dorsal junction. (2) The position of the girdle pain, far below the level of the umbilicus; a lesion

about the sixth dorsal vertebra would have caused a girdle pain distinctly above the umbilicus, probably nearer the xiphoid cartilage. (3) The position of the upper limit of anaesthesia, namely, about two inches beneath the umbilicus. This was too low to be explained by a lesion as high as the sixth dorsal vertebra. Many recorded cases might be quoted to prove this, but it will be sufficient to cite two. Thorburn⁴ records a case of fracture dislocation of the sixth dorsal vertebra, in which *post mortem* the cord was found completely divided opposite the sixth dorsal vertebra. There was complete anaesthesia on the trunk as high as the eighth rib. Again, Hale White⁵ records a case of spinal caries, with backward projection of the seventh to the tenth dorsal spines, in which *post mortem* the cord was found softened and compressed opposite the seventh dorsal vertebra. The upper limit of loss of sensation on the trunk was well above the umbilicus.

Much attention has been devoted within recent years to the subject of the sensory distribution of the spinal segments and nerve roots, and the results obtained by workers, following entirely different lines, are wonderfully similar. Thus Thorburn, Starr, and others have studied the question from the clinical point of view, that is, they have carefully observed cases of injuries and disease of the cord and compared the areas of anaesthesia found in these cases with the *post-mortem* results. Head and Mackenzie, on the other hand, have studied the areas affected by herpes zoster and the areas of pain and cutaneous hyperaesthesia (or rather hyperalgesia) seen in disease of the various viscera. Head⁶ has found that there is a definite limited number of these areas, and he assumes, from comparison with the results of gross organic lesions of the cord, that each of these areas represents the distribution of a particular segment of the cord. According to him, the tenth dorsal segment (that is, the segment of the cord from which the tenth dorsal roots spring) is connected with that zone of the body, the upper border of which passes from the first lumbar spine to the umbilicus, and the lower border from the fourth lumbar spine to a point about $2\frac{1}{2}$ to 3 inches below the umbilicus in the middle line of the abdomen in front. Thorburn,⁷ however, thinks it probable that the umbilicus lies “not at the junction of the ninth and tenth dorsal fields, but certainly no higher than the lowest part of the tenth,” and he is inclined to place it, with “Quain,” between the tenth and eleventh. The upper limit of anaesthesia in the case of J. F. seemed, therefore, to point to a lesion of the cord not higher at least than the level between the tenth and eleventh dorsal segments. This level was seen, on consulting the diagram framed by Gowers,⁸ to correspond to the space between the bodies of the ninth and tenth dorsal vertebrae and to the tip of the ninth dorsal spine.

The conclusion arrived at, therefore, as to the upper limit of compression was that it did not implicate the spine higher than the body of the tenth dorsal vertebra, or the origin of the eleventh dorsal roots. The fact that, above the line of anaesthesia, the abdominal reflexes could not be elicited, especially on the left side, was taken to indicate secondary myelitis, which is so common in cases of compression of the cord.

[NOTE.—Professor Reid⁹ has found, from examination of six spinal cords, that the level of origin of the spinal nerve roots relatively to the spines of the vertebrae varies considerably. By his kindness, I have recently had the opportunity of examining several of these cords, and I find that the site of a lesion, implicating the eleventh dorsal roots, and leaving the tenth intact, corresponds fairly constantly to the level of the middle or lower third of the ninth dorsal vertebra and to the interval between the eighth and ninth dorsal spines. Thus the level of origin of the tenth dorsal root, relatively to the bodies or spines of the vertebrae, may be rather higher than is depicted in the diagram of Gowers, but is still a long distance beneath the level of the sixth neural arch, where the operation showed constriction of the cord in the case of J. F.]

The probable lower limit of the lesion was determined from consideration of the lowest level to which the nerve roots (or the central functions of the cord) were damaged. The following facts seem to indicate a lesion implicating the second lumbar roots but not lower: (1) The cremasteric reflexes, effected by the first and second lumbar nerves, were quite absent. (2) The knee-jerks, effected by the second, third, and fourth lumbar nerves, were so excessive that it seemed unlikely that they could be due to the fourth lumbar nerves alone. (3) There was no sign of special wasting in the

muscles which receive their nerve supply, partially or wholly, from the third lumbar nerves (Ferrier and Yeo,¹⁰ Paterson¹¹). (4) Although the chart, showing the area of anæsthesia after partial relief of the compression by laminectomy, indicated affection of the third lumbar roots according to Thorburn,⁴ the other facts mentioned above made one inclined to adopt the view of Head⁸ and Starr,¹² according to whom the second lumbar roots would be the lowest involved.

The diagnosis, therefore, made during life was that the lesion, probably of the nature of caries of the spine, implicated the cord by pressure between the level of origin of the eleventh dorsal and second lumbar roots inclusive.

I am indebted to Dr. J. Scott Riddell for having had an opportunity of performing a very partial necropsy. The condition found was as follows:

NECROPSY.

An incision was made over the spine, when a considerable quantity of pus escaped, coming apparently from the lower dorsal region of the spine. The spinal canal was opened upwards and downwards from the site of laminectomy. No irregularity of the spine was noted in the lower dorsal or upper lumbar region. The cord was removed through the whole lumbar region and the greater part of the dorsal region, and it was found that there was marked thickening of the dura mater along the whole length of cord removed. There was no pus within the dura. The bodies of certain vertebrae were in great part destroyed and replaced by old caseous nodules, which, being firmly adherent to the thickened dura, were removed along with the cord and were found to project through the dura, causing depressions in the cord opposite. The mass pressed on the anterior and lateral aspects of the cord as far back as, and including the origin of, the posterior roots, while the dura was very thick and firm over the posterior columns. The extent of cord compressed in this way included the part from above the origin of the eleventh dorsal roots to the level of the origin of the second lumbar roots. The cord showed a marked constriction between the origins of the tenth and eleventh dorsal roots. The lower two dorsal roots were much atrophied, and contrasted forcibly in point of size with the tenth dorsal root, which seemed normal. The nodular mass, when examined microscopically, was found to show beautiful old tubercles.

The probable course of events in this case is interesting. There seems little doubt that J. F. did suffer, many years before, from spinal caries about the region of the sixth dorsal vertebra. This caused the angular curvature, and, although the cord remained compressed as shown at the time of operation, any signs of paralysis that may have been present passed away. The disease at that site became quiescent, but another focus appeared lower down, causing the long-continued root pains, and eventually ending in complete paraplegia. The disease at that site did not cause any obvious irregularity of the spinous processes. The rapid improvement after the operation, performed by Dr. Ogston at the level of the sixth dorsal vertebra, was no doubt due to the fact that removal of pressure at any part of the cord would relieve compression on the cord at any and every other part.

The order of recovery of the conducting functions of the cord was in accordance with what is the general rule in cases of compression of the cord or spinal nerves, that is, sensation first and most, motor power last and least. The relative degree of affection of the various forms of sensation, as shown by the record (see diagram, p. 479) taken soon after the operation, was unusual. As a rule, in organic lesions of the cord or its roots, the loss to touch is less extensive and less definite than the loss to pain, while in J. F. there was a distinct area of anæsthesia (v. diagram) corresponding to the distribution of certain dorsal and lumbar roots, and the sensation of touch was markedly defective on the rest of the legs, whereas painful sensation was appreciated as such everywhere. The degree of affection of the sensations of pain and temperature was also unusually dissimilar. There could be no doubt as to the defect of the power of differentiating heat and cold at places where a painful sensation was acutely felt. It is difficult to say why the various forms of sensation should be affected in different degrees in cases of compression of the cord. Attempts have been made, without much success, to find an explanation in the degree of pressure exerted on the different parts of the cord. Thus it is supposed that pressure on the lateral parts of the cord would affect pain (and temperature) mainly, and pressure on the posterior columns would involve touch. If this were true, there should have been more affection of pain than of touch in the case of J. F., as the caseous mass pressed on the anterior and lateral aspects of the cord, while the posterior columns were intact (except for the thickened dura).

Inferences from individual cases are always uncertain, but

any deduction that may be drawn from the case of J. F., with regard to the sensory distribution of the various spinal roots, would accord with the views of Head rather than of Thorburn. As the eleventh dorsal roots were undoubtedly much compressed, and the upper limit of anæsthesia was well below the umbilicus, the case would indicate that the area supplied by the eleventh dorsal root does not reach so high as the umbilicus, as Thorburn is inclined to believe. Again the area of anæsthesia embraced the region supplied by the third lumbar root according to Thorburn, whereas *post mortem* that root was seen to be free from pressure except that exerted by the thickened dura, which pressed quite as much on the fourth and fifth lumbar roots as on the third.

NOTES AND REFERENCES.

¹ Since these notes were made I have ascertained that there was a distinct tuberculous family history. ² *Diseases of the Nervous System*, second edition, vol. i, p. 241. ³ *Op. cit.*, p. 248. ⁴ *A Contribution to the Surgery of the Spinal Cord*, 1889, p. 69. ⁵ *Brain*, vol. xvi, 1893, pp. 375-381. ⁶ *Brain*, vol. xvi, 1893, pp. 1-133. ⁷ *Brain*, vol. xvi, 1893, p. 368, and *Medical Annual*, 1896, pp. 97-101. ⁸ *Op. cit.*, p. 163. ⁹ *Journal of Anatomy and Physiology*, vol. xxiii, 1889, pp. 341-353. ¹⁰ *Brain*, vol. iv, 1882, p. 226. ¹¹ *Journal of Anatomy and Physiology*, vol. xxviii, 1894, p. 183. ¹² *American Journal of the Medical Sciences*, 1892.

REMARKS

ON

PYLORECTOMY,

WITH NOTES OF TWO CASES, AND A DESCRIPTION OF A NEW METHOD OF ATTACHING THE DIVIDED STOMACH TO THE CUT DUODENUM.

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In January, 1896, I operated on a patient for simple stricture due to cicatricial contraction of the pylorus. When the abdomen was opened the stomach and duodenum appeared to be united by a mere cord, and it was obvious that the ordinary operation of pyloroplasty would not meet the requirements of the case. The operation then performed suggested to me the modification of pylorotomy to be described; and finding that it could be satisfactorily done on corpses, and that its details were in accordance with what I believed should be principles in gastro-intestinal surgery, I described the proposed operation to surgical colleagues in the infirmary here six months before the opportunity of practising it presented itself. Before giving a report of the cases I deem it proper to relate certain points in connection with the operation. In the preparation of the patients, 5 grains of calomel, followed by small, frequently-repeated doses of a saline purgative two days before the operation, thoroughly cleared the gastro-intestinal tract. A rest in bed and a limited diet of equal parts of milk and barley water, with $\frac{3}{4}$ ss of whisky and $\frac{3}{4}$ ss of water half an hour before the operation, completed the general preparations. The stomach was not washed out in either. The abdominal wall was prepared in the usual way, by a thorough cleansing and disinfection for several hours with a towel wrung out of a 1 in 1,000 corrosive sublimate solution, and every detail of the operation was conducted with scrupulous antiseptic precautions.

The pylorus was then excised in both cases in the usual way.

The modification I have introduced refers to the method of connecting the remainder of the stomach to the cut duodenum, and will be best understood by a reference to the accompanying diagram (Fig. 3) made by Dr. W. G. Richardson. The opening into the stomach is, as would be expected (and as is shown in the diagram), larger than the opening into the duodenum made by cutting it transversely. In order to overcome the difficulty of uniting the two without the aid of complicated sutures an incision over 1 inch in length was made down the centre of the anterior wall of the already transversely divided duodenum. By spreading the longitudinal cut transversely the duodenal opening was so widened as to correspond approximately with the larger opening in the stomach. All that now remained was to connect the two by a satisfactory suture. Before describing the