to whether the patient had ever before gone through an attack of the disease.

(4)—Cases who had suffered from Typhoid at a Previous Date.

No. of Case.	Date of Examination.	Sex.	Age.	Day of Disease.	Reaction.	Remarks.
126	16/3/97	—	—	6 months	None	
127	27/5/97	M.	21	1 year	"	
128	12/6/97	F.	16	14 months	Imperfect	Now suffering from tuberculous peritonitis with pyrexia.

(5)—Unclassed Cases.

129	27/2/97	M.	22	26th day	None	Apparently typhoid clinically, with one hæmorrhage, but temperature remained elevated, and death ensued, the <i>post-mortem</i> examination revealing general tuberculosis, although Peyer's patches were prominent and discoloured.
	30/3/97	M.	22	57th "	"	
130	26/5/97	M.	41	3rd week	"	A suspicious case of pyrexia in an alcoholic subject. During convalescence there was a relapse lasting a fortnight. Could not obtain a second sample of blood.
131	2/6/97	F.	45	3rd week	"	Was suffering from peritoneal trouble in May. Since then temperature has occasionally for a time risen two degrees above normal at night.
	12/6/97	F.	45	—	Very slight small clumps, but good deal of movement	
	9/7/97	F.	45	—	Ditto	
132	8/7/97	F.	71	—	Movement stopped in 10 minutes, but clumps prove imperfect	Has dorsal caries, with slight hectic temperature. Had a suspicious illness last summer, "inflammation" lasting a month.
	21/7/97	F.	71	—	Fair clumps in 10 minutes, but field not quite clear	

Of the non-typhoid cases one case of typhus fever (Case 120) gave a good reaction. We can only explain this by supposing that he had had typhoid previously, but this could not be satisfactorily ascertained. Another case of typhus fever tested on the same date gave absolutely no reaction, and this accords with the experience of other observers.

With a view to test Bates Block's statement that cases of malaria will react, we obtained serum from a well-marked case of this disease (Case 124), both during a paroxysm and in the interval, and at neither period was any reaction observed.

Of the total cases examined, 34 were obtained from the Belfast Royal Hospital, 37 from the Belfast Union Fever Hospital and Infirmary, 8 from the Ulster Hospital for Women and Children, 8 from the Queen Street Hospital for Sick Children, and 9 from the Garrison Hospital. The remaining 36 cases were sent in by private practitioners.

Our warmest thanks are due to Dr. J. Lorrain Smith for permission to work in his laboratory, and for many valuable suggestions while the work was in progress.

PERIPHERAL NEURITIS CONNECTED WITH PREGNANCY AND THE PUERPERAL STATE.

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In paralysis occurring during pregnancy or the puerperium the seat of the lesion may be either in the brain, as from hæmorrhage or thrombosis; in the spinal cord, as from some form of myelitis; or in the nerve trunks. Of paralyzes due

to lesion in the nerve trunks it is easy to distinguish two widely different groups of cases—namely (1) those due to injury or disease of the nerve trunks, as they pass through the pelvis; and (2) those due to neuritis occurring at the periphery of the nerves, and in which the nerves affected do not necessarily pass through the pelvic cavity.

Of the paralyzes due to affection of the nerve trunks as they pass through the pelvis I intend to say but little. They are more commonly due to injury of the nerves by forceps when the symptoms are generally unilateral, setting in with severe sudden pain at the time of injury (sometimes followed by anæsthesia of the part supplied by the injured nerve) and followed by paralysis of the extensors of the foot and tensor vaginae femoris supplied by the external popliteal and superior gluteal nerves, the pressure having been applied to the lumbosacral cord and the roots of the sacral nerves. Cases of this nature have been recorded by Bury,¹ Thomas,² Bernhardt,³ Mills,⁴ and many others. More rarely paralysis has resulted from pressure of the head of the child on the pelvic roots during prolonged and difficult labour, as recorded by Vinay,⁵ Ramskill,⁶ and Lloyd Roberts.⁷ Other cases of paralysis of the legs from a lesion of the large nerve trunks in the pelvis have had a very different cause, as in a case described by Leyden,⁸ where paralysis came on in the lower extremities with neuralgic pains in the sciatic nerves after puerperal fever, and on *post-mortem* examination there was found phlegmonous infiltration of the cellular tissue of the pelvis, implicating the sheaths of both sciatic nerves. Adams⁹ reports a case where a pelvic abscess coming on after confinement caused a unilateral paralysis.

I have seen a case in which a severe double sciatica led to the discovery of a fibroid tumour of the uterus impacted in the brim of the pelvis. An operation was performed and the tumour was lifted up and fixed to the anterior abdominal wall. There was no septic mischief, and the operation was entirely successful, but the pains in the legs still continued and were followed by double atrophic paralysis of the anterior tibial and peroneal groups of muscles, and afterwards of the muscles of the calf, with marked œdema in both legs. These symptoms progressed in spite of the fact that the fibroid growth not only remained in the position in which it had been placed, but also, from the menopause setting in, diminished until the uterus was practically of the normal size and the whole pelvic cavity was apparently normal, as ascertained by careful bimanual examinations made by most competent gynecologists. It would seem as if the symptoms had been produced by a chronic inflammation of the nerve sheaths in the pelvis set up by the pressure, just as may possibly occur very rarely by pressure of the head of a foetus.

I wish to deal, however, more particularly with those cases in which peripheral neuritis comes on, either during pregnancy or the puerperium, and in which many nerves are affected (not necessarily only those which pass through the pelvic cavity), and in which there is evidence of true peripheral neuritis, similar to that found after taking alcoholic and other poisons. These cases, judging from the few reported, do not seem to be very common, but this may be because they are not sufficiently recognised.

The two following cases have occurred in my practice:

CASE I. Vomiting of Pregnancy, Abortion Procured; Peripheral Neuritis following and affecting both Legs; Gradual Recovery.—Mrs. D., aged 24 years, was married in April, 1895. In November, 1895, she was about four months pregnant, and was suffering from such severe and uncontrollable vomiting that abortion had to be procured. For two or three weeks after this she had complete loss of power over the bladder and rectum, but these symptoms passed off in a fortnight, and never troubled her again. About a month after the abortion had been procured her feet felt very cold and numb; a month after this she found that her legs were very weak, and sensation in them was almost entirely abolished; there was no affection of the upper extremities. The loss of power became complete, she could not stand or walk, the legs were wasted, and finally the knees became contracted and fixed. This condition continued in spite of treatment, and when she first came to Manchester, in July, 1896, the legs were in the same flexed position, with marked talipes equinovarus of both feet, and extreme wasting and total paralysis of the lower extremities, and absence of the knee-jerks. There was some power of flexion of the hip-joints and she could move the great toes very slightly; there was some loss of sensation in the toes. The knee-joints were straightened five times under chloroform, and fixed in splints. Electricity and massage were employed, and the condition improved much for a time, and then became stationary.

I first saw the patient in consultation with Dr. Grosvenor, of Manchester, in October, 1896, when I found all the muscles of the legs, and to a less extent the muscles of the thighs, wasted and powerless, the feet

being in a state of talipes equino-varus, and the knee-jerks absent. There was total inability to stand or walk, both from the loss of power and the contracture of the knee-joints. There was no loss of sensation, and no tenderness on deep pressure of the muscles. Regular massage was advised. I saw the patient again on March 5th, 1897, when she was again pregnant, but had not suffered from vomiting. There was still loss of the knee-jerks, but the muscles of the thighs were fairly strong, and much greater in bulk than formerly; there was only a slight contracture at the knee-joints. The muscles of the legs were also increasing in volume, and gradually regaining their power; but the feet were still both "dropped" (talipes equino-varus), although the toes could now be moved slightly. The right foot could be flexed normally, but the left could only be flexed and adducted, the left peroneal muscles not having regained their full power. The patient could now walk, but with the feet dragging slightly unless well lifted from the ground at each step ("high-stepping gait"); the toes were put down first, followed in the right by the heel; but in the left foot the heel did not touch the ground, as it was held up by the contractures of the left calf muscles. In June Dr. Grosvenor informed me that the patient had had a normal confinement, and that the paralysis was so much better that the patient could take short walks outside. There was no previous history of alcoholism.

CASE 11. *Chronic Pyæmia following Labour: Atrophic Paralysis of both Legs: Gradual Recovery.*—Mrs. H. was quite well until her first child was born in 1892, when she was 18 years of age. The confinement took place in a Manchester hospital, and she told me that shortly afterwards she was very ill, and was operated on for a "liver abscess." She was then removed to the Manchester Royal Infirmary, and I am informed by one of the late resident surgical officers that she was found to be suffering from chronic pyæmia and paralysis of the legs. She was removed to the workhouse, and came under my care early in 1893 in the Manchester Workhouse Infirmary. She was then suffering from total atrophic paralysis of both lower extremities with contractures of the knees; there was absence of knee-jerk, but the arms and sensation were unaffected. She was treated by massage, and was discharged much improved in eighteen months. At the present time (June, 1897) she is practically well, and can do all her ordinary housework. She walks a little lamely because of a rigid contracture of all the joints of the toes into the soles of the feet, but all other muscles of the lower extremities are normal and well nourished. There was no previous history of alcoholism.

In the *Deutsche medicinische Wochenschrift*, 1895, Nos. 8 and 9, will be found an account by Eulenburg of puerperal peripheral neuritis, with a classified account of 38 cases; 8 of these, recorded by Mills, are, however, principally unilateral cases, due either to forceps or to exudations into the pelvis—sometimes associated with phlegmasia dolens—and these I here exclude from consideration, leaving 30 cases. In addition, I have collected 17 more cases of pure neuritis, and these, together with the 2 cases reported above, make up 49 cases, from which I have drawn the following conclusions:

ETIOLOGY.

I find that the disease is more common in multiparæ; in 12 cases the onset occurred during pregnancy, and as the symptoms are so similar to those in which the condition commenced in the puerperium, I think that all should be considered together. Of the cases commencing after delivery, in 16 the symptoms appeared within the first week, in 7 during the second week, in 6 during the third and fourth weeks, in 2 within the second month; in the 5 remaining cases the details are not available. In 15 cases out of the total of 49 there was a distinct history of some form of sepsis with shivering and fever; in 11 cases there was marked and incessant vomiting of pregnancy, so great in some instances that abortion had to be procured, and in 2 of these the symptoms of neuritis did not come on until about three weeks afterwards. In several cases the patients were very anæmic from loss of blood, and in a very few—especially where bulbar symptoms were present—it was difficult absolutely to exclude diphtheria. In 4 cases there was a distinct alcoholic history, and as in many instances there was marked tenderness on deep pressure of the muscles, the question arises as to whether more of the cases were not due to alcohol. One knows that during pregnancy and in the puerperium not a few women take alcohol, and though the doses may only be moderate, yet it must be remembered that comparatively small quantities of alcohol will sometimes induce neuritis, and especially if the subject is not in robust health. There can be no doubt, however, from a careful study of the symptomatology of puerperal neuritis that alcohol is not the cause in the infinite majority of cases; for alcoholic neuritis is practically never associated with paralysis of the cranial nerves. Again, if in alcoholic paralysis the affection of the legs is very severe, then the arms are also affected, and finally such symptoms as severe neuritis of the arms, and more especially of one arm without affection of the legs, is unknown to me in alcoholic paralysis. In many cases reported it is difficult to find any cause for the neuritis of pregnancy and the puerperium, but personally I look upon sepsis or on

incessant vomiting as the most potent factors in producing the condition. In septic poisoning it is admissible to assume that the toxins set up the neuritis just as occurs in diphtheria. As regards the incessant vomiting, two views may be taken. First, the view stated in a paper by Dr. Clifford Allbutt,¹⁰ and kindly forwarded by him to me, that the vomiting itself is due to a toxin, and probably that the neuritis is due to the same toxin. A second explanation which I merely suggest as a hypothesis may be that the incessant vomiting sets up a condition of acetonaemia, and that the acetone or some allied body in the blood produces the neuritis just as may possibly occur in the neuritis of diabetes.

SYMPTOMATOLOGY.

In the great majority of cases the disease begins with sensory disturbances, especially in those parts which are afterwards affected with paralysis of motion. These consist of numbness, tingling, hyperæsthesia, paræsthesia, or severe pains, often shooting in character. In not a few cases there was well-marked tenderness on deep pressure of the muscles, and great tenderness on pressure of the nerve trunks. Later on there may be anæsthesia of certain parts, but in cases of recovery this disappears before the restoration of motor power. The sensory symptoms are followed very soon by motor troubles, and in some cases the motor come on simultaneously with the sensory disturbances, and progress very rapidly over the whole body, running a course not unlike that seen in Landry's paralysis, and ending in speedy death from respiratory failure. Bulbar symptoms were present in 5 cases, and showed themselves either as difficulty of swallowing, alteration of voice or actual aphonia, pains in the fifth cranial nerve or paralysis of the third, sixth, seventh, or twelfth cranial nerves. In one case reported by Schanz there was, in addition to severe multiple neuritis, an atrophy of the optic nerves with blindness. In the 23 cases in which there were general signs of paralysis—that is, attacking both legs and arms—the loss of power was as a rule first noticed in the legs, and more or less rapidly extended to the arms, sometimes only one arm being affected, but more generally both. In many of these cases, also, the trunk muscles were affected with some involvement of the respiratory muscles and very rarely some affection of the bladder and rectum. In all cases the paralysis was of an atrophic character, often with well-marked reaction of degeneration.

In 15 cases the symptoms were confined to the arms, in 10 of these being limited to one side. In 7 the sensory and motor disturbances affected the median and ulnar nerves only, the paralysis being as usual of the atrophic type. In 1 case there was a complete diffuse atrophy of all the muscles of the right arm, in another atrophic paralysis of the upper arm and shoulder muscles on both sides, and in a third a unilateral paralysis of the same region; in 2 cases the ulnar nerve only was affected, and in 2 others the median only. Finally in 1 case there was generalised paralysis of both arms.

In 10 cases the muscles of the legs only were affected, in 8 of these the symptoms being on both sides. The muscles most generally involved were those of the anterior tibial and perineal groups, less commonly those with paralysis of the quadriceps extensor, and in 2 or 3 cases all the muscles of the legs. The position of the legs was almost exactly like that seen in alcoholic paralysis with talipes equino-varus and flexion of the toes with absent knee-jerks. As in other cases the paralysis was atrophic, and there were generally marked sensory disturbances.

In very many of the cases of generalised paralysis there was a marked oedema, especially of the legs, and this also occurred in most of the cases in which the legs only were paralysed.

PROGNOSIS.

In 14 cases there was either no recovery (death occurring fairly rapidly in a few cases) or only a partial recovery, some of the most severely affected muscles remaining permanently paralysed. In 22 cases recovery was complete, and in 13 cases I can find no mention of the termination. The prognosis as regards complete recovery is worst in the generalised forms, and is best in the partial cases, especially where the arms only were affected. It would seem that, just as in alcoholic paralysis, years may elapse before an absolute re-

covery takes place, so that treatment by massage, etc., must on no account be relaxed, but kept up for long periods.

PATHOLOGICAL ANATOMY.

In several cases the nerves have been examined after death, and well-marked evidence of neuritis has been found. Thus Korsakoff and Serbski,¹¹ in a case of septic origin, found degenerative neuritis in both sensory and mixed nerves, the changes being most marked at points furthest from the nerve centres; the ulnar, median, phrenic, and vagus nerves were much degenerated, and there was slight degeneration of the abducens. Lindemann,¹² in his case in which vomiting was the apparent cause, found well-marked general polyneuritis. In a case of Mader's,¹³ commencing with persistent vomiting of pregnancy, there was found extensive neuritis of the sciatic, tibial, and peroneal nerves, and pigmentary atrophy of the extensor muscles of the thigh; but by Marchi's method no changes were found in the spinal cord. Finally, in Solowieff's¹⁴ case associated with vomiting in pregnancy, there was neuritis of the phrenic, vagus, peroneal, and median nerves.

REFERENCES.

- ¹ *Peripheral Neuritis*, p. 357. ² *Pittsburg Med. Journ.*, 1880-81, vol. i, p. 41. ³ *Deut. med. Woch.*, 1894, p. 935. ⁴ *Univ. Med. Mag.*, Philadelphia, May, 1893. ⁵ *Rev. de Méd.*, 1887, vii, p. 596. ⁶ *Obstet. Journ. Great Brit.*, 1880, vol. viii, p. 678. ⁷ *Lancet*, 1880, vol. i, p. 54. ⁸ *Charité Annalen*, 1862. ⁹ *Lancet*, 1880, vol. ii, p. 699. ¹⁰ *Ibid.*, Feb. 27th, 1897. ¹¹ *Arch. für Psych.*, Bd. xxiii, Heft 1. ¹² *Centralbl. für allg. Pathol.*, 1892, August 20th. ¹³ *Neurolog. Centralbl.*, 1895, p. 871. ¹⁴ *Centralbl. für Gynäk.*, 1892, p. 492.

A CASE OF MALIGNANT TUMOUR OF THE BRAIN ORIGINATING IN THE MIDDLE EAR:

SYMPTOMS SIMULATING TEMPORO-SPHENOIDAL ABSCESS: OPENING OF MASTOID ANTRUM AND CRANIUM: PARTIAL REMOVAL OF TUMOUR: CESSATION OF RESPIRATION UNDER CHLOROFORM: TRACHEOTOMY: DEATH TWO AND A HALF MONTHS AFTER OPERATION.

BY

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HISTORY OF CASE PREVIOUS TO FINAL OPERATION, BY DR. BARR.

THE patient was a boy 12½ years of age. He was first brought to me on the 29th March, 16 months before his death, owing to severe pain in the right ear. This had existed intermittently for about three months. The pain occurred in such severe paroxysms that, in the words of the father, "the boy shook all over"; it was most intense at night, but might be absent two or three days at a time, returning without any special cause. On the supposition that it was of dental origin a tooth had been extracted without any effect. While the pain was his only spontaneous complaint, I was informed on inquiry that there had been a very slight discharge from the ear for three months. The hearing on that side was so defective that a watch, ordinarily heard 40 inches from the ear, was perceived at a distance of only half an inch; he had also a buzzing sensation in the affected ear. The tissues in the neighbourhood of the ear were quite healthy and the lymphatic glands of the neck were free from swelling.

Although subject to headaches and rather dull at his lessons, the boy had been considered quite healthy up to the occurrence of the pain; he had, however, prior to that, fallen on the ice, against which his head came with violence. It may be mentioned that his father's aunt was said to have died from a cancerous disease.

In the external auditory canal, but not protruding from it, there was a mass resembling a polypus; it differed from an ordinary aural polypus in its being exquisitely painful to the touch of a probe, which also caused more bleeding than usual. I removed the growth by means of a wire snare, under chloroform; its texture seemed to be peculiarly strong and tough, more like a fibrous polypus than the more common mucous variety, and the bleeding which followed was unusually severe. The growth undoubtedly sprang from the bony walls of the

tympanum; the tympanic membrane being almost entirely destroyed. The after-treatment consisted in the occasional application of chromic acid to the thickened tissue forming the root and the use of antiseptics, including diluted spirit. In the course of a few weeks he went back to school, and was soon after rather unfortunate in receiving a blow on the affected ear, which for the time being produced faintness and sickness.

Four months afterwards he was again brought to me with a similar growth, this time protruding from the external orifice of the ear. It was in like manner removed with the snare, cauterised, and treated antiseptically. Shortly after going home on this occasion he was again unfortunate—contracting scarlet fever, which laid him up for two months.

Finally, on 29th April of last year, he returned with a recurrence of the growth, which protruded as before from the external orifice of the ear. A more radical operation was now performed, and the growth was not only removed from the external auditory canal, but opening the mastoid antrum and the other cavities of the middle ear (which were found occupied by these growths) I cleared them out as thoroughly as possible with a gouge. During all those months severe pains in the ear and also in a less degree in the head occurred intermittently, and the sensitiveness of the interior of the ear was so great that nothing could be done without chloroform. The discharge from the ear had also continued.

Soon afterwards graver symptoms manifested themselves; these were persistent vomiting for several days, increased headache, intensified on the right side, followed (in a few days) by subnormal temperature and pulse, great drowsiness and stupor, ocular paralysis, and optic neuritis, clearly pointing, as was thought, to temporo-sphenoidal abscess. The eye symptoms seemed at this time, as observed by Dr. Ernest Thomson, to be very characteristic of abscess in that region. The following is Dr. Thomson's report:—

In the right eye there is complete ptosis. On elevating the drooped lid it is found that the eyeball is rotated outwards and very slightly downwards. The pupil is dilated *ad maximum*, and does not respond at all. The eye moves freely outwards, very slightly downwards, and not at all in any other direction. In the left there is no ptosis. Pupil active both directly and consensually. The eye moves freely in all directions, except outwards, in which direction the movement is very limited. Diplopia is not now admitted, although complained of previously. Ophthalmoscopic examination shows in the right optic neuritis, with swollen papilla, oedema of surrounding retina, and engorged and twisted veins. Left nerve slightly hyperæmic, with a little blurring of the edge, but not prominent to any extent.

Diagnosis of Eye Conditions.—Right optic neuritis of intense character, commencing left optic neuritis, paralysis of all the branches of the right third nerve. Paresis of right fourth nerve. Paresis, almost amounting to paralysis, of left sixth nerve. From the fact that the right pupil is dilated completely there is probably irritation of the sympathetic fibres to the iris.

It will be generally admitted that these symptoms, taken in connection with the existence of a purulent ear disease, pointed to temporo-sphenoidal abscess, and it was decided, after a consultation with Dr. James H. Nicoll, to trephine the skull in the usual situation of temporo-sphenoidal abscess. The operation was performed on May 19th, 1896, by Dr. Nicoll, assisted by myself, Dr. J. G. Connal, and Dr. Fred Adams, Dr. Connal administering the chloroform.

DESCRIPTION BY DR. J. H. NICOLL OF FINAL OPERATION AND OF SUBSEQUENT EVENTS.

On May 19th I saw the case with Dr. Barr, and we agreed in the opinion that the symptoms as just now detailed by Dr. Barr indicated a lesion in the temporo-sphenoidal lobe. As to the nature of that lesion we were also at one in thinking it probably an abscess, and not a tumour, and this for the following reasons: (a) The boy had, and had had for three months, the most common exciting cause of intracranial abscess in the form of a purulent otitis. (b) The rapidity and progressiveness of the development of the symptoms of intracranial lesion, a matter of five or six days in all. (c) Apart from these considerations the symptoms themselves afforded no sufficient evidence on which to decide between the two probabilities—abscess and tumour. That being so, it was natural to think it more probable that the lesion was the common one, abscess, and not a rarity in the shape of a malignant tumour causing both aural and intracranial symptoms.

Later in the day I operated. In the first place I still further enlarged the very free mastoid opening previously