nerve in the form of a "tension suture." This will tend to produce an intraneural neuroma at a greater distance from the suture line both above and below; and an additional obstacle is placed in the pathway of the regenerating axons, more particularly when the suture material is one which causes much fibroblastic reaction. This is diagrammatically represented in Fig. 11.

CONCLUSIONS.

(1) While plain thread or silk sutures give rise to no irritation, they are relatively inabsorbable.

(2) "Plain sterilized" or "plain iodized gut" are of value by reason of their rapid absorbability, but their low degree of tensile strength and their slightly irritating nature counteract these advantages.

(3) "Japanese silkworm gut" has great tensile strength, and in rate of absorbability and the reaction which it causes holds a place midway between groups (1) and (2).

(4) Chemical antiseptics should be avoided in all suture materials.

In conclusion, we desire to express our gratitude to the Medical Research Committee, whose generosity in equipping and supporting the Research Laboratory have made this work possible, and to Lieut. Colonel D. K. McDowell, C.M.G., whose interest and goodwill have greatly helped our endeavours to combine some research work with the routine duties of a busy hospital.

COMBINED SCLEROSIS WITH HETEROTOPIA OF THE SPINAL CORD.

BY

GEORGE E. RENNIE, M.D., F.R.C.P. LOND.,

SENIOR PHYSICIAN AND LECTURER IN CLINICAL MEDICINE,
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY;

AND

OLIVER LATHAM, M.B., CH.M. SYD.,

DIRECTOR OF THE PATHOLOGICAL LABORATORY, DEPARTMENT
OF LUNACY, NEW SOUTH WALES.

[With Special Plate.]

THE following case, in which the diagnosis of disseminated sclerosis of the spinal type was made during life, presents many points of interest, especially in connexion with the existence of the congenital or developmental defect observed *post mortem*.

M. R., a married woman, was admitted to the Royal Prince Alfred Hospital under my care on April 29th, 1918, complaining of weakness of the legs and some numbness of the feet for about a year. There was nothing of note in her family history.

HISTORY.

She had always worked hard indoors; had been married seven years and had had two healthy children; no miscarriages or stillborn children. There was no history of alcoholism or of venereal disease.

She had had an attack of colitis two years previously, and had not felt well since then. One year ago she began to notice that her legs were getting weak and stiff at the joints; they always felt tired, but she had no pain in them. About the same time she noticed numbness in the soles of the feet. These symptoms became aggravated gradually until she completely lost all power of walking about one month before being admitted to hospital. For some weeks previous she had suffered much from indigestion and constipation, but there was no disturbance of the sphincters.

CONDITION ON ADMISSION.

She was thin but had a good colour. She had no pain or discomfort, and although unable to stand or walk she was quite cheerful, and expressed confidence in her ultimate recovery. The temperature was normal, pulse 116, respirations 24. Urine acid, specific gravity 1020, contained no albumin, sugar, or pus. She was intelligent, had a good memory, was not emotional, and slept well. Nothing abnormal was detected on examination of the cranium or spine. The cranial nerves were all normal. There was no weakness or stiffness in the arms, but all movements of the legs were impaired by marked spasticity, and she was quite unable to walk or stand, even with assistance. The only disturbance of sensation detected was some slight diminution in acuity of tactile sensation on the outer side of both thighs, and some hyperaesthesia on the soles of the feet. Wrist and elbow jerks were present, and equal on both sides but not exaggerated. Both knee-jerks were much exaggerated, and patella and ankle clonus were easily elicited on both sides. There was no disturbance of the sphincters and no trophic changes.

Examination of the blood showed the following result: Red cells, 3,336,000; leucocytes, 4,000; haemoglobin value, 44 per cent.; colour index, 0.65. Differential count of leucocytes: Neutrophile polymorphs, 73 per cent.; small lymphocytes, 18 per cent.; large and transitionals, 8 per cent.; eosinophils, 1 per cent.

Nothing abnormal was detected on examination of the heart, lungs, and abdomen. Lumbar puncture was performed on April 30th, and 35 c.cm. of clear fluid was obtained under pressure. This fluid was found to contain no cellular deposit, and the "Wassermann" reaction with the fluid undiluted and in dilutions of 1 in 2 and 1 in 5 was in each case negative. On the other hand, the "Wassermann" reaction with the blood was positive on the two occasions on which it was tested.

The patient thus presented the clinical picture of "paraplegia in extension," and there was nothing to indicate any immediate unfavourable change in her condition.

AFTER-HISTORY: DEATH.

On the night of May 3rd the patient suddenly became delirious and noisy, but could collect herself sufficiently to answer questions. She said she was very frightened, but could give no reason for being frightened. This state lasted for about an hour; but later in the evening she again became noisy, and passed through a similar but more severe and prolonged attack.

Next day she was strange in her manner, and had delusions about the other patients; she was afraid of being sent away to a mental hospital. On May 5th she was very drowsy and objected to take any medicine. It was now noted that the extensor muscles were relaxed, that she preferred to lie with her legs flexed, and that they were spastic.

On May 8th it was noted that she had well-marked nystagmus and some weakness of the left external rectus muscle of the eye, but no other alteration in the cranial nerves could be detected. The bladder became distended and 30 oz. of urine had to be drawn off by catheter. The urine was clear and contained no albumin, sugar, blood, or pus. She also developed incontinence of the rectal sphincter.

On May 10th it was reported that she had had a quiet night and had passed urine voluntarily at midnight, the urine again being quite normal. At 6 a.m. she became unconscious, the bladder was distended and 32 oz. of urine drawn off by catheter contained a large amount of blood. She died the same day without recovering consciousness and with no material change in her condition except a sudden rise of temperature to 103° and pulse 166.

PATHOLOGICAL REPORT BY DR. LATHAM.

The spinal cord was examined at the Pathological Laboratory of the Lunacy Department of the New South Wales Government.

A. Macroscopical.

The material was received in 10 per cent. formalin, and on naked-eye examination, after dividing the dura mater longitudinally along the ventral surface, there was noted, besides conspicuous tortuous blood vessels, several alterations in the size and appearance of the cord at various levels.

For instance, between the middle and lower cervical regions a constriction appeared, so that the transverse diameter was quickly reduced from 12 to 9 mm., the vertical dimensions remaining the same; the cord again widened until the transverse measured 16 mm. (just above "c" in Fig. 1, Plate II). The constriction was really of greater degree, as may be seen in Fig. 2—a longitudinal section of the part.

About the seventh dorsal the cord had the appearance of having been nipped by a forceps at its removal, and a little lower down there appeared to be a condition of "heterotopia" around one of the posterior horns. As, however, the whole condition much resembled that obtaining in two cords recently described by one of us,¹ and considered to be due to congenital abnormalities, the parts were carefully embedded in celloidin, it having been ascertained that the tissue was exceedingly brittle and fragile. The upper cervical and all the lumbar and sacral regions appeared normal.

B. Microscopical.

The following staining methods were employed: (1) For myelin sheaths, Robertson's Heller's method after mordanting in Weigert's fluids; for degenerating myelin, Busch's modification of Marchi's method; for nerve cells, round-cell infiltration and organisms, Nissl's stain; for neuroglia, a modified van Gieson, using azofuchsin G; and for vascular conditions haematoxylin and eosin. Sections were made from the upper and lower lumbar, lower and middle dorsal, and cervical levels. The myelin stain at once revealed a system degeneration involving bilaterally Goll's and part of Burdach's fasciculi, the crossed and direct pyramidal (and probably part of the vestibulo-spinal and the rubro-spinal too), as well as the direct cerebellar tracts—that is, corresponding to the pathological entity known as Gowers's ataxic paraplegia. There was no evidence of any such lesion above or below these constrictions such as would suggest that these latter had produced a transverse myelitis. It was also easily apparent that the posterior and anterior spinal nerve roots and the root zones were intact. The pia arachnoid was normal; some large round cells with small nuclei chiefly over those parts of the cord which were sclerosed were by Busch's method shown to be compound granular cells, and could be traced right into the cord along the adventitial lymph spaces of the emerging blood vessels, and to be loaded with myelin; in the ventral fissure a few

small round cells were noted, but no general small round-cell infiltration of the pia arachnoid and adventitial sheaths of the blood vessels obtained, such as forms the typical picture of tabes. No pus cells were seen anywhere. Endarteritis obliterans was not observed, but the smallest blood vessels of the cord exhibited arterio-capillary fibrosis, which narrowed the lumen and thickened the outer coats. In the sclerosed parts the blood vessels were engorged and their lymph channels filled with compound granular cells. Occasionally numerous capillary haemorrhages were noted, and some of the sclerosed areas might well have been areas of softening. In these areas the nerve fibres showed a twisted course, with swollen spiral axis cylinders and dilated thinned ill-staining myelin sheaths, and these conditions, especially near the constrictions, indicated a state of things which must have had gradual onset during life. The Busch-Marchi method did not take very well, but nevertheless showed clearly the many degenerating fibres in the sclerosed areas, but did not show any further extension of the diseased process in these areas or any other ones, in marked contrast to the results we obtained in a case of Friedreich's ataxia in a young child recently described by me,² where the ordinary myelin stain showed extensive sclerosis of the posterior columns of long standing, while the Busch-Marchi method revealed a further but quite recent degeneration in the crossed pyramidal, as well as the periphery of the cord, from one posterior horn ventrally right round and including the ventral fissure to the other posterior horn. In the case I now describe the degeneration stopped short and did not involve Gowers's antero-lateral tract nor the more anterior tracts till the inner part of the vestibulo-spinal was reached.

The neuroglial elements were readily seen round the central canal and sclerosed areas, but were brittle, and where there were many haemorrhages were granular or scarcely seen.

The anterior cornual cells stained well and, save for a fair amount of pigment, must be accounted normal; Clark's column cells similarly were functional, but were somewhat obscured by a rather abnormal development of tangle skeins of fine medullated fibres which were found just here.

Appearance Noted at the Different Levels, Heller-Pal Stain.

The mid-cervical shows the tract of Goll sclerosed right to the commissure as well as the posterior edge of Burdach's; also the cortico-spinal, rubro-spinal, vestibulo-spinal, and cortico-spinal ventralis.

The lower cervical shows the first constriction. No regular tracts nor grey matter can be made out, but numerous dilated myelin sheaths and capillary haemorrhages. In tangential section it may be seen in Plate II, Fig. 2, at "B1."

The upper and middle dorsal levels show the greatest involvement of the posterior fasciculi with many capillary haemorrhages in the cortico-spinal tracts and near one posterior horn the commencement of that heterotopia which is such a marked feature of the next section.

Lower Dorsal.—The tracts posterior to the commissure are altogether disturbed by a peculiar displacement of apparently one cortico-spinal and spino-cerebellar tract which form a cap over the posterior spinal nerve roots. The other side of the cord is also affected and the anterior horns quite dissimilar (Fig. 9, Plate II). Clark's column cells are here well marked. Other sections near this level show even greater abnormalities (best understood by examining the accompanying diagrams).

The lumbar regions simply reveal system degenerations involving the fasciculi of Goll (leaving the septal area free), and the cortico-spinal tracts.

It must be mentioned that where the abnormalities are most marked was found most evidences of haemorrhages, degenerative myelitis, and a sweeping bare of all but a few belated twisted swollen myelinated fibres associated with an absence of neuroglial framework.

In Fig. 11 and Fig. 12, Plate II, Busch-Marchi preparations of the lumbar and cervical levels show that although the sclerosed areas come out nearly bare of myelin fibres when examined after Pal's stain yet many of the fibres have only recently degenerated, and can still take on a Marchi reaction, although only poorly. From the greater number of affected fibres in the cortico-spinal tracts one might suggest these were last affected.

The Possible Etiology as Revealed by the Pathological Findings.

We have a cord showing a combined sclerosis of moderately recent causation. The complete absence of damage or any pathological condition in the posterior nerve roots seems to rule out a lymphogenous affection reaching the cord by the lymph system carrying toxins towards the cord.³

These reasons, together with the absence of small round-cell infiltration of the pia arachnoid and non-involvement of the root zone, quite rule out tabes. The absence of any foci of pus cells or inflammatory reaction effectually dismisses the commoner infective conditions. No blood vessels were noted showing specific signs of syphilis, although many hold lues to be a common auxiliary in the etiology of fibro-hyaline degeneration of the short vessels

of the first layer of the cortex. No part of the affected area resembled a gumma. Many of the fibres affected came from cells in Clark's column which gave rise to those fibres—that is to say, only part of the neurone was affected, and that part the long intraspinal axon chiefly supplied by small arteries coming from the posterior and lateral pial blood vessels.

It would appear, then, that inasmuch as the condition was bilateral there was rather a blood condition, a deficiency rather than an irritative toxin, whereby we got a slow decaying rather than an inflammatory reaction of the myelin fibres as well as of the neuroglial framework, as evidenced by the brittleness of the sections, and the granular appearance and poor staining of the glia in certain areas. This would appear to support the contention of Marie Williamson⁴ and Campbell⁵ that these conditions are due to some generalized blood condition which specially affects those tracts where the blood supply is likely to be relatively indifferent. In this connexion, however, it is difficult to account for the escape of Gowers's tract among others when the ventral cortico-spinal tracts were so badly affected.

In view of the *post-mortem* findings in the spinal cord, it is interesting to note that the patient's life could be divided into four periods: (1) A period of good health and activity with no evidence of any weakness or defect which could be ascribed to the heterotopia of the spinal cord. As this must have been a congenital condition we must conclude that it was in no way connected with the nervous symptoms subsequently manifested by the patient. (2) An attack of colitis, the cause of which was unknown, and after which she never recovered her usual health and strength. (3) The period of gradual onset of weakness in the legs and numbness in the feet, which progressed in the course of twelve months to complete paraplegia without any sphincter disturbance or any mental change. (4) The last period of life, extending over the last few days of life, in which there was the most marked change in her mental state—a condition of noisy delirium with obsessions and delusions being followed shortly by coma and death. There was also during this period a loss of sphincter control, and a change in type of the paraplegia from "paraplegia in extension" to "paraplegia in flexion," and the beginning of trophic changes—for I take it that the blood in the urine and the haemorrhagic condition of the bladder mucous membrane are to be regarded as evidence of a trophic change in the bladder—with sudden rise of temperature and death.

As has been said, the diagnosis made during life was disseminated sclerosis of the spinal type, and it was suggested that the cerebral symptoms manifested during the last few days of life were due to an extension of the same morbid process to the brain. A careful macroscopic examination of all parts of the brain failed to reveal any lesion either of a meningitic or parenchymatous character, and we can only conclude that these symptoms must have been due to some acute toxic process.

There was absolutely no history of any ataxy during any period of the disease; from the first and continuously onwards the only symptom was weakness and stiffness of the joints. Nor was there, on examination of the patient, any obvious disturbance of sensation or of the sphincters. In fact, clinically the case was one of pure "paraplegia in extension," which only changed in type during the last two or three days of life.

In the light of these facts it is difficult to correlate the clinical symptoms with the *post-mortem* findings. One would have expected to have had more definite symptoms due to the involvement of the posterior columns, but the more acute onset of the sphincter disturbance and the trophic (?) changes in the bladder, the relaxation of the spasticity of the extensor muscles, and the acute mental disturbance were probably due to the acute changes in the cord, that is, the haemorrhages and the more recent degeneration of the myelin which were most obvious in those parts of the cord which were the site of the heterotopia. And although the heterotopia must have been of congenital or developmental origin and apparently caused no symptoms until the onset of the signs of systemic degeneration, it is difficult to avoid the conclusion that in some way or other it was responsible—as a *locus resistencie minoris*—for the more acute histological changes in the cord which manifested themselves in the change of

symptoms during the last period of life, and probably accelerated the patient's death.

REFERENCES.

¹ Reports from the Pathological Laboratory of the Lunacy Department, New South Wales Government, vol. cxi, p. 54, "Heterotopia of the Spinal Cord." ² A Clinical and Anatomical Report of a Case of Friedrich's Disease, *Medical Journal of Australia*, February 17th, 1917. ³ Orr and Rows: Observations on the Influence of Toxins, *Journ. of Ment. Sci.*, April, 1914. ⁴ Williamson: *Diseases of the Spinal Cord*, 1910.

DIAPHRAGMATIC HERNIA OF THE ENTIRE STOMACH AND GREAT OMENTUM.

BY

J. GRANT ANDREW, M.B., C.M., F.R.F.P.S.G.,

VISITING SURGEON TO THE VICTORIA INFIRMARY, GLASGOW; FORMERLY
LIEUT.-COLONEL (TEMPORARY) R.A.M.C.(T.).

[With Special Plats.]

As this case is unique in respect that x-ray photographs are shown of the entire stomach in the left pleural cavity and after operation in the normal situation, it was thought worthy of publication. The history of the case is also interesting, showing the amount of unnecessary hardship inflicted upon a long-suffering and patient soldier by a failure to recognize the great diagnostic value of x-ray examination.

M. B., a rifleman, aged 22, was shot by a sniper about 7 p.m. on March 12th, 1917, while occupying a captured German trench. The bullet entered the left side of the chest behind and immediately below the twelfth rib, $2\frac{1}{2}$ in. from the middle line, and was extracted from under the skin on the following day at a point 2 in. below and 1 in. to the inner side of the left nipple line, a through-and-through wound. The bullet, as frequently happens, had turned round and was pointing base uppermost. No anaesthetic was given for its removal. Milk alone was given by the mouth at the casualty clearing station and was retained. From the casualty clearing station he was removed to a base hospital in Rouen, arriving in England on April 17th, 1917. Several months were passed in a general hospital and convalescent home before he was considered fit to return to his regimental dépôt, which he did about the middle of July. He complained at his dépôt that he suffered much pain after food of any description and that he was frequently sick. His statements were disregarded and he was given "medicine and duty." In spite of his complaints he was sent back to France on July 27th, that is, four months after receiving his wound. Every day while in France he suffered pain, and was totally unable to digest the ordinary army ration. He was told that he was "swinging the lead," and received little or no sympathy. He stuck it until he could stand it no longer, and he was carried down from the line too weak to go as a sitting case. He returned from France on December 28th, 1917, sent home as a case of gastritis. That label has been his undoing. He arrived at an auxiliary hospital in Scotland on December 31st, 1917. For seven or eight weeks he received treatment for gastritis, after which he spent thirteen weeks in a convalescent home, but without any improvement. A visiting medical officer to the convalescent home marked him as fit for discharge from the service, and for this purpose he was sent back to the hospital to which he had been sent on his return from France. Instead of giving him his discharge a medical board at this hospital gave him ten days' leave and marked him "duty." On the second day of his leave he reported sick, and was immediately admitted to the medical wards of the "mother hospital." Here also for weeks he was treated for gastritis, and was given medicine and milk diet. He was ultimately, on July 25th, 1918, fifteen months after being wounded, discharged from the service as a case of gastritis.

Not being one whit better, he consulted the local Pensions Committee; they sent him to a hospital for pensioners, and there he received the same line of treatment on the medical side of the house. After six weeks' residence in this hospital he was, to use his own term, "fed up," and he intentionally overstayed his leave, and was in consequence dismissed from the hospital.

On again consulting the local Pensions Committee he passed into my care on February 19th, 1919, for the first

time under the charge of a surgeon; and that was by inadvertence. On admission it was noted that his general condition was unsatisfactory. Examination revealed the existence of the scars in the left lower chest wall as before noted. The heart was slightly displaced towards the right, and was irregular in rhythm. The base of the left lung was dull to percussion, and the respiratory murmur was absent in the lower half of this lung, while râles were heard in the upper half in the greater part of the right lung.

His constant complaint, however, was that of pain immediately after or very shortly after food of any description, and a feeling of great distension. From the situation of the through-and-through wound, my opinion at this stage was that the condition might be one of hour-glass contraction of the stomach following perforation by the bullet.

The case was clearly one for examination by bismuth, and he was sent for a radiographic report. The entire stomach, as shown in the accompanying photographs taken by Major James R. Riddell, appeared to be above the diaphragm and in the left pleural cavity. Though his general condition was not good, if relief was to be obtained at all operation was an imperative necessity.

On March 25th, 1919, assisted by Captain Rimer, U.S., A.M.S., and by Captain James Maclean, R.A.M.C.(T.), as anaesthetist, the abdomen was opened by a transverse incision through the left rectus muscle. The transverse colon immediately presented itself. No stomach was visible. An additional vertical incision was necessary to get under the diaphragm. It was then seen by tracing up the bowel from the duodenum that the entire stomach and great omentum, as indicated in the x-ray picture, had disappeared and were on the other side of the diaphragm, the opening in the diaphragm being in the neighbourhood of and indistinguishable from that through which the oesophagus enters the abdomen. With considerable difficulty the stomach was drawn back into the abdomen. The omentum had contracted adhesion to everything it came into contact with, and probably to the under surface of the lung. Any attempt at traction caused haemorrhage, and a portion of the omentum so adherent that it was dangerous to remove was left in the pleural cavity. The opening in the diaphragm was closed by strong interrupted silk sutures. The stomach appeared thickened as if the wall had hypertrophied in the vain effort to pass the food on to the duodenum; but what was still more striking was the absence so far as one could see of any perforation or track of perforation by the bullet.

His recovery was naturally slow, but was not complicated in any way. The wounds healed primarily. When last seen by me, on May 20th, he stated that he was now completely free from sickness and from pain when food was taken. He was taking ordinary diet. The heart was normal in position and in rhythm. There is still some comparative dullness at the base of the left lung with diminished respiratory murmur, vocal fremitus, and vocal resonance.

Since the commencement of the war in 1914 several cases of a similar nature have been put on record, but no case, so far as I have been able to find out, has been published showing by x-ray the herniated stomach. In the BRITISH MEDICAL JOURNAL of April 19th, 1919, Sir James Barr, in writing on chest conditions, referred to a case of diaphragmatic hernia of the stomach successfully operated on by Captain Arthur J. Evans. Captain Evans has not yet published his case, but he kindly sent me a copy of the medical case sheet. The similarity of his case to mine is very striking, both in regard to history and the condition found on operation. Almost a year elapsed—eleven months—in Captain Evans's case before operation. Captain Evans writes: "The most interesting point about the case is how the patient ever managed to survive the most extraordinary trials he must have gone through before it was recognized that he was a serious case." Two years elapsed in my case, and the record, to say the least, is not one to be proud of. In both cases the entire stomach and omentum had passed into the left pleural cavity, and in both the difficulty lay in returning the omentum. The following articles may be referred to by those interested in the subject: "Notes on a case of traumatic hernia of the diaphragm proving fatal seven months after wound," by Captain C. P. Symonds, R.A.M.C., *Journal of the R.A.M.C.*, September, 1917; "La hernie

PLATE I.

FIG. 1.—Near "a" in Plate II; cervical.

FIG. 2.—Near "c"; mid-dorsal.

FIG. 3.—Near "e"; lower dorsal.

FIG. 4.—Near "f" or "g"; lumbar.

FIG. 5.—Near "b" or "e."

FIG. 6.—Near "b" and "e"; between them.

PLATE II.

FIG. 1.—Drawing of cord, natural size, showing levels examined and position of abnormalities. a, Middle cervical. b, Lower cervical. c, Upper middle dorsal. d, Area apparently injured. e, Lower dorsal with heterotopia. f and g, Upper and lower lumbar.

FIG. 3.—Upper cervical.

FIG. 4.—At "e," lower dorsal.

FIG. 5.—Lumbar.

FIG. 2.—Between "b" and "e," Fig. 1.

FIG. 6.—Near "b."

FIG. 10.—Middle upper dorsal, at "c."

FIG. 7.—Near "b" or "e."

FIGS. 8 and 9.—Near "e."

FIGS. 11 and 12.—Busch-Marchi preparations of the lumbar and cervical levels.