- Guns, P., and Coene, R. (1929). *Ann. Malad. Oreille*, **48**, 414.
 Hartmann, E. (1933). *Bull. Soc. ophtal. Paris*, p. 414.
 Heitz, J. (1929). *Arch. Mal. Cœur*, **22**, 383.
 Heritage, K. (1933). *Lancet*, **2**, 1258.
 Hunt, R., and Taveau, R. de M. (1906). *British Medical Journal*, **2**, 1788.
 Janet, H., Vallery-Radot, P., and Huguet, S. (1932). *Bull. Soc. Pédiat. Paris*, **30**, 644.
 Jones, M. S., and Todd, H. (1935). *Biochem. J.*, **29**, 2242.
 Kitakoji, Y. (1931). Quoted in *Ber. ges. Physiol.*, **63**, 58.
 Lanari, A. (1936). *C. R. Soc. Biol.*, Paris, **123**, 1090.
 — (1937). *Ibid.*, **125**, 546.
 Langley, J. N., and Anderson, H. K. (1904). *J. Physiol.*, **30**, 439; **31**, 365.
 Lemaire, A., and Bioy, E. (1934). *Progr. méd.*, Paris, p. 1871.
 Lipschütz, H., and Schilf, E. (1931). *Arch. exp. Path. Pharmacol.*, **162**, 617.
 Loeper, M., Lemaire, A., and Dany (1932). *Bull. Mém. Soc. méd. Hôp. Paris*, **48**, 943.
 Loewi, O. (1921). *Pflüg. Arch. ges. Physiol.*, **189**, 239.
 — (1922). *Ibid.*, **193**, 201.
 — (1933). *Harvey Lectures*, p. 218, New York.
 — and Navratil, E. (1924). *Pflüg. Arch. ges. Physiol.*, **206**, 123.
 — (1926). *Ibid.*, **214**, 678.
 Louyot, P. (1933). *Rev. Méd. Est*, **61**, 258.
 Lucas, C. C., Hall, G. E., and Ettinger, G. H. (1935). *J. Pharmacol.*, **54**, 151.
 McGeorge, M. (1937). *Lancet*, **1**, 69.
 McLaughlin, F. L. (1933). *British Medical Journal*, **1**, 997.
 Mann, P. J. G., Tennenbaum, M., and Quastel, J. H. (1938). *Biochem. J.*, **32**, 243.
 Matthes, K. (1930). *J. Physiol.*, **70**, 338.
 Merklen, P., Warter, J., and Kabaker, J. (1932). *C. R. Soc. Biol.*, Paris, **111**, 213.
 Murakami, K. (1931). Quoted in *Ber. ges. Physiol.*, **61**, 58.
 Orr, H. C. (1936). *British Medical Journal*, **2**, 69.
 — and Young, J. H. (1935). *Ibid.*, **1**, 1119.
 Pagniez, P., Plichet, A., and Decourt, P. (1932). *J. Méd. Paris*, **52**, 872.
 Plattner, F., and Hintner, H. (1930). *Pflüg. Arch. ges. Physiol.*, **225**, 19.
 Saad, K. (1935). *Quart. J. Pharmacy*, **8**, 31.
 Schilf, E. (1932). *Arch. exp. Path. Pharmacol.*, **166**, 22.
 Sciclounoff, F. (1934). *Presse méd.*, **42**, 1140.
 Stedman, Edgar, Stedman, Ellen, and Easson, L. H. (1932). *Biochem. J.*, **26**, 2056.
 Velhagen, K. (1932). *Arch. Augenheilk.*, **105**, 573.
 Verebely, T. von (1936). *Klin. Wschr.*, **15**, 11.
 Villard, H., Dejean, C., and Temple, J. (1932). *Arch. Ophthal.*, **49**, 72.
 Villaret, M., and Justin-Besançon, L. (1928). *Presse méd.*, **36**, 673.
 — Schiff-Wertheimer, and Justin-Besançon, L. (1928). *C. R. Soc. Biol.*, Paris, **98**, 909.
 Wolff, H. G. (1929). *Arch. Neurol. Psychiat.*, Chicago, **22**, 686.

In the Chemistry Section at the Cambridge Meeting of the British Association, August 17–28, the president, Professor C. S. Gibson, will review the recent advances that have been made, chiefly by himself and his co-workers, in the chemistry of gold. This opening address will be followed by a discussion on recent advances in the organic chemistry of the metals with special reference to the noble metals. A discussion on modern methods of chemical analysis, including physical and microchemical methods, will be opened by Dr. J. J. Fox, Chief Government Chemist, and Professor W. L. Bragg will open a discussion on "Clays." The fourth symposium, entitled "Repercussions of Synthetic Organic Chemistry on Biology and Medicine," which has an added interest in view of the exceptional circumstance that the Physiological Section will not meet this year, is being arranged by Professors E. C. Dodds and J. W. Cook. Recent work on the production of new compounds having the biological action of the sex hormones will be described by Professor Dodds and Professor L. Ruzicka (Zürich), and Dr. A. S. Parkes will deal with some of their interesting biological interrelationships. The second half of the programme will be occupied with descriptions of synthetic compounds which are able to induce cancer (Professor J. W. Cook), of new compounds having the physiological action of the life-maintenance hormone of the adrenal cortex (Professor T. Reichstein, Zürich), and of the synthesis of vitamin B₁ and analogous compounds (Dr. A. R. Todd). Specimens and apparatus relating to these discussions will be on exhibition throughout the meeting. By invitation of Sir William Pope a visit will be made to the University Chemical Laboratories, and Sir F. Gowland Hopkins has invited the section to visit the departments of biochemistry and biology, and the Molteno Institute.

SURGICAL TREATMENT OF DISSEMINATED SCLEROSIS BY SYMPATHECTOMY AND GANGLIONECTOMY

TECHNIQUE BY THE ANTERIOR APPROACH

BY

C. F. KOCH, M.D.,

(From the Surgical Division of the City Hospital of Middelburg)

AND

EUGENE DE SAVITSCH, A.B., M.D.,

C.R.B. Fellow and Associé Étranger à l'Institut Bunge, Antwerp

Nothing is more discouraging to the neurologist than the treatment of disseminated sclerosis. The task is made difficult on account of ignorance of the true aetiology of the disease. The correct evaluation of any therapeutic measure is complicated by the irregularity of the symptoms of the disease, with its frequent spontaneous remissions and uncertainty of objective findings. Among the various theories of the aetiology of disseminated sclerosis the vascular hypothesis appears to be the most logical. Putnam (1933, 1931–6, 1937), in his admirable studies, has shown that the histological characteristics of the disease may be closely imitated experimentally by a number of different procedures, all of which have in common an interference with the blood supply of the affected area. These facts, according to this author, give a new importance to the vascular abnormalities which have long been recognized as a characteristic of sclerotic plaques. Putnam further states that if the specific lesion is produced by a simple mechanical interference with the blood supply it is superfluous to postulate a specific demyelinating organism, virus, or toxin to account for such lesions, and that the ultimate aetiological factor should probably be sought in a local vascular abnormality.

The Vascular Hypothesis

Our procedure is based on the assumption that the hypothesis of Putnam is probably correct. In this we are in agreement with Wetherell (1934, 1935) and Royle (1933), who were able to show the practical application of this theory. We consider that the pathological changes in disseminated sclerosis are attributable to an ischaemic phenomenon resulting possibly from a spasm of the blood vessel supplying the parenchyma of the brain and spinal cord. Our aim, therefore, is to institute a procedure which would tend to lessen the alleged vascular contraction, thus allowing a better blood supply to the involved areas. There is but little doubt that this procedure can in no way remedy the complete damage to the nerve cells and their axons, but it is quite conceivable that the improved vascularization may re-establish some of the functions of partially damaged tissue. That this is possible is best demonstrated by the course of the disease itself. Frequent remissions and sometimes total disappearance of symptoms lead us to believe that in many cases the destruction of the nerve cells is in no sense final, and that when they are placed in more advantageous conditions their function may be regained.

The theoretical aspects of this procedure may be summarized as follows. Vascularization of the brain and the spinal cord depends entirely on the carotids and vertebral arteries. These are innervated by the sympathetic nervous

system through the stellate ganglion and the vertebral nerve. Fig. 1 shows in a schematic way the structural relations concerned. As may be seen, should a vascular spasm be present, it would find its responsible nervous factor in either the stellate ganglion or the vertebral nerve, or in both.

FIG. 1.—Stellate ganglion and vertebral nerve in their relation to the carotid and vertebral arteries (schematic figure). 1, Hypothalamus with vasomotor centre. 2, Intramedullary vasomotor tract. 3, Radix medulla anterior. 4, Ramus communicans albus. 5, Stellate ganglion. 6, Ganglion intermedium. 7, Ganglion cervicale med. 8, Superior cervical ganglion. 9, Carotid plexus. 10, Vertebral nerve. 11, Brachial plexus. 12, Ramus communicans griseus. 13, Nervi cardiaci symp. 14, Subclavian artery. 15, Vertebral artery.

Operative Procedure

The extirpation of the upper thoracic and lumbar sympathetic ganglion and trunks by the dorsal approach was carried out successfully by Adson and Brown (1929) in the treatment of Raynaud's disease. The sympathetic gangliectomy by the anterior approach by the same authors failed to give the desired results. On the other hand German (Rieder, 1930) and French (Leriche, see Cadenat, 1933) investigators successfully used the latero-anterior route. The dorsal approach necessitates extensive dissection as well as actual costo-transversectomy, which is likely to be accompanied by a considerable shock to the patient, as in most of the bone operations. The approach of Leriche and Rieder, though definitely less traumatizing, has the disadvantage of limiting the visibility of the operative field. The technique which we propose is an attempt to eliminate the drawbacks of the earlier trials. Our experience is based on some twenty-six operations, most of them in cases of advanced disseminated sclerosis.

PRE-OPERATIVE ROUTINE

The pre-operative preparation of the patient is essentially the same as in any other surgical procedure. A soap-and-water enema and a mild sedative of the barbiturate group on the eve of the operation ensure a restful night to the patient. For pre-operative medication a dilaudin-atropine combination gives

excellent results. We feel that avertin by rectum in doses of 70 to 105 mg. per kilo of body weight, depending on the condition and age of the patient, is the anaesthetic of choice. It has the advantage of permitting the anaesthesia to be induced while the patient is still in his room, and its lasting effect helps greatly in alleviating post-operative apprehension. Avertin alone is practically never sufficient. It has to be supplemented by local infiltration of 1/2 per cent. of novocain solution with 16 drops of 1 in 1,000 solution of epinephrine per 100 c.cm. of novocain. In certain cases a few whiffs of ether have to be given in the terminal stages. It is well to have on hand a positive-pressure anaesthesia apparatus, as occasionally the pleura may be punctured during the course of the operation.

An ordinary thyroidectomy set may suffice, but a few specially devised instruments, as illustrated in Fig. 2, are of

FIG. 2.—Special instruments. A, Depressor. B, Retractor. C, Aneurysm needle.

considerable help. The depressor A has the advantage of a long handle and curved spoon-like spatula which allow the retraction of the moving dome of the pleura during deeper dissection. For retraction of larger vessels the retractor B ensures a considerable amount of visibility with comparatively great safety. A small aneurysm needle (C) for ligation of smaller vessels is optional.

Careful haemostasis and exact knowledge of the anatomical relations of this region are indispensable in this procedure. Even the slightest haemorrhage obscures the operative field, causing a very prolonged and distressing delay. In cases of doubt as to the nature of the structure it is always best to ligate it, this precaution saving a great deal of time in the long run. The anatomy of the region, though fairly uniform, is none the less subject to numerous modifications. The variable calibre and the relative positions of the blood vessels are particularly annoying. The vertebral nerve is a great offender. Its position in reference to the vertebral vessels classically (Hovelacque *et al.*, 1937) is said to be posterior to the vertebral arteries. In some of our cases the relationship was just the reverse. We emphasize this point in order to place the operator on guard against the unexpected possibility.

SOME DETAILS OF TECHNIQUE

After induction of the avertin anaesthesia the patient is placed in a semi-reclining position with the neck hyperextended and the head turned away from the side on which the operation is to be performed. It is essential to see that the patient is so securely fixed as not to permit any change of position during the operation. After the infiltration of the skin with local anaesthetic a semilunar incision is made just above the clavicle extending from the border of the trapezius to approximately one centimetre beyond the midline. (In the first attempt (Koch, 1937) a vertical incision was made parallel to the sternocleidomastoid muscle; it proved to be somewhat unsatisfactory as it permitted but limited exposure of the caudal end of the sympathetic trunk.) The skin and adjoining platysma are separated from adjacent tissues and the cut edges are fastened with silk sutures so as to allow the maximum of gaping. The next step is a division of the clavicular portion of the sternocleidomastoid muscle. Its cranial portion is lifted and sutured to the already fastened upper edge of the skin. This procedure brings into view the

omohyoid muscle, the fascia colli media, fat, and possibly some lymph nodes. The omohyoid muscle is transfixed by two sutures and divided, the sutures preventing either portion from getting lost. The removal or pushing away of the fascia colli media and adjoining fat and the division of the omohyoid muscle expose the scalenus anterior muscle with the phrenic nerve, the inferior thyroid artery, the ascending cervical artery, and the transverse cervical artery. After gentle blunt dissection the thyreo-cervical trunk is brought into view. In some instances, in order to get into the deeper structure, it is not necessary to ligate the entire trunk; the ligation of the inferior thyroid artery suffices. The decision depends on the ease with which it is possible to retract caudally the subclavian artery. Without retracting the subclavian artery it is difficult to reach the vertebral artery with the accompanying sympathetic plexus.

Should it be necessary to ligate and cut the thyreo-cervical trunk it is best accomplished by leaving a fairly large stump so as not to permit the slipping of the ligature. It is felt that it is always safer to put a second ligature just below the first.

The next step is to proceed with the division and ligation of the vertebral vein crossing obliquely from the medial aspect of the scalenus anterior into the pre-vertebral tissues. The ligation of the vertebral vein clearly brings into view the anterior aspect of the vertebral artery and medially to it the ganglion intermedium. This relation is not constant; occasionally the ganglion may be found laterally or anterior to the artery. A nerve twig from the sympathetic trunk to the stellate ganglion, the Ansa Vieusseni, is seen going over the subclavian artery. It may be easily identified by applying traction, which moves the subclavian artery. Using the Ansa Vieusseni as the lead, the sympathetic chain is reached. Once the relationship is established the vertebral artery is stripped of its sympathetic plexus over an area of about one to two centimetres. The dissection is rendered fairly simple by use of a small sharp scalpel. When stripped the artery is pulled up and the underlying tissue examined. After lateral retraction of the scalenus anterior muscle the prevertebral fascia is divided. The stellate ganglion with its numerous branching fibres and occasional veins is then in plain sight. Careless cutting of a vessel in this region results in a brisk and obscuring bleeding. In carrying out the dissection the surgeon's action may be aided greatly by the introduction of the small spoon-like depressor A. The movement of the pleural dome on which is superimposed the costo-cervical artery not only interferes with the visualization of the field but also may lead to an accidental perforation of the parietal pleura. It is self-evident that the dome must be detached from its costal and cervical attachments; this is best accomplished with the aid of a sharp thin scalpel. The costo-cervical artery should always be divided and ligated. The extirpation of the stellate ganglion and of the vertebral nerve is accomplished in one step. The ganglion is grasped firmly by the Thiersch forceps and pulled anteriorly. The rami and the attachments are thus brought into light and may be cut from their upper end with sharp scissors. Once freed from the upper and posterior attach-

ments more forward traction is applied to the ganglion so as to permit the introduction of a finger between the sympathetic trunk and the posterior wall. With the finger as a guide the trunk is detached as far as possible and cut below the first costo-vertebral junction, which is roughly midway between the stellate and second thoracic ganglia. If the operation is done with care, the field is dry and therefore closure may be accomplished without any drainage. If in doubt, a drain may be placed in the dependent portion of the wound. After the repair of the separated muscles the wound is closed in layers in the usual fashion. The patient is kept in bed for a period of about one week, and the other side may be operated on in approximately two weeks.

Complications and Sequels

In the series of patients operated on by this method two fatalities have been observed. One was due to embolism, and the other patient died several hours after the operation of a brisk haemorrhage due to the slipping of the ligature from the thyreo-cervical trunk.

The anticipated Horner's syndrome was observed in every case, though enophthalmos had a tendency to regress,

becoming quite inconspicuous within a few months. The physiological complication to be expected is the inability of the heart to respond with acceleration to any muscular effort (the cardiac nerve is severed from its attachment when the stellate ganglion is removed). For about two months following the operation the patient must avoid any extreme exertion, as it may be accompanied by weakness and even collapse. Shortly after the operation some patients complain of pains in the shoulder and arm on

FIG. 3.—Cross-section of the neck showing the anterior approach to the stellate ganglion. 1. Sternocleidomastoid muscle (sutured end). 2. Sternocleidomastoid muscle (inferior portion). 3. Phrenic nerve. 4. Omohyoid muscle. 5. Clavicle. 6. Scalenus anterior muscle. 7. Subclavian artery. 8. Lung. 9. Vagus, carotid artery, and internal jugular vein. 10. Stellate ganglion.

the side of the operation. The pain lasts but a few days, and usually requires no attention. In certain persistent cases it was found useful to inject a small amount of novocain into the brachial plexus right above the clavicle; this gave immediate and permanent relief. Another complication seen in about one-third of the cases is incontinence. Fortunately it is not lasting, and in our experience always disappeared after the first forty-eight hours. Occasionally there is a paresis of the upper extremity of the operated side lasting but a few days and leaving no residual effects. Two patients perspired copiously in the region of the abdomen, lower back, and lower extremities. So intense was the perspiration that it necessitated special nursing care. This lasted for a period of almost six weeks, and responded but poorly to atropine.

Results

The operation was performed on fifteen cases of disseminated sclerosis, of which ten may be considered for statistical study, six to fourteen months having elapsed

since the operation. Eight of the ten patients are alive to-day.

Practically every case, including the four most recent ones, responded favourably to this surgical procedure, which is in agreement with the findings of Wetherell. At no time did we notice any immediate unfavourable effects in the development of symptoms. We attach considerable importance to this, since trauma in cases of disseminated sclerosis is believed to cause aggravation of symptoms.

It would be premature to draw final conclusions regarding the ultimate possibilities of this treatment. None the less we feel encouraged because in the majority of cases a remarkable degree of amelioration has taken place. It is not possible to attribute all the improvement to spontaneous changes in the course of the disease. All of the patients were in an advanced stage of the disease. We are attempting now to operate on patients in earlier stages, when, of course, the evaluation of results will be considerably more difficult and not easily assessed.

This report attempts to deal primarily with the technique of the operation, and so no detailed account will be given of every case. We feel that it is desirable to present the history of one of the patients in detail in order to give an idea of the type of case we have treated. Since the procedure is in the experimental stage we have so far applied it only to cases which respond to no other method of treatment. The case below is typical of this group.

Case History

A man 39 years old presented himself for an operation at the Middelburg Hospital in June, 1936, with the following story.

After an accident in 1926, when his right thigh was run over by a carriage, he developed a paraesthesia of the injured side. The Wassermann and Kahn reactions were negative, but there was a slightly positive Pandy test. Shortly after the first examination the patient developed some difficulty in walking. There was a slight tremor of the lower right extremity, but there was neither nystagmus nor intention tremor. The speech was normal. The patient was given an arsenic and quinine preparation and asked to report for examination later.

An examination at the beginning of 1927 disclosed general aggravation of the symptoms. Paraesthesia became more pronounced and involved both of the lower extremities. Three years later, in addition to the original complaints, he developed pains in the right side, and there was a marked decrease in

the muscular power of the right leg. The patient began to have difficulties with his vision, and was frequently nauseated. Some six months later Romberg's sign became definitely positive, rotary bilateral nystagmus was noted, and there was a bilateral Babinski reflex. The patient was unable to move, and most of his time had to be spent in bed. Examination in the middle of 1930 showed that the right Babinski reflex had disappeared, but the nystagmus persisted. The patient developed spontaneous jerking of the lower extremities, and there was pallor of both retinae. The patient was no longer able to read. There was a marked ataxia of the lower extremities, and some retention of urine. In 1934, in addition to the aggravation of the symptoms present, pains appeared in the lower limbs. The right leg could no longer support him.

In 1936, examination on the eve of operation revealed the following salient features: diplopia, marked bilateral nystagmus, temporal pallor of both optic disks, inability to read, scanning speech, bilateral intention tremor of the upper extremities, abolition of abdominal reflexes; most of the time the patient had to depend on a catheter for the passage of urine, both knee-jerks were absent, both plantar reflexes were extensor, both lower extremities showed occasional spon-

TABLE A

Case No.	Age of the Patient in Years	Duration in Years of the Disease from the First Observed Symptom	Number of Remissions	Duration of the Final Stage Before Operation
1	39	10	3	± 4 years
2	46	16	No remissions	± 2 "
3	38	5	4	± 1 year
4	45	17	No remissions	± 2 years
5	41	13	"	± 2 "
6	38	15	5	± 1 year
7	40	15	3	± 1 "
8	31	7	1	± 1½ years
9	31	1	No remissions	9 months
10	25	1½	1	8 "
11	32	15	No remissions	2½ years
12	42	17	1	4 "
13	32	5	?	1 year
14	46	9	No remissions	6 months
15	28	8	"	3 years

TABLE B

Signs and Symptoms	Case No. 3		Case No. 4		Case No. 6		Case No. 7		Case No. 8		Case No. 9		Case No. 11		Case No. 13		Case No. 14		Case No. 15	
	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B
1. Fatigue and Weakness ..	xxxx	x	xxxx	x	xxx	xx	xx	xx	xxx	x	xxx	o	xx	o	xx	x	xx	o	x	x
2. Stiffness of Extremities ..	xx	o	xxxx	x	xxx	xx	xxx	xx	xx	x	x	o	x	/	xx	x	xx	x	xxx	xx
3. Ataxia of Extremities ..	xx	x	xxx	xx	xx	xx	xxxx	xxx	xx	x	xxx	xx	x	xx	xx	xxx	xxx	xxx	xxx	xxx
4. Eyes: Nystagmus ..	o	o	o	o	o	o	x	x	xx	xxx	x	o	x	x	x	o	o	o	x	x
5. Diplopia ..	o	o	o	o	o	o	x	o	/	/	o	o	x	x	o	o	x	o	o	o
6. Scanning Speech ..	o	o	o	o	o	o	xx	x	xxxx	x	x	o	o	/	xx	o	o	o	o	o
7. Pain ..	o	o	xx	o	x	o	xxxx	o	x	x	x	o	o	o	o	o	x	x	o	o
8. Intention Tremor ..	o	o	xx	o	xxx	x	xxxx	xx	xxxx	x	xxxx	x	x	x	x	x	o	o	xx	xx
9. Clonus ..	xx	o	xx	o	o	o	/	/	x	x	x	x	x	x	x	x	o	o	x	o
10. Bladder Disturbance (R=Retention; I=Incontinent) ..	R.x	o	o	o	x	x	I.xxx	I.x	I.xxxx	I.x	R.x	o	o	o	R.xx	o	x	o	I.x	I.x
Time (in months) elapsed since operation ..	14		13		13		12		10		10		10		5		3		1	

Explanatory Note: Intensity of symptoms are recorded as follows: x=minimum; xxxx=maximum; /=doubtful; o=absence; A=Before operation; B=After operation.

taneous jerks, and there was a marked ankle clonus. The patient could not walk independently, and was able to move about only with the support of two sticks; at best, the furthest he could progress by himself did not exceed ten metres, and even then he occasionally had to be supported. When moving, the body was bent forward with the two arms stretched forward clutching the two walking sticks in front.

An operation was performed on the left side on June 25, 1936. On the first day after the operation he was unable to move his left leg, and incontinence was absolute. On the second day the spontaneous jerks in the left leg disappeared, though they were still present in the right leg. On the third day the patient was able to move his left leg. There was no change in the other symptoms.

The sympathectomy and ganglionectomy were performed on the right side eleven days after the first operation. On the following day the patient was no longer incontinent. The spontaneous jerks of the right leg were only slightly perceptible. Three days later they disappeared altogether. Paraesthesia of the extremities was no longer present. Two weeks later the patient tried to walk supported by a stick and a nurse. Twenty-four hours after the first attempt he was able to move about with a walking stick. For the first time in several months he was able to lift his legs independently. In about three weeks after the second operation the patient was able to walk without the help of the stick or nurse. The walking, though fairly strenuous, was successful, although the gait was shuffling. Romberg's sign was negative. There was no ataxia. On the twenty-third day the patient was discharged from the hospital. He returned for examination a month later. He moved about independently with apparent ease, though he collapsed some six weeks after the operation following a walk of a few hundred metres. The examining physician stated that the pulse frequency was normal. The collapse necessitated a rest of a few hours, after which the patient felt normal. Three months later he was able to resume his former occupation as an assistant in a grocery shop. He was able to walk about, though he preferred to be supported by a walking stick.

Fourteen months later the patient is still able to walk with the aid of a stick. There is no diplopia. He is able to read. Romberg's sign is negative, and he has no pain.

Conclusion

The case of multiple sclerosis just described was one with a definite downward course. The only transient improvement was in 1930, when the patient temporarily lost his right Babinski reflex. The operation has not only arrested the progress of the symptoms but has also re-established some of the functions.

We are presenting all our cases in tabulated form. Table A gives age of the patient, duration of the disease from the first observed symptom, number of remissions, duration of the final stage before operation; Table B gives the outline of principal symptoms and their changes. Space will not permit detailed consideration of each case. We have confined ourselves to the most distressing symptoms and to those common to most of the cases. Neither will the record of reflexes be given, since the surgical procedure only exceptionally modified the original findings.

The evaluation of the results in Cases 1, 2, 5, 10, and 12 is not included. No. 1 has already been discussed in detail. No. 2, because of a very unsatisfactory physical condition, could be operated only on one side, and died of uraemia eight months subsequently. Patients Nos. 5 and 12 died, one of pulmonary embolism and the other of a haemorrhage of the thyreo-cervical trunk. Patient No. 10, though showing progressive improvement in neurological symptoms, died of intercurrent infection eight months later.

This paper deals primarily with the technique of ganglionectomy and sympathectomy by the anterior approach. Our experience with this treatment in advanced multiple sclerosis suggests the desirability of further trial of this therapeutic measure.

REFERENCES

- Adson, A. W., and Brown, G. E. (1929). *Surg. Gynec. Obstet.*, **48**, 577.
 Cadenat, F. M. (1933). *Presse méd.*, **41**, 1014.
 Delmas, J., and Laux, G. (1933). *Anatomie médico-chirurgicale du système nerveux végétatif*, Masson et Cie.
 Hovelacque, Monod, and Evrard (1937). *Le Thorax: Anatomie médico-chirurgicale*, Paris.
 Koch, C. F. (1936). *Cong. franç. Chir.*, Paris, **45**, 451.
 — (1937). *Nederl. Tijdschr. Geneesk.*, **81**, 425.
 Putnam, T. J. (1933). *New. Engl. J. Med.*, **209**, 786.
 — (1931-6). *Studies in Multiple Sclerosis, I-VII Arch. Neurol. Psychiat.*, Chicago.
 — (1937). *Ibid.*, **37**, 1298.
 Rieder, W. (1930). *Arch. klin. Chir.*, **158**, 355.
 Royle, N. D. (1933). *Med. J. Austral.*, **1**, 586.
 Wetherell, F. S. (1934). *J. Amer. med. Ass.*, **102**, 1754.
 — (1935). *Arch. Neurol. Psychiat.*, Chicago, **34**, 99.

THE TREATMENT OF PLACENTA PRAEVI

A REVIEW OF 286 CASES

BY

O. LLOYD, M.B., B.S.

AND

J. E. GIESEN, M.B., B.S.

Late Resident Medical Officers, Queen Charlotte's Maternity Hospital, London

During our respective terms of office at Queen Charlotte's Maternity Hospital we were much impressed by the results of a scheme in operation for the treatment of placenta praevia. The principle of this scheme was that all cases should be treated by a preconceived routine method. We found that its use greatly facilitated our work and, so far as we could judge, led to an improvement in the mortality rate of this grave condition.

A Standardized Method

Standardization is obviously a desirable feature in dealing with certain emergencies, as it affords an opportunity of assessing the value of the treatment, and those who have to carry this out are "forearmed" with a knowledge of the immediate steps to be taken for the relief of the disaster. It would seem of value to publish the results of a standardized treatment of a condition for which such a variety of methods are advocated. Since this scheme was put into operation 143 cases of placenta praevia have been treated over a period of approximately five years. These cases we shall refer to as Group A. For purposes of comparison we have taken an equal number of cases immediately preceding the start of the scheme. This series of 143 cases will be referred to as Group B. The principles governing the treatment of cases in Group A were laid down by Mr. Leonard Phillips, who assumed responsibility for all cases of placenta praevia. He has kindly allowed us to publish an account of his routine treatment. In order to clarify this account the following definitions are given:

1. A central placenta praevia was considered to be present if the internal os was completely covered by placental tissue