sent to prison, and he thus described his dream. "I heard a voice at the door, and I said, 'Bless me, if that ain't John,' and I took him by the sleeve, it was his shirt-sleeve I felt, and I was half afraid of him, and surprised he was out weeks before his time. Then (in my dream) I dreamt that he tried to frighten me, and make believe he was a ghost by pushing me down sideways, &c., after that I waked and heard no more." This is a very curious account of the blind man's state of mind; he recognized his friend, but the latter behaved in so strange a way as to make the blind man believe he was a ghost. The pushing him down sideways, however, does not suggest a spiritual being to an ordinary mind. It would be a matter of great interest if Mr. Johns, or other persons coming in contact with the blind, would make further investigations into the subject of ghosts as conceived by the blind. The well-established ghost, clothed in white and quite impalpable, can scarcely have a place in the blind man's imagination.

SAMUEL WILKS, M.D.

Abstracts of Papers on Diseases of the Nervous System that have appeared in the Guy's Hospital Reports, 1874-1884.

1. Insular Sclerosis of the Brain and Spinal Cord. By W. MOXON, M.D. 1875.
2. On Meningeal Hæmorrhage. By J. F. GOODHART, M.D. 1876.
3. On Cerebritis, Hysteria, and Bulbar Paralysis. By S. WILKS, M.D., F.R.S. 1877.
4. On Unilateral Atrophy and Spasm. By FREDERICK TAYLOR, M.D. 1878.
5. Report on Cases of Tetanus treated in Guy's Hospital. By FREDERICK TAYLOR, M.D. 1878.
6. A Case of Disease of the Brain with Descending Degeneration of the Spinal Cord. By FREDERICK TAYLOR, M.D. 1879.
7. History of the Physiology of the Nervous System, taken more especially from the Writers on Phrenology. By S. WILKS, M.D., F.R.S. 1879.
8. Note on a Case of Tetanus.—By R. E. CARRINGTON, M.D., and G. A. WRIGHT, M.B. 1879.
9. Reflex Action in Diagnosis. By P. HORROCKS, M.D. 1881.
10. A Case of Symmetrical Softening of the Corpora Striata, followed by Bilateral Descending Degeneration with Secondary Anterior Poliomyelitis. By W. HALE WHITE, M.D. 1882.

11. On Hemianæsthesia. By S. WILKS, M.D., F.R.S. 1883.
12. A Case of Extensive Cerebral Softening with Descending Sclerosis in the Lateral Column. By G. N. PITT, M.A., M.D. 1883-84.
13. Saturnine Lunacy. By J. F. GOODHART, M.D. 1883.
14. The Theory of a Heat-centre from a Clinical point of view. By W. HALE WHITE, M.D. 1883-84.

1. This paper was the first English account of this disease. The author points out that the disease can be diagnosed with certainty: its diagnostic characters are:—I. A peculiar trembling of the head and limbs during movements, ceasing when the parts are supported, so that whilst in bed the patient gives no sign of his malady, but does when he attempts to move, thus differing from the movements in paralysis agitans which are more even, do not stop when the part is at rest, and are not accompanied by any extreme idiocy of manner. The movements of insular sclerosis are most like those of mercury poisoning, but differ in that they affect the head and neck, and have the character of embarrassed helplessness. II. Paralytic weakness, especially of the lower extremities. III. Contractions and rigidity chiefly of the lower extremities; they bear direct proportions to the paralysis. IV. Nystagmus; this symptom may be late, it is earliest in the cerebral form of the disease. V. There is little or no disturbance in power over the excretions. VI. As a rule, the electro-sensibility as well as the electro-motility are normal. VII. The words appear to cost too much pains, and are produced one syllable at a time, each syllable accentuated. VIII. There may be great enfeeblement of mind. Although, as might be expected, considering the diffuseness of the lesion, the symptoms vary; still, amongst this variety, there is a constant set as above enumerated. It is most common at the ages of 25-45; it is very chronic in its course, and death is usually accidental. The morbid anatomy is evidently that of chronic inflammation scattered in a broadcast style, especially in the white substance of the nervous centres producing patches of deep grey tint. The minute anatomy is shown very well in the spinal cord, where new sclerotic tissue is seen to advance inwards from a medullary ray, and all stages of the chronic inflammation may be seen. The paper concludes with a description of eight cases, in two of which there was an autopsy.

2. Meningeal hæmorrhage may be due either to variation in the blood pressure, or disease of the vessels. Out of 41 cases, 28 had either hypertrophied heart or granular kidney. Probably the

condition of periarteritis of the cerebral vessels described by MM. Bouchard and Charcot as causing miliary aneurisms, and so cerebral hæmorrhage, is the same thing as Drs. Gull and Sutton have described as hyaline fibroid degeneration of the arterial walls, and which is associated with chronic renal disease. The theory, therefore, which ascribes cerebral hæmorrhage to anomalies in the vascular tension is probably correct. Syphilitic disease of the arteries is one cause of meningeal hæmorrhage. Aneurism of the vessels at the base is a common cause; it generally exists in young people, and is a sequel to embolism. Mere excitement, or a sudden fit of passion, may, by inducing great pressure within the cerebral vessels, cause death. Three cases due to purpura are recorded. This may or may not be the result of blocking the vessels of leucocytes. The most constant symptom of the fatal cases is coma; of those that do not die, rather persistent stupidity remains. Meningeal hæmorrhage is difficult of diagnosis from alcoholism; the general appearance must be taken into account. Cases undoubtedly get well, but care must be exercised to avoid all excitement. The prognosis must depend upon the suddenness of the onset (one case in ten dies instantly), the completeness of the coma, the condition of the heart, the hardness of the pulse, and the state of the arteries. When the death is sudden, probably the medulla is implicated, either by pressure, or some disturbance of the circulation in it.

3. In this paper Dr. Wilks places on record some cases of cerebritis. In instances of cerebritis a large part of the cerebral hemisphere is involved in the inflammatory process, including the grey matter on the surface, the medullary matter within, and sometimes the central ganglia. As regards symptoms, there is no paralysis; the patient is simply devoid of mental power; there is generally no other symptom than this torpor, even if the meninges are involved. In unilateral meningo-cerebritis there may be some paralysis of the opposite side of the body. A case illustrative of this is given, but the patient did not know that she was paralysed. In this paper the doctrine that Dr. Wilks has often taught is emphasised, viz. that the formation of the animal body in two halves, and with two sets of nerves, necessitates the separation of the nerve-centres whenever an independent action of the limbs is required. The splitting of the spinal cord is a necessity of this independent action, and from this again of necessity follows a separate cerebral ganglion to rule over each. The mode in which

the two sides receiving different impressions can combine into one is by means of the commissure, and the explanation is no doubt of the same kind as that of single vision with two eyes.

The spinal cord may pass into the same condition that the brain does in sleep, and this is probably the explanation of some forms of hysteria with paralysis, and in hysteria with movement, there is over-action.

4. A case is recorded of a woman who, when a child 5 years old, was in the hospital for paralysis of the left arm and leg. She was admitted again when 27 years old. The left upper was much smaller than the right, the arm was close to the side, and there was flexion at the elbow, wrist and metacarpo-phalangeal joints; there were slow constant movements of the fingers. The left lower was also smaller than the right. She died of phthisis. There was found an old lesion in the grey matter of the corpus striatum, more or less atrophy of the right hemisphere, crus, pyramid, and olivary body, of the left hemisphere of the cerebellum and left side of the spinal cord. The right side of the base of the skull was smaller than the left. The history of the case was particularly clear; there could be no doubt but that the disease in the corpus striatum was primary; the atrophy of the other parts of the brain, as well as of the osseous and muscular structures, secondary. Some cases of congenital paralysis will bear a similar interpretation. The conclusion of Cotard, that partial atrophy of the brain is not a primary affection, but the result of traumatic, inflammatory, or other lesions, is justified. In the case here recorded there was no secondary degeneration, because the internal capsule was not involved. The case is interesting in confirming anatomy by the atrophy of the opposite half of the cerebellum. Eight additional cases are collected at the end of the paper. Out of the nine cases, seven commenced at or before the fifth year. Some of the cases were ushered in by convulsions, some by exanthemata; the paralysis is equally right and left, but always more in the upper than the lower. Sometimes the muscles are much wasted; sometimes in a state of painful tonic contraction, with slow involuntary movements; sometimes in a state of spastic contracture. The majority were subject from time to time to convulsive seizures, with or without loss of consciousness. There was very often some mental deficiency. The points of diagnosis from infantile paralysis are the hemiplegic distribution, the absence of marked atrophy in most cases, or the hypertrophied condition of the muscles that may be present in

some, the co-ordinated spastic contraction, the electrical reaction, the fits and the mental condition. The relation of this spastic hemiplegia, with slow movements, to the newly-described athetosis is discussed.

5. In this paper the cases during eleven years are recorded. Out of 51 cases available for statistics, 8 recovered; the traumatic cases were found to be more acute than the idiopathic; recoveries occurred more amongst cases of slight injury than amongst cases of severe; more recoveries took place amongst the cases in which the tetanic symptoms followed late upon the injury than amongst those in which they came on quickly; a more slow and chronic type of disease was more frequent amongst cases of mild injury than amongst cases of severe injury. In 12 out of 28 cases in which a post-mortem was made, there was lung mischief, in 13 nothing special was found. Of the 8 cases that recovered, 1 had Calabar bean; 1, Calabar bean and chloral; 1, Calabar bean, chloral, and morphia; 1, chloral; 1, chloral and iodide of potassium; 1, opium; 1, curara; 1, nitrite of amyl. In most of the Calabar-bean cases the drug was very rapidly pushed, either by subcutaneous injection or by the mouth; one patient took 125 grains of the extract in thirteen days, on one occasion he had seven successive doses of one grain each at intervals of fifteen minutes; 8 cases had Calabar bean alone; 2 had Calabar bean and chloral hydrate; 2 had Calabar bean, chloral hydrate, and morphia; 12 had chloral, and one of these had iodide of potassium added; only 1 case was treated simply with opium; 4 cases had curara; 3 cases had nitrite of amyl. The drugs used unsuccessfully were, quinine—bromide of potassium, bromide of ammonium, succus conii, Cannabis indica, nicotine, atropine, and aconite. Each case depends so much on its own merits, that, before any rigorous conclusions can be drawn, the article must be read in extenso.

6. A boy, aged 6, fell on his head six months before admission to the hospital; since then he has complained of pain, has seemed stupid, and has vomited; has lost power over his legs, and become blind. After admission, he got slowly worse; the face became paralysed; tonic contraction of the arms set in. At the end there was difficulty of swallowing; he gradually became emaciated and died. At the autopsy the following parts of brain had a gelatinous appearance; the posterior part of the corpus callosum and the posterior lobe on each side continuous with it; only the

posterior part of the corpus striatum was affected; the optic tracts ended in a mass of gelatinous material occupying the posterior part of the optic thalami and geniculate bodies. Sections of the pons, medulla, and cord showed disease occupying just those seats usually occupied by secondary degenerations, but rather darker and more defined than usual. The nature of the gelatinous material in the brain is discussed. Tumour, inflammation, and embolism appear to be excluded, and the degeneration shows no evidence of what preceded it. The secondary degeneration in the pons and cord is very interesting, because the posterior part of the internal capsule only was affected, and it therefore supports the views of Flechsig. Histologically, the condition in the cord was remarkable for the entire absence of granule cells, the absence of dense connective tissue, the great development of nuclei in the sheath of the vessels, and the substitution of a material, colloid in consistence and granular in structure, for the nervous and supporting elements of the tissue.

7. In this very interesting paper Dr. Wilks gives an account of some of the writings of the phrenologists, and of the transactions of the Phrenological Societies, and shows that they were not altogether worthy of the derision which their views excited. Dr. Wilks says, "I cannot but be struck with the great object which the writers presented to themselves, and the mode in which they proposed to prove the truth of their doctrine. Their object was the same as is now considered most rational. They discarded the notion that the brain was to be regarded as simply associated with the mind and there left, but they looked upon it as a compound and complex organ. They were the first who replaced the old method of the anatomists of slicing up the brain, by unfolding and dissecting it. They showed that it was made up of parts, each having its own function." Reference is made to a very able paper in the 'Phrenological Journal' for 1836, by Mr. Watson, entitled, "What is the use of the Double Brain?" and it is shown by quotations that the author had anticipated the views of Dr. Wilks on the duality of the brain, owing to which we have a double consciousness. Dr. Wilks takes this opportunity of giving clearly and fully his views on this subject. The phrenologists were remarkably accurate in the position in which they placed the seat of language; thus, Gall says, "The manifestation of verbal language depends upon a cerebral organ, and this cerebral organ lies on the posterior part of the superior orbital plate." By

numerous quotations it is shown, that the phrenologists of the first half of the present century were far in advance of the physiologists in their ideas of the locality of a part of the brain concerned with language. The phrenologists were also more correct in their views about sleep than many scientific writers of their time. In proof of this, it is mentioned that Dr. Caldwell records the case of a girl, in whom the brain, owing to loss of some of the bone of the skull, could be seen to protrude during activity, and to recede during sleep.

This paper by Dr. Wilks is so full of interesting discussions and references, that the reader must go to the original, as it is impossible to abstract it properly.

8. In this case the cervical enlargement of the cord was flattened on the right side; a cavity occupied the right cervical anterior cornu for its whole length: there was a smaller but similar cavity on the left side in the lumbar region; the cavities contained a rough shaggy material of a reddish-brown colour. The borders of the cavity showed marked degeneration of the nervous tissue. There was considerable hyperæmia, and several extravasations of blood were present.

9. In this paper a very good account is given of the best methods of obtaining reflexes, and also the diseases of the nervous system are classified according as to whether the reflexes are unaltered, absent, or exaggerated. The author performed some original experiments for the purpose of showing that the myotatic contractions were due to the loss of tone in the muscles, and some impairment of the reflex-loop. A frog's muscle was removed from the body, but could not be made to contract by pulling on its tendon, although it responded to mechanical irritation directly applied to the fibres; but if the nervous system was uninterfered with, the muscle being left in the body, it was easy to get myotatic contraction by pulling on the tendon. It is concluded that the reason why the first experiment failed was because of the loss of tone, because the author thinks the time experiments that have been performed are conclusive proof that myotatic contractions are not reflex. The theories for the increased production of myotatic contractions in lateral sclerosis are discussed. Directions are given for obtaining the various clonuses and the front tap contraction.

An experiment is described which showed that the fifth nerve

has in it no fibres which cause dilatation of the pupil on strongly stimulating a sensory nerve, but that this result is brought about by the third nerve, probably through inhibiting the tonic influence, kept up reflexly by this nerve upon the circular fibres of the iris. The oscillations of the pupil are about twice as many for far as for near distances; this oscillation is absent in locomotor ataxy. Exhaustive or extreme pain causes dilatation of the pupils. Thus they are dilated after an epileptic fit, or after having a tooth extracted. These facts are useful in diagnosing malingering.

10. A boy, *æ*t. 6½, ran away from school, but was shortly afterwards found lying in the snow, and when picked up, could not stand, having right hemiplegia. After this attack he seemed stupid, never spoke or read anything, and has always suffered from loss of control over the bladder and rectum. This case is too long to condense satisfactorily. The patient had various nervous symptoms, such as paralysis of various parts of his body, loss of intellect, &c.; but the chief interest lay in the extraordinary hyperpyrexia, which at first was not constant, but towards death gradually became more so. It was very persistent; cold baths had to be very frequent to keep it under; the highest temperature reached was 107°. At the post-mortem examination, the brain and cord were the only parts affected. There the only abnormality was a small soft patch, a quarter of an inch in diameter, at the anterior part of each corpus striatum. There was well-marked degeneration on both sides down the crura into the cord, taking the usual course; the inflammation had at some places spread into the anterior cornua. The question is discussed whether the change in the lateral columns is primary; but the author concludes that the change in the anterior cornua is secondary. The hyperpyrexia is discussed in his paper on the "Heat-centre Theory," in the succeeding volume of the Reports.

11. Dr. Wilks points out, that whilst hemiplegia produced by disease of the corpus striatum is not a paralysis of half the body, but only of parts of it, hemianæsthesia produced by a lesion of the sensory fibres would in most cases be not only a complete loss of sensation of the entire half of the body, but of the special senses also; but hemianæsthesia accompanying hemiplegia is rare and exceptional; whenever loss of sensation exists with hemiplegia, the lesion will be found to affect the back part of the internal capsule. Complete hemianæsthesia, such as is above mentioned as theoretically

possible, is unknown to Dr. Wilks. All the complete cases of complete hemianæsthesia that he has seen are functional, and get well. Bearing in mind the dual action of the brain, it is possible that the hemianæsthesia is due to the cessation of sensory function of one half of the brain, and the cases which follow on a blow may be due to some concussion affecting half the brain only. Many facts and arguments are brought forward to prove these two points. Cases are given to show, that on rare occasions, male patients may present the same symptoms of this nature as female.

12. A girl, æt. 2, admitted for right hemiplegia, left facial paralysis, double optic neuritis, nystagmus, unequal pupils and vomiting. On admission, the case was thought to be one of tubercular meningitis; but as the child lingered on in an almost moribund condition, the diagnosis of cerebral tumour was made. The child died about seven months after admission. At the post-mortem examination, in addition to the extraordinary emaciation, so great, that the superficial cervical plexus could be observed under the skin, there was observed extensive softening of the middle part of the right cerebral hemisphere; no cause whatever could be found for it. There was well-marked descending sclerosis of the crossed pyramidal tract in the cervical region on the left side: most of the organs of the body were tuberculous; but there was no evidence of tubercular meningitis, and although the right middle cerebral artery was small, there was no evidence that the softening was due to this, for it did not correspond to the distribution of the artery.

13. The resemblance between the cerebral symptoms of lead-poisoning, of alcoholism, and of paralysis, is pointed out. Also the importance of not confusing the diagnosis, because the treatment is different, although it is extremely probable that plumbism will give rise to general paralysis. The similarity in the pathology of the three conditions is very marked, all being characterised by wasting cerebral changes; and in the case of lead and alcohol the analogy may be pushed still further, for both lead to persistent contraction and thickening of the vessels with arterial spasm. Therefore opium, digitalis, nitrite of amyl and nitro-glycerine will be of use.

14. The author points out that nervous non-inflammatory pyrexia is present in the following maladies:—1. Tumour of the cord, especially of the cervical region. 2. Tumours of the brain, especially of the pons. 3. Hæmorrhage of the brain, especially of the pons. 4. Embolism of certain cerebral arteries. 5. Ill-defined

degenerative changes in the brain. 6. Insular sclerosis. 7. Locomotor ataxy. 8. Obscure nervous cases that die without any change being found post-mortem. 9. Obscure nervous cases that get well, "Hysterical pyrexia." 10. Mental disease. 11. Injury to the spine. 12. Injury to the brain. Lowering of the temperature is present in—1. Tumours of the brain; 2. Cerebral hæmorrhage; 3. Degenerative changes in the brain; 4. Mental disease; 5. Injury to the spine. Cases are given illustrating all the above conditions. The author shows that all the cases may be explained, by supposing that there exists on the cerebral cortex, near the fissure of Rolando, a calorific centre which presides over the thermogenetic tissues of the opposite side of the body, restraining their activity. The fibres from the centre proceed down, by means of the corona radiata near the central ganglia, through the crura, pons medulla and cord, decussating in their course. In the cases of organic disease of the brain accompanied by non-inflammatory pyrexia, the lesion destroyed these fibres in some part of their course, thereby causing a rise of temperature, owing to the inhibitory action of the calorific centre being cut off. In the cases in which the temperature fell, it was due to the fact that the inhibitory fibres were stimulated. The physiological side of the question is fully discussed, and it is shown that the clinical facts agree with physiological experiments. Whether or not the calorific centre is a genuine calorific one, or whether it be a muscular vasomotor one, is not yet decided; probably the former is the true view. In the cases of hysterical hyperpyrexia the explanation most probably is that there exists functional disturbance of the calorific centre.

W. HALE WHITE, M.D.

I. On a Diagnostic test for Sciatica.—DE BEURMANN (*Archives de Physiologie*, April 1884, p. 375). Place the patient in a recumbent position, keep the leg extended, and gradually flex the thigh on the pelvis. Pain is caused, and he resists the movement.

Flex the thigh in the same way, but with the leg flexed. No pain is caused, and no existence is encountered.

The pain is caused, according to the author, by the tension put on the sciatic nerve, which, as he shows by experiment on the dead body, is greater in the first manipulation than in the second. He thinks the manipulation described furnishes a diagnostic sign of pain due to inflammation of the nerve as opposed to pain from disease of the joint or of neighbouring structures, and that it